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A TREATISE

ON THE

PRINCIPLES AND PRACTICE

OF

MEDICINE;

DESIGNED FOR THE USE OF

PRACTITIONERS AND STUDENTS OF MEDICINE.

BY

AUSTIN FLINT, M.D., LL.D.,
LATE PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE
IN THE BELLEVUE HOSPITAL MEDICAL COLLEGE, NEW YORK, ETC.

SIXTH EDITION,
REVISED AND LARGELY REWRITTEN BY THE AUTHOR.

ASSISTED BY

WILLIAM H. WELCH, M.D.,
PROFESSOR OF PATHOLOGY IN JOHNS HOPKINS UNIVERSITY, BALTIMORE,
AND
AUSTIN FLINT, M.D., LL.D.,
PROFESSOR OF PHYSIOLOGY IN THE BELLEVUE HOSPITAL MEDICAL COLLEGE, NEW YORK.

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PREFACE.

THE Preface to the fifth edition of this treatise, written in December, 1880, might well serve as a preface to this, the Sixth Edition, as regards the general statements of the author's aims and labors in bringing "the work, in all respects, up to the level of the present state of advancement in both the Principles and the Practice of Medicine. Time and effort have not been spared for this end."

The treatise embraces in its scope, General Pathology, as well as Practical Medicine; and, while each of these departments of knowledge was in no inconsiderable degree advanced by zealous workers in different countries during the period between the publication of the fourth edition, in 1873, and the issue of the fifth edition, in 1881, the progress, especially in General Pathology, within the five years since 1881 has been immense. Certain of the advances within this half-decade were of such interest and importance that the author added, in 1883, an Appendix to the fifth edition, in order that the remarkable researches of Koch and others on the bacillus tuberculosis should be put before his readers as soon as these observations had assumed a permanent place among the established facts in medical science. It is almost unnecessary to add that these and other recent observations in bacteriology have been incorporated in this, the Sixth Edition, in the body of the work. The claim, in the Preface to the fifth edition, "that the eliminations, substitutions, and additions rendered it essentially a new work," can with equal propriety be made for the present edition as compared with the edition issued in 1881.

The careful and thorough revision, of which this edition is the result, was practically completed by the author's own hand, in March, 1886, with the assistance, as in the revision for the fifth edition, of Dr. William H. Welch. The labor which has since been expended on the work, by Dr. Welch and by the writer of this Preface, has been mainly that of carrying the book through the press. After the final revision had become far advanced, the author made arrangements, which have been carried out in exact accordance with his views and wishes, by which he would be spared the labor of close proof-reading. This part of the work has been performed, perhaps, with even more anxious and critical care than if it had been done under his own eye.

It can hardly now be considered indelicate to allude to certain qualities of mind, developed by training and opportunities most unusual, which enabled the author to produce, as the crowning work of his long professional life, this treatise on the Principles and Practice of Medicine.

The basis of the work is an unbroken series of records of cases in private practice and in hospitals, begun in 1833 and continued for more than half a century, covering sixteen thousand nine hundred and twenty-two folio pages of manuscript, written with the author's own hand. These records embrace carefully-written histories of cases in all departments of practical medicine, observed under varied conditions of life, climate, and general surroundings. Soldiers in camp and barracks; the rich and the poor; those affected with diseases incident to lives of ease and luxury and paupers in hospitals; the pioneers of Western New York and the inhabitants of the metropolis; patients in the wards of the almshouse and hospitals of Buffalo, of the Marine Hospital in Louisville, Kentucky, the great Charity Hospital in New Orleans, Louisiana, the Bellevue Hospital, the Charity Hospital, the dispensaries, and similar institutions in the city of New York; cases observed in the experience of a quarter of a century as a general practitioner, and of more than another quarter of a century as a consulting physician, including the epidemics which have occurred in this country within the last fifty years,—the experience derived from these various sources of observation, carefully recorded, studied, and analyzed, was finally used in the composition of this treatise, the first edition of which appeared in 1866. In the mean time, the author's original contributions to practical medicine, embodied in special treatises, in communications published in medical periodicals, and in Transactions of medical societies, have left their impress upon many departments which, in recent years, have been classed as specialties; although he was always a physician, never a specialist. A student of the history of practical medicine will often find observations and ideas, assumed to be of recent date, which had been anticipated by the author many years before.

It would be tedious to enumerate the systematic works, original articles, reviews, etc. published by the author within the last fifty years; but the scientific spirit which pervades all of his writings is well illustrated by the following extract from the Preface to the fifth edition of this work:

"In making changes, the author has not been influenced by any sense of obligation to maintain consistency of views with the previous editions of this treatise, or with other works which he has written. Whenever statements are found to vary from those made at a prior date, the simple explanation is that the latter, in the light of more recent reflection and enlarged knowledge, seem to him no longer tenable. He has endeavored to regard his own writings, in this point of view, divested of the partiality of authorship, and to subject them to as critical an examination as if they were the writings of another."

Added to a literary training and a recorded experience almost without parallel, was an exceptional knowledge and application of the best methods of teaching medicine, rendering the author one of the most popular and successful medical teachers of his time; a statement which will be echoed by thousands of practitioners, throughout this country and in foreign lands, who have attended his lectures. The lectures given by the author, in 1885–86, completed his fiftieth regular course on the Principles and Practice of Medicine.

Such, in brief, is the history of the author of this treatise as a practitioner, writer and teacher. While sentiments of affection and veneration have led the writer to take the responsibility of recording this history here, they have not carried him beyond the limits of accurate statement, or, he hopes, those of propriety and delicacy.

In the Preface to the fifth edition the author "tenders his thanks to the present publishers for a continuance of the courtesy and kindness which rendered most agreeable his relations, for a quarter of a century, with their predecessors, Blanchard & Lea and Henry C. Lea." Judging by the popular criterion of commercial success, the publishers can certainly testify to the appreciation, on the part of the profession, of the completeness with which each one of the preceding five editions has been made to represent the actual condition of medical knowledge at the time of its publication; and it may be safely asserted that during the last twenty years no systematic work on the Principles and Practice of Medicine, printed in the English language, has been more extensively circulated and read, and has had a greater influence on the practice of English-speaking physicians, than has the present treatise. It is also safe to say that the work is now more carefully and thoroughly revised and rewritten, and is presented in a better scientific and literary form, than any one of the former editions.

While it is unnecessary to specify all of the changes which have been made in the Sixth Edition, it may not be out of place to indicate some that are important as tending to perfect its symmetry and to bring the matter closely up to the existing state of knowledge in the department of Practical Medicine. The most important of these additions and alterations are the following:

Among the entirely new articles, special attention may be called to the following: Infectious Tumors; Syphilitic Disease of the Lungs; Cerebral Syphilis; General Considerations relating to Inflammatory and Structural Diseases of the Spinal Cord; Spastic Cerebral Paralysis of Children; Hereditary Ataxia; Myxœdema; Multiple Neuritis; General Pathology of Fever; and Milk Sickness. In addition to these new features, many articles have been entirely rewritten; and in nearly every article changes and additions, some of them very important, have been made.

In certain parts of the work, especially in Section IV., articles have been rearranged so as to follow each other in a more rational and logical sequence than in former editions.

As already stated, the Sixth Edition contains a full consideration of recent discoveries concerning the bacterial origin of various infectious diseases, as will be rendered evident by a consultation of the article on Vegetable Parasites in the chapter on Etiology, and articles in the chapters treating of Tuberculosis, Typhoid Fever, Cholera, etc.

Five diagrammatic engravings have been introduced, in connection with the nervous system, with the view of illustrating the descriptions involved in treating of the topical diagnosis of cerebral and spinal diseases.

By careful condensation and rearrangement and by the rigid omission of obsolete matter the many additions contained in this edition have been accommodated without an increase in the number of pages, but in no instance, it is believed, have clearness and accuracy of description been sacrificed to brevity.

As in the case of the fifth edition, the labor of revision has been shared by Dr. William H. Welch, Professor of Pathology in the Johns Hopkins University and formerly Professor of Pathological Anatomy and General Pathology in the Bellevue Hospital Medical College. Dr. Welch has contributed, in Part I., the first seven chapters and a large part of the eighth chapter. He has also revised, and in great part rewritten, the descriptions of anatomical characters of the diseases considered in the rest of the volume, and has written the articles or parts of articles relating to bacteriology. In the words of the Preface to the fifth edition, "It is believed that these portions of the work will serve as a digest of the essential facts pertaining to general and special pathological anatomy, as far as this important branch of study bears upon practical medicine." As regards the entire work, Dr. Welch writes that the "revision throughout has been thorough and complete; and it is believed that the new edition has taken cognizance of all of the more important discoveries in pathology and clinical medicine since the previous edition was issued."

In the words of the author, repeating the language of the Preface to the first edition, "In submitting the work to the judgment of his fellow-teachers and practitioners, the author ventures to hope that it may be found to represent fairly the existing state of the science of medicine with respect to the subjects of which it treats, and to reflect the views of those who exemplify, in their practice, the present stage of progress of medical art."

AUSTIN FLINT.

NEW YORK, August, 1886.

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THE
PRINCIPLES AND PRACTICE
OF
MEDICINE.

INTRODUCTORY CHAPTER.

Scope of the term Medicine—Use of the term in contradistinction to Surgery and Obstetrics—Subdivisions of the Different Departments of Medicine, or Specialties—The General Object of this Work—Meaning of the phrase Principles and Practice of Medicine—Definition of Pathology—Division into General and Special Pathology—Nomenclature of Diseases—Subdivisions of General Pathology: namely, Morbid Anatomy, including Morbid Changes of the Fluids of the Body; Etiology; Symptomatology; Diagnosis; Prognosis; Prophylaxis, and Therapeutics—Relations of these Subdivisions to Special as well as to General Pathology—Definition of Disease—Definition of Health—Relationship of Pathology to Physiology—Progress of Pathological Knowledge.

MEDICINE, in the largest sense of the term, comprehends everything pertaining to the knowledge and cure of disease. In a more restricted sense the term is used in contradistinction to Surgery and Obstetrics. The latter are properly departments of Medicine in the comprehensive sense of the term, and, although they may be cultivated separately, they cannot be disconnected from principles which are common to them and to Medicine in its restricted sense. The medical profession embraces all who devote themselves to the study and practice of medicine proper, surgery, and obstetrics, either separately or combined. The physician is a member of the profession who devotes his attention to the diseases which belong to the department of medicine proper—*i. e.* medicine in the restricted sense of the term. The physician may, or may not, undertake the duties which belong to surgery and obstetrics. In this country most physicians are, of necessity, to a greater or less extent, also surgeons and obstetricians—in other words, general practitioners. It is only in cities and large towns that practitioners can devote themselves exclusively or chiefly to surgery and obstetrics as separate departments of medicine. The distinction of physican, surgeon, and obstetrician in this country is purely conventional. The only degree conferred by our universities and medical colleges is that of Doctor of Medicine, which authorizes the practice of either or all the departments, and the same is true of licenses to practise medicine.

The division of medicine into the three departments which have been named is natural, and has contributed to the knowledge acquired in each department. Subdivisions have also been found convenient and useful. The latter are commonly known as specialtics, and they who devote themselves to particular subdivisions are called specialists. The more important of the subdivisions now recognized as specialties are affections of the eye and ear, of the skin, of the genito-urinary system, diseases peculiar to females, orthopædic surgery, disorders of the mind and nervous system, diseases of the throat, nose, and larynx, and of the heart and lungs. Specialties result from an increase of knowledge rendering it difficult or impossible for one mind to compass all that has been ascertained in each of the three departments of medicine. The special cultivation of the several subdivisions of medicine leads to a further development of knowledge relating to each subdivision, and hence conduces to the progress of medicine. But as the great principles of medicine are common to medicine proper, surgery, and obstetrics, so with regard to the subdivisions: they cannot be completely isolated from the departments to which they respectively belong. A particular class of affections cannot be studied satisfactorily to the entire exclusion of others and without reference to the general laws of disease. Directing the attention exclusively to a specialty leads to the habit of attributing to it an undue relative prominence, and of regarding the diseases belonging to it as of paramount importance, when they may be secondary or merely incidental to others which, from being overlooked or not sufficiently appreciated, fail to receive appropriate treatment. It is never advisable to pursue medical studies with exclusive reference to a specialty, or to adopt one at the outset of medical practice.

The object of this work is to present the outlines of Medicine proper; that is, of Medicine in contradistinction to Surgery and Obstetrics. *The Principles and Practice of Medicine* is a title of this department considered as a province of medical teaching. This title is here adopted in preference to others, used by English and American authors, such as the Theory and Practice of Physic, General and Special Pathology, Pathology and Practical Medicine, or the Science and Practice of Medicine. The Principles and Practice of Medicine comprehend everything pertaining directly to the knowledge and cure of those diseases which the physician is called upon to treat. The province of medical teaching thus designated properly enough embraces the prevention of disease, and it may include anything which concerns the conduct of the physician in the treatment of patients affected with disease.

The study of disease as a province of scientific knowledge is called *Pathology*. This province consists of two important divisions—namely, *General* and *Special Pathology*. It is desirable to have a clear understanding of the terms which distinguish these two divisions of pathology. Diseases are presented in particular forms or species, constituting what are commonly known as individual diseases. The circumstances which give to the different diseases their individuality will be noticed hereafter. Now, the study of individual diseases constitutes special pathology. On the other hand, there are morbid conditions which are not peculiar to any individual disease, but are common to a greater or less number of diseases. The study of these conditions constitutes general pathology. Inflammation, for example, is a morbid condition which exists in a large number of individual diseases. The study of inflammation as a condition common to different diseases belongs to general pathology, while the study of the individual inflammatory diseases belongs to special pathology. To take another illustration: a morbid condition which enters into a number of individual diseases is called fever. In this sense of the term fever it belongs to general pathology, but the study of the different forms of fever, or individual fevers, belongs to special pathology. The relation of general to special pathology is analogous to the relation of general to special anatomy, the former describing the several tissues which enter into the composition of the different organs of the body, and the latter describing the particular organs composed of the tissues. As the number of tissues is small in comparison with the number of organs, so the morbid conditions belonging to general pathology are few as compared with the great number of diseases belonging to special pathology.

The province of medical teaching, entitled the Principles and Practice of Medicine, comprises both general and special pathology. The subjects which belong to the principles of medicine are derived from general pathology. The principles of medicine and general pathology are, in fact, synonymous terms, each term having the same scope of application. And, in like manner, the subjects which belong to the practice of medicine are derived from special pathology. These two terms relate to the same division of pathological knowledge, the former term being somewhat more comprehensive in its scope than the latter. The principles of medicine thus, on the one hand, and the practice of medicine on the other hand, constitute divisions which coincide with the two divisions of pathology distinguished as general and special. Moreover, these divisions are in accordance with the distinctions expressed by the terms Science and Art. "Science is knowledge reduced to principles; art is knowledge reduced to practice." The principles of medicine constitute medical science; the practice of medicine is the exercise of medical art. The object of this work being to present the outlines of both the principles and the practice of medicine, or of both general and special pathology, or, again, of medical science and art, it will be divided into two parts corresponding to these divisions. The outlines of the Principles of Medicine, or General Pathology, will form the *First Part* of the work, and the *Second Part* will be devoted to the Practice of Medicine, or Special Pathology. In adopting this arrangement, however, I shall not be bound by it so closely as to treat of the topics belonging to general pathology exclusively in the first part of the work. It will be more convenient to defer the consideration of some of these topics, and to treat of them incidentally in connection with individual diseases. Moreover, I shall treat of general pathology in its relation to medicine, passing over or noticing very briefly those topics which are chiefly important in a surgical point of view. Although the general principles of pathology are common to both medicine and surgery, certain topics have relations especially to either the one or the other of these departments. The terms *medical pathology* and *surgical pathology* are used in conformity with this distinction.

The subjects of General Pathology—namely, the morbid conditions common to a greater or less number of individual diseases—are to be considered under various points of view; hence this division of pathology admits of several subdivisions. One point of view relates to nomenclature, or the naming of diseases. The great desideratum in nomenclature, as applied to diseases, is that the name of each disease shall express the morbid condition involved and its situation. The names which were formerly applied to different forms of disease were frequently fanciful, and many of these are still in use, owing to the difficulty and inconvenience of displacing them after they have become established in medical literature; and in not a few instances it is by no means easy, with our existing knowledge of the essential character of morbid conditions, to substitute more appropriate names. Some approach, however, has been made toward a nomenclature which shall measurably secure the advantages derived from this source in other branches of knowledge, more especially in chemistry. The existence of inflammation, which enters into so large a number of individual diseases, is expressed by the suffix *itis* (ἰτης) added to the anatomical name of the part affected. Thus, bronchitis, pneumonitis, pleuritis, peritonitis, etc. are names denoting the inflammatory character of the diseases to which they refer and the particular structure which is the seat of the inflammation. The suffix *rhœa* (ῥεω) denotes the existence of the morbid condition known as transudation, or flux, occurring in a situation where the liquid escapes upon a mucous surface: examples are enterorrhœa, bronchorrhœa, gastrorrhœa, cystorrhœa, terms which have not, as yet, come sufficiently into vogue. The suffix *rhagia* (ῥηγνυμι) expresses a flow of blood, or hemorrhage from a mucous surface: examples are metrorrhagia, gastrorrhagia, enterorrhagia, bronchorrhagia, in like manner terms which have not displaced others in common use. The suffix *algia* (ἀλγος) signifies a morbid condition characterized by pain without inflammation. Thus, neuralgia is a general term applied to this condition affecting any nerve or nerves; gastralgia, enteralgia, pleuralgia, etc. are terms severally expressing the neuralgic character of the affection and its seat. Words ending in *æmia* (αἱμα) are applied to certain morbid conditions of the blood: examples are anæmia (impoverishment of the blood), uræmia (morbid accumulations of urea in the blood), septicæmia (putrid infection of the blood), and pyæmia (purulent infection of the blood). Words ending in *uria* (οὐρον) are applied to certain morbid conditions of the urine: examples are albuminuria, hæmaturia, oxaluria. The prefix *hydro* (ὑδωρ) denotes a dropsical affection of the part named; as, hydrothorax, hydrocephalus, hydro-peritoneum, hydro-pericardium. The prefix *pneumo* (πνευμα) denotes the presence of air in the part; as pneumothorax, pneumo-pericardium. The name of an inflammatory disease to which *peri* is prefixed signifies inflammation of the membrane investing the part inflamed, and the prefix *para* denotes inflammation of the neighboring connective tissue: examples are perihepatitis, perinephritis, parametritis. etc. The suffix *pathy* (παθος) is used to express the fact of a morbid condition of a part without indicating its character: instances are arthropathy and encephalopathy. A termination in *oma* signifies a tumor, as sarcoma, carcinoma, myxoma.

It is thus seen that the effort to introduce names expressive of the character and seat of morbid conditions has, in a measure, succeeded. Further improvement in nomenclature will doubtless be made as our pathological knowledge increases.

An important subdivision of General Pathology relates to the appreciable morbid changes of the solids and fluids of the body. The study of all changes appreciable by the naked eye or with the help of the microscope constitutes a branch of pathology of great importance called morbid anatomy. Morbid anatomy is not confined to the study of the changes which occur in the tissues or solid parts; it embraces any changes which the eye can appreciate in the fluids of the body. The latter, not less than the former, are to be distinguished as anatomical, whereas those changes which are not visible either with the naked eye or by means of the microscope, but require for their detection processes of analysis or the employment of reagents, are usually considered as falling, not within the scope of morbid anatomy, but within the domain of animal chemistry. Morbid changes not visible or not yet ascertained with our present means of observation are distinguished as *functional*, and are also said to be *dynamic*. Doubtless in all the so-called functional or dynamic deviations from health there are either molecular or cellular—that is, anatomical—changes at present inappreciable, or at all events undiscovered, which may be hereafter ascertained by continued investigation with improved means of observation. Appreciable anatomical changes are distinguished as *lesions*. The study of the minute anatomy of the tissues and fluids of the body with the microscope is called *Histology*, and the term *Morbid* or *Pathological Histology* is sometimes used to designate that part of morbid anatomy relating to those abnormal changes which are the objects of microscopical research. The study of the origin and development of pathological processes is called *Pathogenesis*.

Anatomical changes, or lesions, belong to general pathology in so far as they are common to a greater or less number of individual diseases. Their consideration, as involved in individual diseases, enters into special pathology, or the practice of medicine. Such is the extent as well as importance of morbid anatomy that it constitutes a distinct branch of medical knowledge. Treatises are specially devoted to it; and these are to be studied, in conjunction with the examination of morbid specimens, in order to become fully and practically acquainted with the various alterations in structure, form, size, etc. which are incident to disease. It is a fact, however, not to be lost sight of, that lesions do not constitute, but are the results of, disease. In other words, they are always due to underlying morbid actions or processes which may not be directly appreciable or well understood, but in which really consists the local disease. This fact, although obvious, is liable to be overlooked. Lesions are of course serious or otherwise according to their character, their situation, and the amount of structural change involved. In *Part First* will be found the outlines of general medical pathology, and in *Part Second* is given a concise account of the lesions characterizing different individual diseases.

Another subdivision of General Pathology relates to the causation of disease. The study of the causes of disease is called *Etiology*. As belonging to General Pathology, or the Principles of Medicine, this branch of medical knowledge will be considered in *Part First* of the work; and the causes involved in the production of each of the individual diseases will be embraced in the account of the latter in *Part Second.*

The great number and variety of phenomena or events to which disease gives rise constitute another subdivision of General Pathology. These phenomena or events are called symptoms, and their study constitutes a branch of medical knowledge called *Symptomatology* or *Semeiology*. Considerations relating to symptomatology, as belonging to General Pathology, will claim attention in *Part First;* and the symptoms of individual diseases respectively, forming, as they do, a highly important part of Special Pathology, or the Practice of Medicine, will be considered in *Part Second.*

Closely connected with Symptomatology is another subdivision of General Pathology, called *Diagnosis*. Diagnosis is the discrimination of diseases from each other. General considerations relating to this branch of medical knowledge will enter into *Part First*. In treating of individual diseases in *Part Second* the means of discriminating them will be found to possess an importance second only to their treatment, and to be an essential prerequisite for the latter.

Another subdivision which will claim notice in both parts of this work is *Prognosis*, or the prediction of the termination of diseases.

The prevention of disease forms a branch of medical knowledge called *Prophylaxis*. This belongs alike to General and Special Pathology.

Lastly, the treatment of disease is called *Therapeutics*. General principles relating to the treatment of disease may be appropriately considered in connection with General Pathology. This portion of the subject is distinguished as *General Therapeutics*. I shall devote to it, together with prophylaxis, a chapter in *Part First*. It is hardly necessary to add that the treatment of individual diseases, distinguished as *Special Therapeutics*, is, in a practical view, the most important of the different aspects under which they are to be considered, being, in fact, the great end of both the principles and the practice of medicine.

It is thus seen that the subdivisions of General Pathology, which will be taken up in the first part of this work, represent also the different points of view under which individual diseases are to be considered in the second part of the work; that is, individual diseases, as well as the morbid conditions common to a greater or less number of diseases, are to be considered with reference to the morbid changes, either of solids or fluids, which they may respectively involve, together with their causes, their symptoms, their discrimination or diagnosis, their prognosis, their prevention, and their treatment. Morbid anatomy, etiology, symptomatology, diagnosis, prognosis, prophylaxis, considered as branches of medical knowledge, belong to General Pathology, and to these may be added general therapeutics; and, on the other hand, Special Pathology, or the Practice of Medicine, considers the truths contained in these subdivisions of General Pathology in their application to individual diseases.

Pathology has been defined as the study of disease, but disease has not yet been defined. The definition of disease is confessedly difficult. It is easier to define it by negation, to say what it is not, than to give a positive definition—that is, a definition based either on the nature or essence of the thing defined or on its distinctive attributes. Disease is an absence or deficiency of health, but this is only to transfer the difficulty, for the question at once arises, How is health to be defined? And to define health is not less difficult than to define disease. If all the tissues and organs of the body have their normal integrity and properties, if the fluids of the body be in no respect abnormal, if all the functions of the organism be completely and harmoniously performed, health undoubtedly exists. But this perfection of health is purely ideal; it never actually exists. An examination of the bodies of the healthiest persons would probably reveal lesions of some kind; certain deviations from the normal composition of the different fluids are not inconsistent with the evidences of health in other respects; functions of different parts may be disordered to a certain extent without sufficient disturbance to constitute disease. Gradations of health are implied in the qualifications of this term in common use. If the term health expressed a well-defined state, it would be a pleonasm to add to the term, as is often done, the adjectives good, excellent, etc.; and, on the other hand, to speak of health as poor, bad, miserable, etc. would involve a solecism. In short, health and disease are so imperceptibly merged into each other that the line of demarcation cannot be drawn with precision. And this is true of other departments of knowledge. It is not easy, for example, to settle upon the characters which mark the boundaries of the animal and the vegetable kingdom. But as there is rarely any practical embarrassment in distinguishing an animal from a vegetable, so with regard to health: if an important disease of any kind exist, the fact of its existence is in most cases sufficiently obvious. If, however, it be desirable to define disease otherwise than by saying that it is the absence or deficiency of health, the definition proposed by Chomel is, perhaps, as good as any other. According to this author, disease may be defined to be *a notable disorder affecting more or less of the constituent parts of the living organism as regards either their material constitution or the exercise of their functions.*[1]

By regarding disease as the absence or deficiency of health we are led to the consideration of the relationship of pathology to physiology. Physiology studies the operations which go on in the healthy organism. The morbid conditions which are the subject of pathological study are these operations disordered or perverted. Pathology has been called morbid physiology. Both are, in fact, parts of one science, the science of life, or biology. Both are alike occupied with vital properties, actions, and processes, the difference being that physiology investigates them under the circumstances of health, and pathology under the circumstances of disease. The division is arbitrary, although sufficiently marked and appropriate.

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[1] "Un désordre notable survenu, soit dans la disposition matérielle des parties constituentes, du corps vivant, soit dans l'exercise des fonctions." For an enumeration of the various definitions proposed by different writers, and some excellent remarks on the subject, the same author may be consulted: *Elémens de Pathologie générale*, quatrième édition.

Such being the relationship of pathology to physiology, it might be expected that the former would advance in proportion to the progress of the latter. This is measurably true. While our knowledge of pathological conditions does not consist of deductions from what is known of the operations within the organism in health, but is derived from the direct study of disease, every important physiological discovery sheds more or less light on the department of pathology. In striving to penetrate into the nature of morbid conditions, it is evident that the chief difficulty arises from the imperfection of our knowledge of the properties, actions, and processes of health. There will be frequent occasions in the progress of this work to remark that the pathologist may expect to be better able to explain the phenomena of disease when the physiologist has succeeded in elucidating more fully the phenomena of health.

In proceeding now to present the outlines of Medicine the aim of the author will be to give a truthful representation of pathological knowledge as it exists at the present moment. The progress of pathological knowledge has wrought, within late years, much change in both the principles and practice of medicine. Concerning further progress and its effects, it would be in vain to speculate; but it is not to be expected that a faithful exposition of medicine as it exists at the present moment will serve as a lasting guide for the student and practitioner. And in the study of medicine next in importance to an acquaintance with what is actually known is a just appreciation of the limits of our present knowledge. The latter is often important as regards its bearing on the treatment of disease, and it conduces to a condition of mind most favorable for either contributing to, or keeping pace with, the continued progress of knowledge.

PART I.

PRINCIPLES OF MEDICINE, OR GENERAL PATHOLOGY.

CHAPTER I.

DISTURBANCES OF THE CIRCULATION.

Local Anæmia—Hyperæmia—Hemorrhage—Thrombosis and Embolism—Dropsy.

THE subjects embraced in General Pathology may be classified and considered under the following headings:

1. Disturbances of the circulation.
2. Inflammation.
3. Active alterations of the tissues.
4. Passive alterations of the tissues.
5. General pathology of the blood.

The disturbances of the circulation embrace *local anæmia*, *hyperæmia*, *hemorrhage*, *thrombosis and embolism*, and *dropsy*.

Local Anæmia.

Local *anæmia*, or *ischæmia*, signifies a deficiency of blood in a part. General anæmia, or oligæmia, will be considered in connection with the pathology of the blood. Local anæmia is due either to an increase of the resistance naturally offered to the flow of blood through a part or to the presence of new obstacles within or outside of the vessel. Increase of the natural resistance is caused by contraction of the arteries in consequence of direct stimulation of their muscular coat or under vaso-motor nervous influence. The pallor of the skin from the effects of cold, and that of the face at the onset of an epileptic paroxysm or in consequence of violent emotions, are examples of local anæmia in consequence of spasm of the arteries. Atheroma, obliterating endarteritis, thrombosis, and embolism may be cited as causes of local anæmia acting within the vessels; compression of the arteries by tumors, exudations, or bandages illustrates the effect of external agents. As will be explained in treating of embolism and thrombosis, the presence or the absence of anastomoses is of great importance in determining the degree of anæmia which follows the obstruction of a blood-vessel. By collateral anæmia is understood the diminution in the amount of blood in a part in consequence of its excessive accumulation in other parts. An anæmic part is generally pale, shrunken, dry, and, if exposed to the air, cool. The effects of long-continued and marked anæmia of a part are atrophy and frequently fatty degeneration. If the anæmia be extreme, death of tissue may result.

Hyperæmia.

An increased amount of blood in the vessels of a part constitutes *hyperæmia.* Two forms of hyperæmia are recognized—*active* or *arterial,* and *passive* or *venous.* Active hyperæmia is also called fluxion. The term congestion is generally employed as a synonym for hyperæmia, although some understand by congestion only active hyperæmia. In active hyperæmia an increased amount of blood is brought to a part by the arteries.

The usual cause of active hyperæmia is a relaxation of the muscular coats of the arteries of a part, so that there results a diminution of the resistance naturally offered by the arterial tonus to the circulation of the blood. This relaxation may be the result of irritation of vaso-dilator nerves (neuro-tonic congestion), or of paralysis of vaso-constrictor nerves (neuro-paralytic congestion), or of some influence acting directly upon the coats of the arteries. Although these three modes of production of active congestion have been proven, it is not generally easy in a given case to determine which of the three factors is involved.

Collateral or *compensatory hyperæmia* is the transmission of an increased amount of blood to a part in consequence of local anæmia of another, usually an adjacent, part. The sudden removal of long-continued pressure upon the arteries may be a cause of active hyperæmia; as, for instance, the over-distension of the abdominal vessels following rapid withdrawal of large accumulations of fluid in the peritoneal cavity.

In active hyperæmia the velocity of the blood-current is usually increased. The affected part is bright red in color, swollen, and, if superficial, warmer than normal. Active hyperæmia is usually an acute, transitory condition.

Passive hyperæmia is due to some obstruction to the flow of blood in the veins. From the nature of the obstruction it is also called mechanical hyperæmia and venous hyperæmia, inasmuch as it is venous blood which accumulates in the parts. Passive hyperæmia is frequently a chronic condition. The abnormal hindrances may be either within or without the veins. Thrombi are the most frequent obstacles within the veins. General venous hyperæmia follows obstruction to the flow of blood through the heart. The veins may be compressed from without by clothing, bandages, tumors, exudations, newly-formed fibrous tissue, etc.

By *hypostasis* or *hypostatic congestion* is understood venous hyperæmia of dependent parts of the body under the influence of gravity and of enfeebled heart's action. In most parts of the body the anastomoses between the veins are so many that a single vein, or even several, may be obstructed without serious disturbance of the circulation. Occlusion of the portal vein cannot be compensated for thus by collateral circulation. Venous hyperæmia of the lower extremity also follows thrombosis of the femoral vein, inasmuch as the arrangement of the valves in the anastomosing veins does not permit the formation of a sufficient collateral circulation.

In passive hyperæmia the blood is dammed back upon the veins and capillaries of the part, the velocity of the circulation is lessened, the vessels become over-distended with blood, frequently a transudation of serum and a diapedesis of red blood-corpuscles ensue, and an increased amount of lymph flows from the obstructed region. The affected part is usually bluish-red in color, swollen, frequently œdematous, and, if exposed to the air, cooler than normal. The diagnosis of hyperæmia cannot always be made upon post-mortem examination, as the distribution of blood may vary greatly from that present during life. This is particularly true of active hyperæmia.

Hemorrhage.

Hemorrhage is the escape of blood through the walls of the vessels or of the heart. When the extravasation is through the ruptured wall of a vessel, it is called hemorrhage by *rhexis;* when the red blood-corpuscles are pressed through the unruptured vascular wall, it is denominated hemorrhage by *diapedesis*, or simply diapedesis. When the hemorrhages are minute, they are called *petechiæ* or *ecchymoses;* when the blood infiltrates a circumscribed part of the tissues uniformly without tearing them, the extravasation is a *hemorrhagic infarction;* when the escaped blood forms a tumor, it is called a *hæmatoma.*

According to the source of the blood, hemorrhages are classified as cardiac, arterial, capillary, and venous. The causes of hemorrhage by rhexis are various, and the consideration of many of them belongs to the domain of surgery. Rupture of healthy vascular walls is usually due to traumatism or to local elevation of the blood-pressure. Newly-formed blood-vessels, such as those in granulation-tissue and in tumors, easily rupture in consequence of the imperfect development of their coats. Of the diseases which weaken the walls of the vessels and favor their rupture, the most important are aneurism, atheroma, ulcerative processes acting from without, infiltration of the vascular walls by new growths or inflammatory products, and fatty degeneration. Elevation of the general blood-pressure probably never causes rupture of healthy vessels, but it may contribute to the giving way of those which are diseased.

Hemorrhage by diapedesis occurs in venous hyperæmia, in inflammation, in hemorrhagic infarctions, and in districts in which the circulation of blood has been temporarily arrested for many hours. In these cases it is probably due to nutritive changes in the vascular walls, which are thereby rendered more permeable. These assumed changes, however, cannot be recognized by our present means of investigation. The red blood-corpuscles pass through the walls of the veins, and especially of the capillaries, making their way through the cement-substance between the endothelial cells. Diapedesis is a passive process as far as the red corpuscles are concerned. Hemorrhages by diapedesis are generally small, but exceptionally they are considerable. It is not always possible to determine whether an extravasation is the result of rhexis or of diapedesis.

Many diseases are accompanied by a hemorrhagic tendency, such as purpura, scurvy, phosphorus-poisoning, leucocythæmia, pernicious anæmia, and a number of infectious diseases, as septicæmia, yellow fever, smallpox, and malignant endocarditis. The hemorrhage in many of these cases is doubtless due to a weakened condition of the vessel-walls in consequence of the disordered composition of the blood; but whether the blood escapes by rhexis or by diapedesis has not been established in the majority of instances. In some septic diseases attended by capillary hemorrhages, such as malignant endocarditis, hemorrhagic smallpox, and hæmophilia neonatorum, blood-vessels plugged with colonies of micrococci have been found in the ecchymosed districts; but in many similar cases no relation could be demonstrated between the hemorrhages and the presence of bacteria.

The changes which take place in a hemorrhagic extravasation lead to the gradual absorption of most of its constituents. The fluid parts are absorbed; the fibrin becomes granular and is taken up; the white blood-corpuscles, in part, wander into the tissues and the absorbents, but in greater part disintegrate and are absorbed. Some of the red corpuscles are carried away by the lymphatics, others remain and undergo pigmentary transformations. The formation of pigment takes place, in great part, in wandering cells, which take up the corpuscles or fragments of them. The resulting granular and crystalline hæmatoidin pigment may remain in the cells or be set free. Thus, only pigment may remain to tell of the former extravasation, and even this pigment may be in time absorbed. These changes may be complicated by acute and chronic inflammatory processes. The formation of so-called apoplectic cysts in the brain will be described in treating of cerebral hemorrhage.

Thrombosis and Embolism.

A *thrombus* is a coagulum formed during life in the heart or in the vessels. A thrombus is designated as *occluding* when it completely fills the interior of the vessel, and as *parietal* when it only partially obstructs the vessel to the wall of which it is attached.

The formation of the thrombus is the result of some change in the vascular walls. The normal condition of the endothelial lining of the vessels is essential to the preservation of the fluid state of the blood. Structural changes which impair the endothelium are causes of thrombosis. The most important of these changes are inflammation, atheroma, calcification, degenerations, tumors, injury, and compression of the vessels. Retardation of the circulation is a most important cause of thrombosis. It acts also by impairing the nutrition of the vascular endothelium. Thus are explained the thrombi formed in aneurisms and in varices. The so-called *marantic* thrombi are the result of great weakening of the circulation from extreme debility and from much-enfeebled heart's action. The veins of the lower extremities and of the pelvis, the appendix of the right auricle and the apex of the right ventricle, and, in children, the cerebral sinuses, are favorite seats of marantic thrombi. They often begin to form in the pockets of the valves in the veins of the lower extremities. A thrombus usually extends at least from the point of its formation to the nearest branch given off from the vessel; it may extend farther forward and backward, and may grow into the collateral branches.

A thrombus is composed of fibrin and blood-corpuscles. It is now generally believed that fibrin is the result of some reaction between fibrinogen found in solution in plasmatic fluids and one or more substances produced by the destruction of protoplasmatic elements. In the mammalian blood the protoplasmatic elements which come into consideration are the leucocytes and the minute bodies known as blood-plates. According to the popular theory of Schmidt, the leucocytes play the chief rôle and furnish fibrino-plastin and fibrin ferment. Of these, fibrin ferment is the most important, if not the sole active element. That the blood-plates have an important share in the formation of thrombi seems to be established.

Thrombi vary in structure according to their formation from blood in motion or at rest. *Red*, *white*, and *mixed* thrombi are thus distinguished. Red thrombi are formed from blood at rest—as, for instance, in a vessel enclosed between two ligatures—and are composed of red and white corpuscles and fibrin combined in the same proportions as in a blood-clot outside of the body. Most thrombi are formed from the blood in motion. Such thrombi are grayish or reddish-gray in color. Zahn, who studied experimentally the production of these thrombi in the mesenteric vessels of frogs, found that they were caused by an accumulation of white blood-corpuscles, and his conclusions have been universally accepted. It has, however, recently been shown by those who have observed the process in the vessels of warm-blooded animals that the white thrombi are produced by an accumulation of blood-plates.[1] Recent white thrombi, when examined microscopically, are found to consist of fibrin, much granular material, and a variable number of intact red and white blood-corpuscles. The granular material, according to the older view, is the result of the disintegration of leucocytes, but according to a later view it consists of blood-plates or their fragments.

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[1] Bizzozero, Lubnitzky, Eberth and Schimmelbusch, and Osler.

It is of great practical importance to be able to distinguish between thrombi and decolorized post-mortem clots, particularly those which are found in the cavities of the heart, and which are so often erroneously assigned by the inexperienced as the cause of death. Thrombi are opaque, granular in appearance, distinctly stratified, and adherent to the wall of the vessels. Decolorized post-mortem clots consist of a lower, red cruor mass and an upper yellowish-white fibrinous material; they have a gelatinous, moist, translucent appearance, are not distinctly stratified, and are not intimately adherent to the vascular wall.

A thrombus after its formation undergoes *organization*, *softening*, or *calcification.* The so-called organization of a thrombus results in the formation of vascularized connective tissue in the place of the thrombus. The thrombus itself breaks down and is absorbed, taking no part in the formation of the new tissue. The process of organization is essentially an obliterating endarteritis, the new connective tissue being derived either from fixed cells in the walls of the vessel (endothelium), or from wandering cells which have escaped by emigration from the vasa vasorum or the surrounding tissues. The new blood-vessels communicate with the vasa vasorum and also with the lumen of the occluded vessel. Partly by the latter communication, but chiefly by the contraction of the new connective tissue, canals may be formed which connect the interior of the vessel on the peripheral side of the thrombus with that on the central side. By this so-called canalization of the thrombus, the circulation may be re-established nearly in its old channels.

It is important to distinguish two kinds of softening of a thrombus. The one is *simple*, or *bland softening;* the other is *mycotic*, or *septic softening.* In simple softening the central part of the thrombus breaks down into a grayish pulp which may be more or less stained with altered blood-pigment, and which in its gross appearance resembles pus. This pulp consists of fatty and albuminous granules, fatty degenerated leucocytes, and blood-pigment. This form of softening is particularly common in the globular thrombi of the heart, which are thereby made to resemble abscesses or cysts with purulent contents. In consequence of the softening, fragments of the thrombus are easily broken off, and are transported by the blood-current as emboli, which in this case are of a bland nature. Far more serious is septic softening. This occurs in infectious thrombi. Here the thrombus and the softened mass contain bacteria, chiefly micrococci. Portions of the thrombus or of the softened material entering the circulation form infectious emboli, which produce the most disastrous results, causing, wherever they lodge, metastatic abscesses. It is these mycotic thrombi and emboli which cause the most important lesions of pyæmia.

Sometimes thrombi, particularly those in varices and in the pelvic veins, shrink and become impregnated with calcareous salts, forming concretions called phleboliths.

The effects and symptoms of thrombosis, in the first place, depend upon the *mechanical obstruction* to the circulation. The degree of this obstruction depends upon the situation and the extent of the thrombus. A parietal thrombus may produce so little obstruction as to give rise to no symptoms. Completely-occluding thrombi produce no mechanical hindrance when lodged in vessels provided with anastomoses which afford a sufficient collateral circulation. Thus a thrombus in one of the venæ comites of an artery forms no apparent obstacle to the circulation. On the other hand, thrombosis of the portal vein (pyle-thrombosis) or of the femoral vein (phlegmasia alba dolens) is followed by well-marked symptoms of venous obstruction. These symptoms of venous thrombosis are passive hyperæmia, with more or less transudation of serum, and diapedesis of red blood-corpuscles. The mechanical effects of arterial thrombi are anæmia of the part supplied by the artery, and necrosis with or without hemorrhage. These effects are usually absent when the thrombus is seated in an artery the branches of which communicate freely by anastomosis. As these mechanical effects of arterial thrombi are the same as those of emboli, they will be more fully considered presently while treating of the latter. On account of their slow formation, a sufficient collateral circulation may be developed to render thrombi comparatively harmless in situations where emboli produce their characteristic effects.

Independently of their mechanical action, thrombi may be injurious by containing some irritative *infectious* principle. Such thrombi contain micro-organisms and cause suppuration and sometimes necrosis in the vascular wall and surrounding tissues.

An *embolus* is a plug of some material transported by the blood-current from one situation to another. The term *embolism* is applied to the process of obstruction of a blood-vessel by an embolus and to the disturbances resulting therefrom. An embolus may consist of any substance which makes its way into the circulation. The majority of emboli are the severed fragments of thrombi. But emboli may consist also of bacteria or other parasites, of bits of a tumor, of fragments of diseased cardiac valves, of concretions of lime, of clumps of pigment, of oil-globules, or of bubbles of air.

It is plain that an embolus can hardly be arrested in its course through the veins, with the exception of the vena portæ, or in the cavities of the heart, since the course of the blood-current in them is from smaller to larger vessels. In rare instances, however, when there is some obstruction to the flow of blood, a venous embolus may take a retrograde course. Thus, an embolus derived from a thrombus in one of the veins of the lower extremities may lodge in one of the renal veins, its onward passage through the inferior vena cava being impeded; as, for instance, by the obstruction to the return of blood to the heart in a severe attack of coughing (Von Recklinghausen). In general, however, embolism relates to the arteries and the vena portæ, while a thrombus may be formed anywhere in the vascular tract.

Disturbances of two kinds attend the lodgment of emboli. The one kind of disturbance is due to the mechanical obstruction to the circulation; the other kind of disturbance attends only infectious emboli, and is dependent upon the presence of some poisonous principles, usually bacteria, in the embolus.

While all arteries of the body are open for the reception of emboli, it is noteworthy that in certain situations the obstruction of an artery by a bland embolus is absolutely harmless, whereas in other parts it is followed by characteristic structural and functional changes. The main condition upon which this difference depends is the character of the arterial distribution in the various parts of the body. When an embolus lodges in an artery supplied with abundant anastomoses—for instance, a muscular artery or one of the arteries of the extremities—a collateral circulation is usually established, which prevents the part from suffering in its nutrition. The effect is widely different if insufficient anastomosis, or none whatever, exist between the occluded artery and other arteries. In certain organs and parts of the body the branches of the arteries do not anastomose with each other, communications existing only between the capillaries and between the veins. These arteries without anastomoses are called by Cohnheim terminal arteries. Such arteries are the pulmonary, the renal, the splenic, the arteries beyond the circle of Willis supplying the basal region of the brain, the central artery of the retina, and the coronary arteries of the heart. The branches of the vena portæ also form no anastomoses with each other.

When an embolus lodges in an artery where no sufficient collateral circulation can be established, the part supplied by the obstructed artery is cut off from its blood-supply and dies. The mode of death is that designated as anæmic necrosis or coagulation necrosis. The necrotic mass of tissue constitutes an *embolic infarction*. In some situations there is usually an extravasation of blood into the necrotic part. The infarction is then spoken of as *hemorrhagic;* when there is no hemorrhage the infarction is called a *white* or *anæmic infarction*. The essential, because the constant, change in embolic infarctions is not the hemorrhage, but the necrosis.

The coagulation necrosis of embolic infarctions is characterized by a disappearance of the nuclei of the cells and by a coagulation of their protoplasm into a substance resembling fibrin. The affected tissue is rendered hard in consistence. In the brain, however, there is so little protoplasm that no appreciable coagulation can take place, so that the effect of embolism of a terminal artery of the brain is softening instead of hardening of the tissues.

In hemorrhagic infarctions the blood is extravasated by diapedesis. Blood is sent from the surrounding capillaries or from minute arterial twigs into the capillaries of the obstructed district, but not with sufficient force to propel the blood with normal rapidity into the veins or to nourish the part. As a result of this insufficient supply of fresh arterial blood the walls of the capillaries and small veins suffer in their nutrition and allow a diapedesis of red blood-corpuscles. Another possible source of the extravasted blood is a regurgitation of the blood from the veins which connect with the capillaries, the arterial supply of which is shut off. As the blood-pressure on the peripheral side of the obstruction is reduced to nothing or almost nothing, there is apparently no reason why this backward flow of blood should not take place. It has, however, been shown by experiments of Litten that a return flow of blood from the veins is not essential to the production of a hemorrhagic infarction.

It is not very clear why some infarctions are hemorrhagic and others are white. Probably, as suggested by Weigert, much depends upon the character of the affected tissue. Where the tissue is lax, containing wide spaces, there is plenty of room for the infiltration of blood. Hence in the lungs infarctions are always hemorrhagic. In the kidney, on the other hand, the occurrence of coagulation necrosis renders the tissue so dense that the blood cannot penetrate into the interior of the infarction. Hence infarctions of the kidney are always white in the centre, usually with a hemorrhagic margin. The consistence of the spleen after the occurrence of coagulation necrosis is such as to admit of the infarction being either white or hemorrhagic, according to the force of the circulation.

The organs in which embolic infarctions occur are the kidney, spleen, lungs, brain, heart, retina, intestine, stomach, and testicle. Emboli in branches of the portal vein do not produce infarctions, as the hepatic artery can supply the lobular capillaries. Infarctions are generally wedge-shaped (not so in the brain), corresponding to the area of distribution of the obstructed artery. The base of the wedge is toward or usually at the periphery of the organ. A hyperæmic zone containing many emigrated white blood-corpuscles is found around the borders of the infarction. This reactive inflammation results in the formation of new connective tissue, which takes the place of the infarction, many of the constituents of the latter being absorbed. The termination is in a cicatrix of fibrous tissue which contains pigment when the infarction was hemorrhagic. The special characters of embolic infarctions of different organs will be considered under the diseases of these organs in Part II. of this work.

The presence of disease of the arteries and an enfeebled circulation are important factors in determining the effects of an embolus. Thus, embolism of the femoral artery produces no permanent injury when the force of the circulation is normal, but it may be followed by gangrene of the lower extremities if the heart's action be feeble and the anastomosing arteries atheromatous.

Embolism of the main trunk of the pulmonary artery, of certain arteries of the medulla oblongata, or of one of the coronary arteries of the heart causes sudden death. In certain situations and under certain circumstances the ischæmia caused by an embolus is of short duration. Thus, a temporary loss of function disappears when the vessels beyond the seat of obstruction are filled by collateral arterial branches.

In addition to their mechanical effects, *infectious* emboli, such as those coming from the cardiac valves in malignant endocarditis or from thrombi in pyæmia, produce suppurative inflammation wherever they lodge. Thus, even capillary emboli, when infectious, produce abscesses. The multiple abscesses in pyæmia are for the most part of embolic origin.

Fatty emboli and emboli composed of air-bubbles demand a few words of separate consideration.

The source of *fatty emboli* is usually to be found in inflammation or traumatism of parts rich in fat. The most important cause is fracture of the bones accompanied by extensive laceration of the marrow. The oil-globules thus set free enter the open mouths of ruptured veins, and are transported to the pulmonary capillaries, which are often thereby extensively occluded. The fat may pass through the pulmonary capillaries and lodge in the glomeruli of the kidneys, in the capillaries of the brain, and in other capillaries of the body. It has been proven by experiments that fatty embolism, even when very extensive, is generally innocuous.

The sudden entrance of a large quantity of air into the blood-current, such as may occur by incision of the large veins near the heart, has been long known as a cause of rapid death. The fatal termination is due to the accumulation of the air in the right cavities of the heart, the contraction of which is unable to force the elastic air forward. The air remaining in the right auricle and ventricle is an obstacle to the entrance of blood from the venæ cavæ, and arrests the pulmonary and systemic circulations.

Dropsy.

A certain amount of serous fluid, known as the lymph, constantly transudes through the capillary walls into the tissues. When a greater quantity of this fluid transudes than can be absorbed by the lymphatics and blood-vessels, the fluid accumulates in the interstitial spaces and cavities of the body. This condition is called *dropsy*. The term *œdema* is applied to the accumulation of serous fluid in the tissues, particularly in the meshes of connective tissue. Subcutaneous œdema extending over the greater part of the body receives the name of *anasarca*. Dropsies of the serous cavities are designated by prefixing *hydro* to the name of the cavity affected; thus, hydrothorax, hydropericardium, hydro-peritoneum, or ascites, and hydrocephalus. Hydrocele is a serous accumulation in the tunica vaginalis testis. The term *hydrops* also signifies a serous effusion, usually into a cavity. Accumulations of serous fluid in glands and canals, the outlets of which are obstructed—for example, in the Fallopian tube (hydrops tubæ), in the gall-bladder (hydrops cystidis felleæ)—are to be distinguished from real dropsical effusions, although the appearance of the fluids may be similar.

A distinction may be made between transudations and exudations, the term transudation being confined to dropsical effusions, while exudation is applied to inflammatory products. The two terms are, however, often used synonymously.

Dropsies may be divided into three classes—*inflammatory*, *mechanical*, and *cachectic* or *hydræmic*.

Inflammatory dropsy is due to a moderate degree of inflammatory alteration in the coats of the blood-vessels. The so-called *collateral œdema*, often found in the neighborhood of inflammatory infiltrations and of tumors, is an inflammatory œdema. Of the same nature are most cases of œdema glottidis, of hydrocele, and of internal hydrocephalus.

Mechanical dropsy is the result of some obstruction to the current of blood in the veins. Mechanical dropsies may be general or local. The dropsy is general when, in consequence of disease of the heart or of the lungs, the return flow of blood from the venæ cavæ is impeded. The most frequent of the mechanical causes of general dropsy is valvular disease of the heart. General dropsy is characterized by anasarca and transudation into the serous cavities. Examples of local dropsies due to mechanical causes are the hydroperitoneum resulting from obstruction of the vena portæ, as by thrombosis or by cirrhosis of the liver, and the œdema of one of the extremities in consequence of thrombosis or compression of its veins.

It might be supposed that obstacles to the flow of lymph in the lymphatic vessels would cause an abnormal accumulation of lymph in the tissues and serous sacs. But it has been found that not only the peripheral lymphatics, but even the thoracic duct, may be completely occluded without producing local or general dropsy. The lymph does not accumulate under these circumstances, because relief is afforded by collateral lymphatic channels, and because the blood-vessels act as absorbents when the lymphatics are occluded.

Hydræmic or cachectic dropsy is the result of an impoverished and abnormally watery state of the blood. This state of the blood, which is called hydræmia, appears, however, to be the indirect and not the immediate cause of the dropsy. The experiments of Cohnheim and Lichtheim have shown that when the vascular walls are healthy, artificial hydræmia, produced by the injection of large quantities of dilute solution of common salt into the blood-vessels of animals, causes no increased transudation in the situations in which dropsy occurs in man. It is believed that the hydræmia acts in the causation of dropsy by inducing in the vascular walls some change which renders them more permeable. The most important cause of hydræmic dropsy is Bright's disease. This form of dropsy, generally in a lesser degree, may attend many other cachectic conditions, such as tuberculosis, cancer, malarial cachexia, and chronic dysentery. Cachectic dropsy is prone to appear in dependent parts of the body, and where the connective tissue is particularly lax in texture, as in the eyelids and about the genitals.

Dropsical effusions are generally clear, transparent, colorless or slightly yellow, and alkaline in reaction. They may be stained by an admixture of blood or of biliary coloring matter. They contain the same salts as the plasma of the blood, and in about the same proportions. The amount of albumen varies between 0.4 and 5 per cent., being less than that present in the blood-plasma. Under like conditions the percentage of albumen varies with the situation of the transudation in the following order: tunica vaginalis testis, pleura, pericardium, peritoneum, subcutaneous tissue, subarachnoid spaces, the percentage being highest in hydrocele and lowest in subarachnoid œdema. Of the different forms of dropsy, the amount of albumen is greatest in the inflammatory variety and lowest in cachectic dropsy. Dropsical effusions differ from inflammatory exudations in the smaller amount of albumen present, in the paucity of cells, and in the absence or small quantity of fibrin present. Most transudations contain fibrinogen; hence dropsical transudations often coagulate spontaneously when withdrawn from the body, in case fibrin ferment by the disintegration of cells or by admixture with blood is set free. Examined microscopically, serous transudations are found to contain a few leucocytes, and also red blood-corpuscles, the latter sometimes in abundance. In rare instances transudations in the pleural and in the peritoneal cavities have been rendered milky in appearance by an admixture of chyle derived from the chyle-vessels, particularly the thoracic duct, in consequence of their occlusion or rupture. Such a transudation is called hydrops chylosus.

CHAPTER II.

INFLAMMATION.

ACCORDING to the classical definition of inflammation, a part is acutely inflamed when it is hot, red, swollen, and painful. But this enumeration of the four cardinal symptoms (calor, rubor, tumor, dolor) gives no information as to the pathological nature of the process. No topic in medicine has been the subject of so much research and speculation as the nature of inflammation, but even at the present time it is impossible to give a complete and correct definition of inflammation from a pathological or an etiological standpoint. From the earliest times two opposing theories regarding the process of inflammation have been held. According to the one, the essential phenomena in inflammation are referable to the blood and to the blood-vessels; according to the other, the essential changes are in the solid tissues outside of the blood-vessels. A great advocate of the former theory was John Hunter. Recently it has been developed especially by Cohnheim. Virchow, on the other hand, rejecting the Hunterian doctrine, maintained that the primary and chief effect of an inflammatory irritant is the excitation of the cells of a part to increased functional and nutritive activity, and that hyperæmia and fluid exudation from the blood are secondary to this excitation. When Virchow made his researches on inflammation, nothing was generally known concerning the emigration of white blood-corpuscles or their wandering powers; and he naturally concluded, in the application of the law *omnis cellula e cellula*, that all cell-elements present in inflammatory exudation were produced by proliferation from the pre-existing fixed cells, most frequently from the connective-tissue cells. The discovery by Recklinghausen, in 1863, of the existence of wandering cells, resembling pus-cells, in the tissues, rendered doubtful the interpretation which Virchow has given to many of his observations. A new era was introduced in the history of inflammation by Cohnheim's discovery, in 1867, of the emigration of white blood-corpuscles from the vessels, and by his studies of the inflammatory process on living frogs. It is true that as long ago as 1846, Waller, an English observer, saw the passage of white blood-corpuscles through the unruptured vessel-walls, but this isolated observation had remained fruitless, and had fallen into oblivion when Cohnheim repeated the discovery.

The phenomena of inflammation were observed first by Cohnheim microscopically upon the mesentery and tongue of the curarized frog. The first effect of the application of an inflammatory irritant (the mere exposure of the mesentery to the air suffices) is a dilatation of the arteries, then of the veins, and, last of all, of the capillaries. At the same time the velocity of the blood-current is increased. After a variable time the blood begins to flow less rapidly, although the calibre of the vessels remains unchanged. In some of the capillaries it may come to complete stagnation, or stasis, but this is not essential or usual except when the action of the irritant is intense. As the rapidity of the blood-current lessens, the white blood-corpuscles accumulate along the inner surface of the veins, where they remain nearly stationary. The remarkable phenomenon of emigration now takes place. A portion of a white blood-corpuscle soon appears upon the outer surface of a vein or a capillary, as a bud-like process connected by a delicate thread of protoplasm with the remnant of the corpuscle inside. As the outer portion of the cell increases in size the inner diminishes, until, finally, the entire corpuscle lies free outside of the vessel. The corpuscle passes through the cement-substance between the endothelial cells lining the vessel. It is not decided whether the corpuscles make their way through the vascular walls by their active amœboid movements, as the name emigration would imply, or whether they are pressed through by a process of filtration. Outside of the vessels they assume amœboid movements and change their shape and place. While the inner surface of the veins becomes plastered, as it were, with stationary white blood-corpuscles, the current of red blood-corpuscles continues unabated in the central part. White blood-corpuscles migrate from the veins and the capillaries, but not from the arteries. Red blood-corpuscles also pass through the capillary walls by the passive process of diapedesis. Their number is not usually great, but exceptionally it may be so considerable as to give a hemorrhagic character to the exudation. Coincidently with the processes of emigration and of diapedesis the fluid constituents of the blood filter through the walls of the vessels. This fluid exudation resembles in its composition the plasma of the blood, but it contains less albumen. It contains the fibrin-generators, and in most places finds the conditions necessary for the spontaneous coagulation of fibrin. A fibrinous effusion is, in the vast majority of cases, an inflammatory exudation. In simple inflammations of mucous membranes and in suppurative inflammations (abscesses) no fibrin is formed. By a process of reasoning which cannot be entered into here Cohnheim concludes that these various phenomena of inflammation can be explained only upon the assumption that the effect of the inflammatory irritant is to produce a molecular alteration in the walls of the vessels, which increases their permeability to the fluid and corpuscular elements of the blood and increases the friction between the blood and the vessel-wall.

While the accuracy of Cohnheim's observations of the process of inflammation in frogs has been established, and their applicability to the same process in man has been recognized, there has been much controversy as to the part taken by the fixed cells outside of the vessels in the inflammatory process. Do they produce pus-cells, as Virchow taught? It is maintained by Cohnheim that the sole origin of pus-cells is to be found in emigration of the white blood-corpuscles, and that the fixed cells in inflammation either remain unchanged or degenerate, or proliferate and produce other cells of their kind (regenerative process), but never give rise to pus-cells. On the other hand, Stricker and his school hold to the original doctrine of Virchow, that all varieties of fixed cells—connective-tissue cells, epithelial cells, nerve-cells, muscle-cells—proliferate in inflammation and produce pus-cells. The difficulty in determining these points at issue lies chiefly in the fact that it has not yet been found possible to observe satisfactorily the process of change in the living fixed cells during inflammation; so that we are compelled to infer the nature of the process from a study of the phases, the interpretation of which is often various and open to fallacies. Especially is this true of the attempt to interpret the significance of phases apparently transitional between one form of cell and another, and to judge of the origin of a cell from its form. Hence the evidence that fixed cells produce pus-cells is of a different nature and less convincing than the proof that pus-cells come from emigrated white blood-corpuscles. It is impossible to describe here the many experiments and observations made with reference to the settlement of this question. It must suffice to state that the weight of evidence is decidedly in favor of the view that fixed cells do not remain passive in inflamed parts, but that they become amœboid, proliferate, and produce young cells. Unless we arbitrarily restrict our definition of pus, these young cells are to be regarded as pus-cells, which may therefore be derived from either emigrated white blood-corpuscles or from fixed cells. The dominating elements in acute inflammation, however, are the emigration of white blood-corpuscles and the exudation of the fluid constituents of the blood in consequence of as yet undefined changes in the vascular walls.

As is evident from the foregoing description, the *products* of inflammation are fibrin, serum, pus-cells, and red blood-corpuscles. In inflammation of mucous membranes an increased secretion of mucus may take place. Exudations are classified as *fibrinous*, *serous*, *purulent*, *hemorrhagic*, and *mucous*, according to the relative proportion of the inflammatory products which they contain. *Fibrinous* exudation is best illustrated by the acute inflammations of serous membranes, where the fibrin is deposited as a layer upon the serous surfaces. These fibrinous layers can be readily stripped off, and are sometimes called *false membranes* or *coagulable lymph*. More or less white and red corpuscles are present in the meshes of the fibrin, and in the interior of the serous sac is a variable amount of fluid containing also fibrin and exuded cell-elements. Fibrinous exudation may be present also in inflammation of the solid tissues, in the air-cells of the lung, and upon mucous membranes. Fibrinous exudation upon mucous surfaces is called croupous or diphtheritic in accordance with a distinction to be explained hereafter. *Serous* exudations bear considerable resemblance to transudations, but they are generally richer in albumen and in cells. The main distinction, however, is that they appear with the signs of inflammation. The exudation in so-called collateral œdema (which is really inflammatory) is generally serous; likewise the exudation in the early stage of many inflammations of mucous membranes. Inflammatory affections of the skin are accompanied often by the formation of vesicles containing serous exudation. Although pus-cells are present in all inflammatory exudations, it is only when they are sufficiently abundant to render it opaque that the exudation is said to be *purulent*. Pus consists of a fluid part (liquor puris) and of cells. Pus-cells are round, membraneless, and contain usually two or three nuclei enclosed in a cell-body of protoplasm, in which, as a rule, molecules of fat are present. Pus-cells belong to the widely-distributed group of cells known variously as leucocytes, lymphoid cells, wandering cells, embryonic cells, and small, round, indifferent cells. Their origin from emigrated white blood-corpuscles has been described. It is believed that pus-cells increase in number by division. Inflammations producing pus are suppurative. The pus may be exuded upon free surfaces; it may infiltrate the tissues (purulent infiltration), or it may be contained in cavities produced by the breaking down of tissue. Such cavities containing pus are called *abscesses*. It is probable that suppurative inflammations are due to the action of bacteria. When the exuded red blood-corpuscles are sufficiently abundant to give a red color to the exudation, the inflammation is said to be *hemorrhagic*. According to Cohnheim, hemorrhagic exudations are referable to an intense action of the inflammatory irritant upon the vascular walls. Tuberculous, cancerous, variolous, and scorbutic inflammations of serous membranes are liable to be hemorrhagic. The source of the hemorrhage may be from rupture of the vessels or from diapedesis.

As has already been mentioned, the terms *croupous* and *diphtheritic* are applied to fibrinous exudations upon mucous membranes. Unlike the inflammations of serous membranes, the ordinary inflammations of mucous membranes are not accompanied by a fibrinous exudation. The term *catarrhal* is sometimes applied to these simple inflammations of mucous membranes characterized by an exudation of serum, mucus, and some pus-cells. According to the careful investigations of Weigert, fibrin is present in the inflammations of mucous membranes only when the epithelial covering is partly or wholly destroyed. The epithelium may be destroyed from various causes, among the most important of which is coagulation necrosis. The necrosis may extend deeper than the epithelium into the subjacent tissues. When only the epithelium is destroyed, the fibrinous exudation lies upon the membrana propria of the mucous membrane, from which it can be readily stripped off without loss of substance. This form of exudation is called *croupous*. When the primary necrosis involves the tissue-cells as well as the epithelium, the fibrinous exudation extends from the surface into the tissue of the mucous membrane and cannot be removed without loss of substance. This second form of exudation is denominated *diphtheritic*. It is to be observed that croupous and diphtheritic exudations require destruction of the epithelium only in one place, and that they may extend thence over the surface of the surrounding intact epithelium. The fibrin in croupous and diphtheritic inflammations is derived partly from the blood, partly from metamorphosis of the epithelial and other cells, and perhaps partly from fibrinoid degeneration of the intercellular substance (Neumann). (Further details concerning these varieties of inflammation will be found in Part II. of this work, in connection with croup and diphtheria.) It is to be noted that the terms croupous inflammation and diphtheritic inflammation are used in a purely anatomical sense, without reference to the diseases called croup and diphtheria.

The term *parenchymatous inflammation* was introduced by Virchow to indicate the origin of the inflammatory process in the parenchymatous cells of an organ. These—for example, the hepatic cells—under the influence of an irritant swell up, become more granular than normal, proliferate or more frequently undergo fatty degeneration, and perhaps return to their healthy condition. The propriety of regarding these changes as inflammatory is at least doubtful. Most of them are also embraced under the names albuminous or parenchymatous degeneration. Many writers, however, still make use of the expression parenchymatous inflammation, especially with reference to the kidney, liver, and spinal cord.

The terminations of inflammation may be various. A complete restoration of an inflamed part to its normal condition is called *resolution*. This, as a rule, occurs only in the milder grades of inflammation. The fluid part of the exudation can be readily absorbed; emigrated white blood-corpuscles may re-enter the blood-vessels or may wander into the lymphatics, or they, as well as red blood-corpuscles and fibrin, may undergo fatty degeneration and then be absorbed. The formation of new connective tissue, or the *organization* of the exudation, as it is called, is a frequent termination of some forms of inflammation, particularly of the fibrinous exudation on serous membranes. Such organizing exudations are sometimes called *plastic* or *adhesive*. Fibrinous exudations in the bronchi are sometimes called plastic (plastic bronchitis), although no connective tissue is formed. It is certain that the fibrin is absorbed, taking no active part in the development of new tissue. The new connective-tissue cells may spring from emigrated white blood-corpuscles or from the pre-existing connective-tissue cells (including endothelial cells). The new tissue is at first very rich in capillaries, derived from the old capillaries, and in cells. Subsequently it becomes more fibrous, less vascular, and it contracts. Considerable accumulations of pus cannot be absorbed, but when not evacuated they either remain without changing their consistence or become inspissated. Such dried masses of pus may undergo cheesy and calcareous transformations, and, becoming encapsulated, remain innocuous.

Chronic inflammations may be secondary to the acute or subacute from their origin. In the latter case they may be unattended by any free exudation. Chronic inflammation most frequently results in the formation of new connective tissue.

CHAPTER III.

ACTIVE ALTERATIONS OF THE TISSUES.

Pathological New Formations—Regeneration—Hypertrophy—Tumors— Carcinoma.

Pathological New Formations.

THE active alterations of the tissues embrace the pathological new formations. These will be grouped under the headings *regeneration*, *hypertrophy*, and *tumors*.

The pathological new formations are the result of increased activity of cells which are incited to abnormal growth and proliferation. The usual process of cell-proliferation is that known as indirect cell-division. This is characterized by so-called nuclear figures, which follow each other in definite order (karyomitosis). These figures are produced by peculiar arrangements of little threads of nuclear substance which can be colored by various staining dyes while the substance between the threads remains unstained. Thus are formed the so-called coil, wreath, star, æquatorial plate, and spindle figures. Out of the mother nucleus are produced two daughter nuclei. This is accompanied by division of the cell-body, each segment surrounding one of the nuclei. (For the details of this process of cell-division, as well as of other possible modes of cell-production, works on normal histology may be consulted.)

The doctrine of the so-called specialization of cells bears upon pathological new growths. Embryological researches have shown that, after the formation of the three blastodermic layers, the cells in each produce only certain kinds of cells and tissues—that, for instance, connective tissue and muscle can spring only from the middle blastodermic layer, and epithelium only from the external and internal blastodermic layers (part of the genito-urinary epithelium possibly excepted). There is much which supports the view that under pathological conditions definite cells can be reproduced only by their kind—that, for example, new epithelial cells are always the offspring of pre-existing epithelium, and cannot be developed out of connective-tissue cells or white blood-corpuscles. This view is best established for epithelial and nervous tissue. Connective-tissue cells, however, can be produced apparently by emigrated white blood-corpuscles as well as by pre-existing connective-tissue cells.

Regeneration.

The subject of the regeneration of tissues after their destruction by wounds and pathological processes is treated of *in extenso* in works on surgical pathology and will be considered here only in outline. Some structures are reproduced with great ease, as, for example, connective tissue and epithelium; others with more difficulty, as muscular tissue and peripheral nerve-fibres; and, finally, others not at all, such as the central nervous system and the parenchyma of most organs.

Fibrillated connective tissue is present in nearly all pathological formations. It may be produced slowly out of the pre-existing connective tissue or it may be formed out of *germinal* or *granulation-tissue*, as it is called. This granulation-tissue is composed chiefly of cells with scanty and ill-defined intercellular substance and with blood-vessels without compact walls. The cells are of various shapes—round, epithelioid, fusiform, and branched. The granulation-cells may be derived from emigrated white blood-corpuscles or from proliferated connective-tissue cells. The round cells become epithelioid and spindle-shaped, and finally assume the various forms characteristic of connective-tissue cells; the intercellular substance increases in amount, probably at the expense of the cells, and becomes distinctly fibrillated, and the cells which constitute the embryonic walls of the capillaries change into flat endothelial cells. The fully-formed fibrous tissue is not so rich as the granulation-tissue in cells and blood-vessels, some of which, therefore, are destroyed in the process of development.

New-formed blood-vessels are produced by solid protoplasmatic offshoots from the walls of pre-existing capillaries. These offshoots unite with other similar offshoots and with the walls of existing capillaries. These prolongations, at first solid, subsequently become hollowed out in their centre, the cavity thus formed communicating with the lumen of the capillary vessels and receiving blood from them. At the same time nuclei develop, and the protoplasm divides around the nuclei into cells which now represent the endothelium of the newly-formed capillaries. By additions to their coats of connective tissue and of smooth muscular fibres, and by widening of their lumina, these capillaries may change into arteries and veins.

Among the circumstances under which connective tissue and blood-vessels are formed in the manner described may be mentioned the repair of wounds, the healing of ulcers, and the development of adhesions after inflammation of serous membranes. The regenerative changes frequently proceed side by side with inflammatory alterations, from which they cannot usually be clearly separated.

Epithelium is endowed with an especially active power of regeneration. It has been experimentally demonstrated[1] that incised wounds of the cornea may be completely filled by newly-developed epithelial cells within twenty-four hours. Nearly all recent observations go to prove that the new epithelium is always the offspring of the pre-existing epithelial cells.

Regeneration of *striated muscular fibres* occurs after their degeneration in fevers, particularly typhoid fever, and in some other diseases, and sometimes after their destruction by wounds, especially subcutaneous wounds. Defects in muscular tissue, produced by wounds, however, are more frequently filled by cicatricial connective tissue. It is probable that the new muscular tissue is produced only from the old muscular tissue by proliferation of the muscle-corpuscles. Smooth muscular tissue can be produced apparently either from pre-existing smooth muscle-fibre cells or from connective-tissue cells.

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[1] Von Wyss, *Virchow's Archiv*, Bd. 69, 1877.

The investigations concerning the regeneration of *peripheral nerve-fibres* have been many, but have not led to entire unanimity of opinion. Section of peripheral nerve-fibres is followed by their rapid degeneration, extending from the point of section to their peripheral terminations. The degenerated peripheral nerve-fibres are replaced by new axis-cylinders, which grow probably from the intact axis-cylinders in the central portion of the nerve. These axis-cylinders subsequently become invested with myeline and tubular sheaths. Notwithstanding some apparently confirmatory observations, there is no conclusive evidence that nerve-elements are regenerated in the central nervous system of the adult. This is certainly true as regards the reproduction of ganglion-cells.

The consideration of the reproduction of bone and of cartilage, belonging almost exclusively to surgery, cannot be entered into here.

Hypertrophy.

The term hypertrophy is applied to enlargement of a part from an increase of its normal constituents, the structure and arrangement remaining essentially unaltered. Parts which become enlarged in consequence of a deposit of materials foreign to their normal composition, or from a disproportionate excess of certain of their normal constituents, are not, properly speaking, hypertrophied. Two forms of hypertrophy have been distinguished—namely, *simple*, or *true hypertrophy*, and *numerical hypertrophy*, or *hyperplasia*. In simple hypertrophy there is an increase in the size of the anatomical elements, but not in their number; in hyperplasia the number of these elements is augmented. It is not usually practicable to carry out this distinction, as the two forms of hypertrophy are frequently combined. Hypertrophy may proceed from a diminution of the disintegration or waste naturally incident to the life and activity of cells, while the process of assimilation continues in normal force; but it more frequently results from an excess of appropriation, by the part, of nutritive supplies. This excess of nutriment is generally consequent upon exaggerated and prolonged increase of the function of the part. The most frequent examples of hypertrophy are the physiological hypertrophy of the voluntary muscles following persistent exercise, and the pathological hypertrophy of the heart when it is called upon to overcome abnormal obstacles to the circulation, such as are presented by valvular lesions. Hypertrophy of the muscular tunic of the bladder in cases of long-continued urethral obstruction also illustrates the production of this lesion by prolonged increase of function.

Hypertrophy of one of the kidneys usually ensues when the function of the other is either lost or greatly impaired. This is found to be the result of the removal of one of the kidneys in an inferior animal: under these circumstances the task of excreting the urine falls upon a single organ, its functional activity is doubled, and, as a result, the organ becomes enlarged.

The examples of hypertrophy which have been cited are conservative lesions and conduce to the welfare of the body. Microscopical examination in muscular and in glandular hypertrophies reveals an increase in the number, and usually in the size, of the muscular fibres and of the epithelial cells. The increase in amount of the connective tissue of a part which is for a long time hyperæmic, especially when the hyperæmia is due to inflammation, may be referred partially to the augmented supply of nutriment, and thus is to be considered as hypertrophic.

Tumors.

Tumors constitute pre-eminently the pathological new growths. The boundaries of the conception attached to the term tumor cannot be easily defined. Tumor literally means swelling, but not every morbid swelling is a tumor in the technical sense. Particularly to be separated from real tumors are swellings due to inflammatory processes, to œdematous infiltration, and also, though less easily distinguished, those consequent upon hypertrophy. Tumors are composed of anatomical elements similar to those present normally in the body. The terms *homologous* and *heterologous* no longer indicate the presence or absence of elements wholly foreign to the normal organism; but morbid growths are now called homologous when their structure is similar to that of the parts in which they are developed—for example, a fatty tumor in adipose tissue; while the term heterologous is applied to growths differing in structure from the parts in which they are seated—for instance, epithelial tumors in connective tissue.

Tumors are classified and named according to the physiological type of tissue of which they are composed. This basis of classification, proposed by Johannes Müller, has been firmly established by the exhaustive researches of Virchow. We thus distinguish tumors formed after the type of connective tissue in its manifold forms, of epithelium, of muscular tissue, of nervous tissue, and of blood-vessels. Mixed tumors are those in which two or more forms of tumor are combined.

Cysts occupy a special place among the tumors. Many are to be classed rather among the passive than the active alterations of the tissues. Three forms of cysts may be distinguished: *first*, those due to dilatation of pre-existing cavities by liquid accumulations (such are the retention-cysts); *second*, those resulting from the formation of a pathological cavity by the softening and breaking down of tissue; and *third*, those actually of new formation, as the dermoid cysts.

An important clinical division of tumors is into *benign* and *malignant*. The signs of malignancy are the invasion of the surrounding tissues, the property of local recurrence, the formation of metastases, and the development of cachexia. Benign tumors displace, compress, and perhaps lead to atrophy of, the tissues in their neighborhood, whereas those tumors which grow into and invade the surrounding tissues are generally malignant. Tumors which return *in loco* after their removal are frequently, although not necessarily, malignant. Another evidence of malignancy is the development in various parts of the body of secondary or metastatic tumors of the same structure as the primary growth. The metastases are due to the transportation by the blood- or lymph-current of cells from a primary or secondary tumor. The cachexia is the sequel of the tumor, and not a primary dyscrasia. With one exception, it has not been found possible to determine anatomical criteria of malignancy in a tumor. The exception is carcinoma, which is always malignant. From this statement it is not to be inferred that recovery may not take place after complete removal of the tumor. Some forms of sarcoma may prove equally malignant, but malignancy is not a necessary attribute of sarcoma as it is of carcinoma. Of other tumors, a considerable number prove, under exceptional circumstances, malignant.

We possess very little positive information as to the causation of tumors. The question has been much discussed as to the local or the dyscrasic origin of tumors, particularly of malignant tumors, but it now seems to be settled in favor of their local origin. Transmission by inheritance has been shown to be a predisposing influence in the development of some tumors, particularly carcinoma. Local irritation, such as results from injuries, has been often adduced as a cause of the growth of tumors; but this influence of pathological and of physiological irritation has been doubtless exaggerated, and at the most can be regarded only as an exciting cause. Cohnheim has advanced the hypothesis that all true tumors (he separates from these a group denominated infectious tumors, to be mentioned presently) are the result of some abnormality in embryonic development. According to this theory of the embryonic origin of tumors, the germs of the tumor, perhaps consisting of misplaced embryonic cells, are brought by the individual into the world. They may remain dormant for a variable length of time, and then, under the influence of some exciting cause, possibly an injury, may begin to grow. The limits of this work will not permit a consideration of the ingenious arguments by which this hypothesis is supported.

There is a class of tumors for which the name *infectious* has been proposed, which are closely allied to inflammatory new formations, and, in fact, are classified among them by some writers. They are formed after the type of lymphatic or of granulation tissue. It is important to distinguish the infectious tumors from other tumors. They will receive separate consideration in the following chapter. Of the other tumors space will permit hardly more than a mere enumeration. Carcinoma, however, will be considered somewhat more in detail, on account of its frequency and importance.

Tumors in structure analogous to some of the different forms of connective tissue:

1. After the type of embryonic connective tissue—*sarcoma*. Of all varieties of tumor, sarcoma and carcinoma are undoubtedly the most important. Sarcomata are composed mostly of cells, and are classified according to the form of cell which predominates. An important general division is into small-celled and large-celled sarcomata, of which the former are usually the more malignant. The three leading varieties of sarcoma are—*first*, round-celled; *second*, spindle-celled; and *third*, giant-celled or myeloid sarcoma. The cells are separated from each other by a small amount of intercellular substance or stroma, which may be formless or fibrillated. In most of the so-called alveolar sarcomata the cells in the apparent alveoli are not in immediate contact, as in carcinoma, but are separated by a scanty amount of intercellular substance. Where this is not the case the propriety of calling the tumor sarcoma, and not carcinoma, is questionable. Sarcoma is very often combined with other forms of tumor, as in osteo-sarcoma, myxo-sarcoma, glio-sarcoma, etc. Melano-sarcoma is characterized by the presence of black pigment-granules in the cells. Its most frequent point of departure is the choroid coat of the eye and the integument. As sarcoma springs from the universally distributed connective tissue, there is hardly any part of the body from which it may not take its origin. The giant-celled and the ossifying sarcomata generally spring from bone or periosteum. It has already been mentioned that sarcoma embraces both benign and malignant growths. The smaller the size and the larger the number of the cells of the tumor, the more malignant and rapidly growing is it as a rule. Hence the round-celled sarcomata and the melano-sarcomata are the varieties most to be feared.

2. After the type of mucoid tissue—*myxoma*. Myxomata present more or less of a translucent, gelatinous appearance. They spring from connective tissue. They have been observed as multiple tumors growing from the interstitial tissue of the nerves (constituting a variety of false neuromata), but they are generally local. They are benign as a rule. Metastases have been observed.

3. After the type of lymphatic tissue—*lymphoma*, *lymph-adenoma*, *lympho-sarcoma*. Most of the new growths which have been embraced under these

names are either hyperplasiæ, as is the case with the so-called lymphomata of leucocythæmia and pseudo-leucocythæmia, or else they belong to the group of infectious tumors. Genuine tumors, composed of lymphatic tissue, do, however, occur.

4. After the type of neuroglia—*glioma*. The gliomata are confined to the brain, spinal cord, and retina. The most typical gliomata are composed of peculiar branched cells, called spider-cells and brush-cells, similar to those present in the normal neuroglia or connective tissue of the central nervous system. They are often combined with sarcomatous tissue (glio-sarcomata). They frequently appear in the brain, more as infiltrations than as tumors. They do not occasion metastases, but are often the cause of death from their localization in important parts.
5. After the type of fibrillated connective tissue—*fibroma*.
6. After the type of adipose tissue—*lipoma*.
7. After the type of cartilage—*enchondroma*.
8. After the type of bone—*osteoma*.
9. After the type of blood-vessels—*angioma*.

The last five varieties of tumor have clinically, as a rule, a purely local importance. Enchondroma may exceptionally become generalized, and the same is true, in a less degree, of the other forms, except angioma, which never gives rise to metastases.

Certain tumors of peculiar and variable character occurring on the dura mater, pia mater, and serous surfaces, more rarely elsewhere, originate from endothelial cells. They are therefore called *endotheliomata*.

Tumors composed of muscular tissue—myomata:

1. After the type of striated muscle—*rhabdomyoma* or *myoma striocellulare*. Tumors composed of striated muscle-cells are very rare, and are generally congenital. They have been found in the kidneys, testicle, parotid gland, and other parts.
2. After the type of smooth muscle—*leiomyoma* or *myoma lævicellulare*.

The most frequent seat is the uterus, where, combined with more or less fibrous tissue, the myomata constitute the so-called uterine fibroids.

Tumors composed of nervous tissue—neuromata:

It is customary to call all tumors growing from the nerve-trunks neuromata. It is therefore necessary to distinguish between *true* and *false* neuromata. Tumors composed actually of newly-formed nerve-fibres occur, but they are rare, the majority of the so-called neuromata being myxomata and fibromata. According to Klebs, glioma develops from nerve-elements.

Tumors the most important constituent of which is epithelium—papilloma, adenoma, carcinoma including *epithelioma:*

A papilloma is composed of papillæ, often very large and irregular in shape, which consist, like normal papillæ, of vascularized connective tissue covered with epithelium of variable thickness. This tumor is due sometimes apparently to hypertrophy of the papillæ in consequence of chronic inflammation. The soft papillomata of the bladder, rectum, and uterus constitute the most important forms, and often occasion considerable hemorrhage.

An adenoma is composed of glandular tissue, in which the type of the gland can be easily recognized. It always develops from a pre-existing gland. The acini, tubules, or follicles of an adenoma are not completely filled with the epithelial elements, but preserve a lumen. This fact, together with the distinctly-preserved glandular type, distinguishes adenoma from carcinoma. As important examples of this variety of tumor may be cited adenoma of the breast, of the uterus, of the rectum, and of the ovary. The large multilocular ovarian cysts are to be ranked among the adenomata.

Carcinoma.

Pathologists now agree in making the criterion of carcinoma, or cancer (the two terms are interchangeable), a definite anatomical structure, and no longer call a tumor cancer simply because it possesses the signs of malignancy which have been enumerated. Other tumors, as well as cancer, may assume a malignant course. The essential feature of carcinoma is, not the presence of any particular form of cell, for *specific* cancer-cells do not exist, but the arrangement of the cells in spaces called *alveoli*, which lie imbedded in vascular connective tissue called *stroma.* The name alveolus, as here applied, is somewhat misleading, as the spaces may be of any shape or size, and always communicate with each other. Thus, upon section, the cancerous alveoli appear broad or narrow, round or elongated or branching, and contain from three or four to several hundred cells. The cells within these alveoli are sometimes called the cancer-cells in distinction from the cells in the intervening stroma. These cancer-cells may be of the most variable size and shape; they are at least larger than white blood-corpuscles, and in a general way may be said to resemble epithelial cells in shape. This resemblance is sometimes close, at other times remote. In their relation to each other, however, the resemblance to epithelium is exact. The cancer-cells in an alveolus lie in close proximity to each other, not separated by any intercellular substance, or at least by no more than a scanty cement-substance like that between epithelial cells. If the cells be brushed or shaken out of an alveolus, this space is found to be empty. All of the tissue between the alveoli forms the stroma, which consists of fibrillated connective tissue, usually very vascular and at times very rich in cells. An old and still employed classification of cancers is into *scirrhous*, *encephaloid* or *medullary*, and *colloid* cancer. This subdivision, not being based upon essential anatomical differences, is not very satisfactory. The terms scirrhous and encephaloid refer to the consistence of the tumor. A scirrhus is a hard cancer in which the fibrous stroma predominates; encephaloid or medullary cancer is soft in consequence of the small amount of stroma and excess of cancer-cells. There is no essential structural difference between the two forms. The encephaloid cancer is naturally the more rapid in its growth and the more malignant.

A more satisfactory classification of cancers is into (1) flat-celled or squamous epithelioma; (2) cylindrical-celled epithelioma; (3) carcinoma simplex; (4) colloid carcinoma; and (5) melanotic carcinoma.

Flat-celled epithelioma occurs in the skin and in mucous membranes covered by squamous epithelioma. It is particularly liable to occur where the skin becomes continuous with mucous membrane, as in the lips, eyelids, vulva, and glans penis. Frequently in this form of cancer some of the flat epithelial cells contained in the alveoli undergo the metamorphosis into keratin, as occurs normally with the epidermal cells, and these horny cells become compacted together into little balls called epithelial or cancroid pearls. Skin cancers, although malignant, are less likely to produce metastases than most other forms of cancer, and when early and completely removed recovery is often permanent.

Cylindrical-celled epithelioma occurs in mucous membranes covered by cylindrical epithelium—namely, in the stomach, intestine, and corpus uteri. It is often difficult to distinguish between these tumors and adenomata occurring in the same situations. The distinction is to be based on the preservation of the normal gland-type in adenoma, while the glandular arrangement is irregular and not typical in carcinoma.

The term epithelioma in the restricted sense is confined to the two preceding forms of cancer, while the name *carcinoma simplex* is used to designate the ordinary cancers, in which the cells are not distinctly flat or cylindrical, but rather resemble glandular epithelial cells. Most of the so-called scirrhous and encephaloid cancers belong to this group.

In *colloid* cancer the alveoli contain partly cells and partly a translucent, gelatinous material which is thought to be derived by colloid metamorphosis from the cancer-cells. The alveolar structure of colloid cancer being generally evident to the naked eye, this variety has received also the name *alveolar cancer*, although, strictly speaking, every cancer is alveolar. The nature of colloid cancer, and even the propriety of classifying it with other forms of cancer, have not been established. It grows most frequently from the abdominal viscera or peritoneum, and is less likely to produce metastases than other forms of cancer.

In *melanotic carcinoma* black or brown pigment is present in the tumor. Most of the tumors which have been described as melanotic cancers are in reality melanotic sarcomata.

A subject which has been discussed of late years more than any other pertaining to the pathology of carcinoma relates to the origin of cancer-cells. It has been the teaching of Virchow, in accord with his scheme of pathogenesis, that cancer-cells develop out of connective-tissue cells. On the other hand, the belief has gained ground, chiefly through the researches of Thiersch and of Waldeyer, that cancer-cells not only resemble epithelial cells in shape and in relation to each other, but that they are actually the offspring of epithelial cells. It is Waldeyer's view that all varieties of cancer are of epithelial origin, and are therefore, strictly speaking, epitheliomata. The epithelial origin of the flat-celled and of the cylindrical-celled cancers has been established beyond doubt; but the epithelial derivation of the great majority of the other forms of cancer, in which the cells less perfectly preserve the type of epithelium, has been rendered extremely probable. Nevertheless, there are exceptionally tumors which in structure must be ranked as cancers, but in which the cells not only have not been proven to spring from epithelium, but are almost certainly derived from connective-tissue cells (including endothelium). As our means of determining the genesis of cells are so imperfect, it seems best for the present not to introduce the epithelial origin of the cells as an element in our definition of cancer, but to rely solely upon the alveolar structure above described, and to do this notwithstanding the belief that the great majority of cancers are of epithelial origin.

In their growth the cancer-cells push their way especially into lymph-vessels and lymph-spaces, so that oftentimes the alveoli are dilated lymph-spaces. The metastatic or secondary growths which are such a frequent accompaniment of cancer preserve, with very few exceptions, the type of the primary growth. The material, doubtless consisting of cells from the primary tumor, which causes the metastases, is transported especially by the lymphatic vessels, so that the neighboring lymphatic glands are early involved. The metastases may also occur through the blood-vessels, as is the rule with sarcoma.

Carcinoma is purely local in its origin and is not preceded by any dyscrasia. The cancerous cachexia follows the development of the primary and secondary growths. As cancer most frequently develops after forty years of age, the time of life becomes an important factor in the differential diagnosis. Nevertheless, cancer may occur early in life. In fact, cancer of the kidney is more frequent in children than in old persons. On account of the frequency of cancer of the breast and of the uterus, females are more subject to cancer than males.

Carcinoma may grow from any organ possessing epithelium. Primary cancers of bone, lymphatic glands, serous membranes, and other parts devoid of epithelium, are certainly very rare. A frequent metamorphosis in cancers is fatty degeneration with disintegration of the cell-elements. In this way ulcerations are produced. By combined fatty degeneration and increase of the fibrous stroma a cancerous tumor may be much reduced in size and activity, but it is doubtful whether recovery ever takes place in this way.

CHAPTER IV.

ACTIVE ALTERATIONS OF THE TISSUES (CONTINUED).

Infectious Tumors—Syphilis—Leprosy—Glanders—Actinomycosis—Tubercle—Scrofula.

Infectious Tumors.

THE infectious tumors may be regarded as inflammatory new formations due to the presence of certain specific viruses, which, so far as known, are vegetable parasites. These tumors, which are not to be confounded with genuine tumors, have many common characteristics. They are composed chiefly of a lowly-organized tissue resembling granulation-tissue. Hence Virchow called these tumors granulomata. The vascular supply of these new growths is generally very imperfect, so that they are liable to degenerate and die. They occur in the form of multiple nodules, often widely distributed over the body. With the nodules are often combined diffuse growths of the same tissue, as well as ordinary inflammatory changes. The presence of a specific virus is proven by the inoculability of these tumors in animals which are susceptible to the disease. This specific virus has, in all cases as yet known (with the exception of actinomycosis), been found to be some form of bacteria.

The infectious tumors are the nodular masses produced by syphilis, leprosy, glanders, actinomycosis, and tuberculosis. Of these diseases, the only one of which a full consideration falls within the scope of this work is tuberculosis.

Syphilis is attended by many inflammatory lesions which do not differ anatomically from ordinary inflammations. The *syphiloma* or *gumma*, however, presents certain structural peculiarities. A gumma is a nodule, usually larger than a tubercle, composed at first almost wholly of lymphoid cells. Subsequently, the centre of the nodule undergoes a peculiar hard, caseous metamorphosis. Around this caseous centre is usually a zone of dense fibrous tissue, and in the periphery of the nodule, as well as in the surrounding tissue, are accumulated round and fusiform cells. Blood-vessels are present in gummata. Gummata may be found in the liver, membranes of the brain, heart, testicle, bone, skin, and other parts. They often in part disappear spontaneously or under appropriate treatment.

In 1884, Lustgarten, in Weigert's laboratory, discovered in syphilitic products a bacillus which is probably to be regarded as the specific virus of the disease, although further observations are necessary before a positive opinion can be formed. The syphilis bacilli very closely resemble in appearance the tubercle bacilli. They are of about the same size and shape as the tubercle bacilli, and they often appear as somewhat curved or bent rods. The ends of the bacilli are slightly thickened. They may contain clear, oval, glistening spots, which are probably spores. The bacilli are found in the interior of cells, either singly or in groups. They occur, as a rule, in very scanty numbers, and long search is often required to discover them.[1] According to Lustgarten, they are constantly present in syphilitic products, but all observers have not been so fortunate in finding them. They have not as yet been cultivated outside of the body.

The lesions of **Leprosy** occur most frequently in the skin and in the nerve-trunks, but they may be found in various mucous membranes and in the viscera. The most characteristic of these lesions are nodules made up mostly of lymphoid and epithelioid cells. The lesions of leprosy contain characteristic bacilli, often in enormous numbers. These bacilli, which are doubtless the cause of the disease, may be found free or enclosed in cells. They are found especially in large spheroidal or irregular clumps. The bacilli lepræ are slender, often pointed rods, 4–6 μ in length. They form spores. They are best stained by the same method as that employed for tubercle bacilli, but, unlike the latter organisms, they are also colored by the ordinary staining processes for bacteria. Cultivation and inoculation experiments with the leprosy bacilli are as yet imperfect.

Glanders is a contagious disease of horses which may be transmitted to man. The nodules of glanders are most frequently found in the nose of the horse, but in this animal as well as in man the nodules may be found in the skin, the mucous membranes, and the viscera. The nodules usually suppurate, and extensive ulcerations often take place. Small bacilli, not unlike those of tuberculosis, but nevertheless distinguishable from them, are found in the lesions of glanders. These bacilli can be cultivated outside of the body in blood-serum, and the disease can be readily produced by the inoculation of the pure cultures. The evidence that these bacilli are the cause of glanders is therefore conclusive.

Actinomycosis is a disease produced by a peculiar vegetable parasite called actinomycis, or ray-fungus. The disease affects cattle, swine, and human beings. It has been most frequently observed in the jaws of cattle, but it may also affect the lungs, intestinal tract, and other parts. The disease is characterized by new growths of granulation-tissue, which often contain foci of suppuration, and which may in some places be transformed into fibrous tissue. The new growths are frequently extensive, and they were formerly mistaken in many cases for sarcomata. The parasite occurs in the granulation-tissue and in the pus in the form of ray-like masses, often visible to the naked eye as little yellowish or whitish specks. According to Boström, actinomycis belongs to the highly-developed forms of schizomycetes; others regard it as a fungus. Inoculation and cultivation experiments have yielded positive results.

Tubercle.

Tuberculosis is a disease produced by infection with a specific vegetable parasite known as the bacillus tuberculosis. This organism, the discovery of which by Koch was announced in 1882, is a slender rod 2 to 5 μ long (one-quarter to one-half the diameter of a human red blood-corpuscle). It often appears bent or slightly curved. Its most characteristic physical property is its behavior toward staining agents. While, on the one hand, it stains with more difficulty than most bacteria, on the other hand, after it has been stained by the proper methods, it retains the staining dye after treatment by acids and other agents which extract the color from other bacteria.[1] The bacillus tuberculosis can be cultivated outside of the body in sterilized gelatinized blood-serum. The organism develops in these cultures very slowly, and most favorably at about the temperature of the human body. It does not grow at a temperature less than 30° C. (86° F.). The bacillus multiplies by transverse division. Within the body it develops oval spores, of which from two to six may be present in a single rod. Tubercle bacilli containing spores may preserve their vitality at least for many months in the dry state outside of the body, but under ordinary circumstances they cannot multiply out of the body, as they do not find the proper conditions of temperature. The tubercle bacillus is therefore to be regarded as a genuine parasite requiring for its growth and multiplication conditions found naturally only in the animal body.

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[1] The syphilis bacilli may be stained by the following process (De Giacomi's): The preparation is first stained for twenty-four hours with fuchsin; it is then washed for a few seconds in distilled water to which a few drops of liquor ferri chloridi are added; it is then decolorized for a few seconds in concentrated liquor ferri chloridi, after which it is treated, as usual, with alcohol, xylol or oil of cloves, and Canada balsam.

It need hardly be said that the discovery of the bacillus tuberculosis has greatly elucidated the pathology of the tuberculous processes. In the presence of this organism we possess a certain proof of the tuberculous nature of a given lesion.

Infection by the tubercle bacillus may take place through the respiratory tract, the digestive tract, wounds of the skin, or the genito-urinary passages. The poison is most frequently absorbed by the respiratory tract.

Of the lesions produced by the invasion of tubercle bacilli, the most characteristic, anatomically, are little nodules called miliary tubercles. When young, and before they have undergone degeneration, these tubercles are gray and translucent in color, somewhat smaller than a millet-seed in size, and hard in consistence. Examined microscopically, undegenerated miliary tubercles are found composed of a reticulated basement-substance and epithelioid cells, giant-cells, and lymphoid cells. In the centre of a typical tubercle are one or more giant-cells surrounded by a zone of epithelioid cells, while the lymphoid cells are most abundant in the periphery of the nodule. In some instances a tubercle is made up wholly of epithelioid cells; in other cases scarcely any but lymphoid cells may be found. It has been held that the first step in the formation of a tubercle is the accumulation of lymphoid cells, which change into epithelioid cells, and these into giant-cells. The investigations of Baumgarten, however, have rendered it probable that the first effect of the lodgment of the tubercle bacillus is a proliferation of the fixed cells of a part (epithelial cells, connective-tissue cells), resulting in the production of epithelioid cells. The lymphoid cells are emigrated white blood-corpuscles, which infiltrate the primary nodule of epithelioid cells. The tubercle giant-cells are large cells containing many nuclei, which are usually arranged in the form of a more or less complete ring in the periphery of the cell. Tubercle giant-cells are produced by repeated division of the nuclei of epithelioid cells, the cell-body not taking part in the division, in consequence of impairment of its structure by the presence of bacilli (Weigert). Tubercles are devoid of newly-formed blood-vessels, and the old vessels soon become impervious.

Partly in consequence of this lack of vascular supply, but chiefly by the action of the tubercle bacilli, miliary tubercles do not long remain in the condition which has been described. The cells in the central part of the tubercle soon lose their nuclei, and their protoplasm is converted into a hyaline mass or breaks down into a granular detritus. These changes, which are due to coagulation necrosis, are embraced under the names hyaline degeneration and cheesy degeneration. The majority of miliary tubercles found at autopsies present three zones—namely, a central caseous necrotic mass, a middle zone of epithelioid cells associated often with giant-cells, and a peripheral zone of lymphoid cells. Miliary tubercles may increase in size by peripheral growth, but large tuberculous nodules are generally formed by the coalescence of a number of miliary tubercles.

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[1] As already remarked, the bacillus of leprosy stains under the same conditions as the bacillus tuberculosis.

Miliary tubercles are not the sole tuberculous products. The tubercle bacilli may cause the diffuse growth of a tissue identical in structure with that of miliary tubercles; that is, composed of a basement-substance containing epithelioid, giant, and lymphoid cells. This diffuse tubercle-tissue also undergoes cheesy degeneration. Such diffuse tuberculous masses are sometimes called infiltrated tubercle.

Ordinary inflammation—that is, inflammation with the production of fibrin, serum, and pus—is also often associated with tuberculous lesions, and is probably to be regarded as a direct effect of the action of the tubercle bacilli.

The minute structure of the tuberculous lesions varies considerably with the character of the tissue in which they occur. Peculiarities of structure due to this cause will be considered in Part II. of this work in connection with the tuberculous diseases of special organs, so far as these diseases fall within the scope of a work on practical medicine.

Tubercle bacilli are found in all tuberculous lesions. They are generally less abundant in the caseous than in the cellular parts of the tubercle. In the caseous wall of phthisical cavities communicating with the external air, tubercle bacilli, however, are exceptionally abundant. Giant-cells are favorite seats of the bacillus.

Tuberculosis may be local or general. Thus the disease may be localized in the skin (lupus), in the lymphatic glands, in the joints, in the lungs, in the kidneys, etc. The tubercle bacilli are frequently conveyed by the lymphatic current. When large quantities of the bacilli rapidly gain access to the general blood-current, as when tuberculous masses grow into the pulmonary veins or the thoracic duct, an eruption of miliary tubercles occurs throughout most of the organs of the body, constituting the disease called acute miliary tuberculosis.

The infectious nature of the tuberculous products is proven, not only by their diffusion throughout the body in the manner described, but also by their inoculability in animals. That the infectious agent consists of the bacilli is proven by the fact that inoculation of pure cultures of the bacilli, which after a certain number of generations contain nothing else which was present in the original tuberculous material, no less surely produces artificial tuberculosis in animals. That the bacilli constitute the essential or specific cause of tuberculosis is proven by the facts that inoculation by no other substance than tuberculous material is capable of producing tuberculosis in animals, and that these organisms are constantly present in tuberculous lesions.[1]

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[1] The observers who believe that they have produced tuberculosis in animals by inoculation with indifferent substances have mistaken the nodules which form around foreign particles for genuine tubercles. These nodules differ from tubercles in the absence of infectious properties, in the absence of tubercle bacilli, and in the absence of cheesy degeneration. Another source of error has been a lack of proper precautions in excluding unforeseen infection with the tuberculous virus.

Tuberculous caseous material and tuberculous pus may prove infectious even when they are free from bacilli. The substance then contains the spores of the bacilli. These spores we are unable at present to recognize, but their existence is demonstrated by the subsequent development of bacilli in the substance and in the tubercles produced by its inoculation. Any other assumption would lead to the unwarranted doctrine of spontaneous generation of the bacilli.

Scrofula.

The term scrofula is of very old date, and is used with a rather comprehensive and indefinite latitude of signification. It is applied to a diathesis or constitutional condition observed especially in young persons who are the offspring of tuberculous parents or who are in bad hygienic surroundings. Scrofulous persons are liable to inflammations, particularly of the mucous membranes, lymphatic glands, skin, and periosteum. These inflammations are of a chronic character; the exudations have a tendency to cheesy degeneration rather than to resolution or organization. The inflammatory products are rich in cells, many of which are larger than those usually present in inflammation. The lymphatic glands, especially those of the neck, enlarge, often coalesce with each other, and undergo cheesy metamorphosis. The name scrofula was adopted with reference to these glandular swellings in the neck. The various affections attributed to scrofula often disappear as the individual grows older and the hygiénic surroundings are improved. There is an intimate relation between scrofula and tuberculosis. The cheesy deposits in scrofulous lymphatic glands are generally tuberculous. Many writers do not recognize the existence of scrofula as a special cachexia independent of tuberculosis.

CHAPTER V.

PASSIVE ALTERATIONS OF THE TISSUES.

Atrophy—Necrosis—Parenchymatous Degeneration—Cloudy Swelling—Fatty Metamorphosis—Mucoid and Colloid Degeneration—Amyloid or Waxy Degeneration—Hyaline Degeneration—Calcareous Degeneration—Pigmentation.

THE passive alterations of the tissues will be considered under the heads *atrophy*, *necrosis*, *degenerations*, and *infiltrations*.

Atrophy.

The term *atrophy* expresses a condition the reverse of hypertrophy. In atrophy the histological elements of a part diminish in size or in number without undergoing further change of structure. Diminution of volume and of weight characterizes atrophy, unless a deposit of fat or of some other substance compensates for the loss of material. An atrophic organ is generally dry, firm, and anæmic. Atrophy may be a *physiological* process, as is the case with the normal involution of the thymus gland and of the female genital organs. Senile atrophy is perhaps also to be included among the physiological atrophies, although pathological causes are often present. *An insufficient supply of nutriment* is a most efficient cause of atrophy both local and general. Examples of local atrophy due to this cause are the wasting of the hepatic parenchyma in cirrhosis of the liver and the atrophy of parts subjected to pressure (compression atrophy). A general atrophy, although in unequal degree, of the organs and tissues of the body is the result of insufficient supply of food and of malassimilation. *Functional inactivity* is another cause of atrophy. This atrophy may be in part explained by the diminished blood-supply which is known to attend suspended function. As examples of this functional atrophy may be cited the wasting of the muscles in paralysis, of the bone in ankylosis, and of the intestine below a preternatural anus. *The withdrawal of nervous influence* may be a cause of atrophy. A rapid atrophy of the voluntary muscles follows degeneration and atrophy of the multipolar ganglion-cells in the anterior horns of the spinal cord, or lesions suspending the functions of the peripheral nerves which connect the muscles with these ganglion-cells. So rapidly does this atrophy progress that it cannot be attributed merely to loss of function. It seems necessary to assign a trophic or nutritive function to these ganglion-cells. Examples of this *neurotrophic atrophy* are progressive muscular atrophy, infantile spinal paralysis, and peripheral palsies.

The microscopical examination of atrophied parts reveals a diminution in number or in size of the cells, usually both. A division into *simple atrophy*, in which there is only a diminution in size, and *numerical atrophy*, or *aplasia*, in which there is diminution in number of cells, is not of practical importance. In the atrophy of adipose-tissue cells Flemming observed an infiltration of the cells with serum and an increase in the number of nuclei. This so-called *atrophic proliferation* shows that increase in nuclei is not sufficient evidence of an active proliferation process. In the neurotrophic muscular atrophies there is also an increase in number of the muscle-corpuscles and an increase of the interstitial tissue. In atrophy of the heart a deposit of brownish pigment usually occurs in the atrophic muscular fibres, hence the name *brown atrophy*. In atrophy of the kidney, and sometimes in muscular atrophy, especially in pseudo-hypertrophic paralysis, an excessive deposit of fat occurs about the kidney and between the muscle-fibres.

Necrosis.

The death of any part of the body is called *necrosis*. Necrosis of external parts of the body, attended by shrinkage and mummification of the tissues, is called *dry gangrene*. Necrosis accompanied by putrefaction of the dead tissues is called *moist gangrene*. The putrefaction is due to the action of a ferment generated by certain bacteria. Simple necrosis, with softening and liquefaction of the tissues, is called *colliquative necrosis*. Necrosis with coagulation of the dead tissues is called *coagulation necrosis*.

The causes of necrosis are manifold, but they may be grouped under two headings—*first*, agencies which directly destroy the vitality of the cells; *second*, arrest of the circulation. Frequently the two classes of causes are combined in their action.

The agencies which cause disintegration of the cells may be classified as *mechanical*, *chemical*, and *thermic*. There is also to be included, the action of certain *specific poisons*, such as those of snake-bites and of various micro-organisms.

The causes which arrest the circulation may act primarily on the arteries, veins, or capillaries. Obstructions in the arteries lead to necrosis in situations where no sufficient collateral circulation can be established, as in the kidneys, the spleen, some parts of the brain, etc. (See *Embolism and Thrombosis.*) Venous obstruction can hardly lead to necrosis, on account of the relief afforded by abundant collateral channels. If all the veins of a part be occluded when arterial is combined with venous obstruction, necrosis follows, as in strangulated hernia and in the constriction of a part by a tight bandage. Obstructions in the capillaries attended by necrosis are generally due to agencies which in addition impair the life of the tissue-cells. The gangrene of the extremities which follows the prolonged ingestion of ergot is usually attributed to the contraction of the small arteries induced by this substance. The endemic occurrence of ergotism from eating diseased grain seems to have disappeared.

Under pathological conditions the resistant power of the body may be so diminished that comparatively slight causes suffice to produce necrosis. Thus, in feeble, bed-ridden persons moderate pressure and uncleanliness often produce necrosis in the form of bed-sores (decubitus). These bed-sores may occur in a very short time, even in twenty-four hours, after injuries and diseases involving the gray matter of the spinal cord. This acute necrosis is attributed to neuropathic disturbance. Diabetes mellitus is a disease in which the resistant power of the tissues is greatly weakened, and in which gangrene is not infrequent. *Senile gangrene* is usually the result of the obstruction of arteries which are already extensively atheromatous, so that a collateral circulation can with difficulty be established.

The appearance of necrotic parts varies with the form of necrosis. Necrosed bone and tendon may preserve their normal appearances. In dry gangrene, which is represented by certain forms of senile gangrene and of gangrene following frost-bites, the dead tissues are dry, shrivelled, and dark in color. In moist gangrene the affected part is humid, of a dark color from diffusion of blood-pigment, of a penetrating odor, and it contains various new gaseous and chemical substances. Of the latter, some are crystalline, such as leucin, tyrosin, hæmatoidin, fatty acids, and triple phosphates. Bacteria of different forms are found in great abundance. Moist gangrene is usually met with only in parts of the body exposed to the air, as the extremities, the lungs, and the uterus. Gangrene in internal organs may, however, occur by the transportation thither of the bacteria of putrefaction, as by means of emboli detached from putrid thrombi. Colliquative necrosis is illustrated by anæmic necrosis of the brain, in which the affected part is soft, sometimes almost liquid, in consistence, and contains fragments of disintegrated brain-substance. The liquefaction is probably due to the absence of coagulable material in the brain.

Essential for the production of *coagulation necrosis* are—*first*, that the tissue contain a sufficient amount of coagulable substance; *second*, that the dead tissues shall be richly permeated by lymphatic fluid; and *third*, that fermentative or other chemical agents hostile to coagulation shall be absent. The coagulable substance in the tissues is protoplasm and possibly other albuminous substances. That the dead tissues shall be pervaded by lymph requires that they shall be more or less completely surrounded by living tissues. Agents which antagonize coagulation are the bacteria which cause suppuration and decomposition, and the epithelium covering mucous membranes.

The chemical process of coagulation is to be regarded as a combination of the fibrinogen contained in lymphatic and other plasmatic fluids, with certain substances (fibrin ferment and fibrino-plastin ?) furnished by the death of protoplasm. In the coagulation of fibrin in the blood and in inflammatory exudations the fibrin ferment is furnished by the destruction of leucocytes. In the coagulation necrosis of tissues the tissue-cells furnish certain constituents of fibrin, and are themselves directly transformed into fibrin or a substance resembling it.

Coagulation necrosis may affect entire organs or parts of organs or individual cells. It occurs in a great variety of lesions, the most important of which are infarctions, atheroma of vessels, tubercular cheesy foci, tumors, typhoid fever, relapsing fever, diphtheria, smallpox, and the waxy degeneration of muscles.

The most characteristic microscopical change in a part which is the seat of coagulation necrosis is the loss of nuclei in the cells. The cell-protoplasm often presents a hyaline, homogeneous appearance, and it may be distorted in shape. Subsequently there is usually granular disintegration of the affected tissue. The gross and microscopical appearances and the chemical processes are not the same in all of the different forms of coagulation necrosis, but it is impossible to enter here further into histological details, some of which will be referred to in appropriate places in the following part of this work. Some pathologists include many of the changes which have been described as belonging to coagulation necrosis under the name of hyaline degeneration, which will be described subsequently.

Parenchymatous Degeneration—Cloudy Swelling.

The names parenchymatous degeneration or inflammation, albuminous infiltration, granular degeneration, and cloudy swelling are used as synonyms for the same metamorphosis. This change consists in the appearance in cells, and sometimes in intercellular substance, of abundant albuminous granules. Especially subject to this alteration are the muscular fibres and the parenchymatous cells of the glandular organs, particularly the renal epithelium, the hepatic cells, and the peptic cells. The affected parts appear swollen, opaque as if boiled, and the normal markings, as of the hepatic acini and of the renal striæ, appear obscured. The microscopical examination shows the cells swollen and filled with fine granules, which dissolve in acetic acid and in potash, but are insoluble in ether. In the kidney the epithelium of the convoluted tubes, and in the liver the hepatic cells, are normally so granular that the macroscopical appearances are more conclusive as to the existence of this change than the microscopical. The muscular fibres of the heart are liable to this change. They then contain albuminous molecules which obscure the normal striation. The cells in inflamed parts undergo parenchymatous degeneration.

The most important causes of cloudy swelling are infectious diseases, such as typhus and typhoid fever, pyæmia, puerperal fever, diphtheria, and poisoning with phosphorus, arsenic, or the mineral acids. Cloudy swelling has been developed within six hours after extensive burns (E. Wagner). The view of Liebermeister, that it is solely the influence of high temperature in general diseases which causes this metamorphosis, has been abundantly disproven. Cloudy swelling is sometimes followed by fatty degeneration. If its causes disappear, there is usually a return to the normal condition. Parenchymatous degeneration undoubtedly impairs the function of the affected parts.

Fatty Metamorphosis.

It is customary to distinguish between *fatty infiltration* and *fatty degeneration* of cells. In fatty infiltration or fatty growth the fat has been believed to come from without the cells, and to infiltrate them in the form of large drops, and in fatty degeneration to be derived by direct metamorphosis from the cell-substance, and to appear in the form of molecules. But these distinctions cannot be maintained. Thus, in so-called fatty infiltration, while the fat may be derived from without, it is equally certain that it may be produced directly from the protoplasm of the cells. Thus, no one would consider the drops of oil so frequently found in growing cartilage-cells as evidence of fatty degeneration; still, the fat here can hardly be formed otherwise than from the substance of the cells. Again, there can be no purer example of fatty infiltration than the absorption of fat by the intestinal epithelium, in which, however, the fat appears in the form of fine granules. Most writers consider the fatty liver resulting from poisoning by phosphorus as an instance of fatty degeneration, although here the fat appears in large drops.

The chief distinction between fatty infiltration and fatty degeneration—to continue to use the old terms—is more chemical than morphological. In fatty infiltration of a cell the protoplasm is displaced by the fat, but does not suffer materially in its integrity except by a slow process of atrophy from compression; if the fat be derived from the albuminous constituents of the cell, these are, in great part at least, renewed. In fatty degeneration, on the other hand, the cell-substance is directly converted into fat, and is not at all, or only insufficiently, regenerated. Hence fatty degeneration is also called fatty atrophy. A considerable degree of fatty infiltration may exist without interfering materially with the function of the affected cells, although in extreme degrees this must suffer. But fatty degeneration is a much more destructive process, and interferes to a much greater extent with the function of the cells. It is not always possible to draw a sharp line of distinction between fatty infiltration and fatty degeneration. The problems involved are certainly more complex than was formerly believed.

In determining the nature of the process it is of the greatest importance to take cognizance of the cause. The accumulation of fat in the organism is the result of its incomplete oxidation. The causes of fatty metamorphosis, therefore, are—*first*, excessive supply or excessive formation of fat; and *second*, diminished oxidation of the fat.

Too abundant ingestion of rich food, especially of fat and of carbohydrates. leads to *obesity* or the excessive accumulation of fat in its natural dépôts. Constitutional peculiarities, often hereditary, the nature of which is not understood, favor the development of corpulence. An excessive formation of fat seems to be sometimes the result of increased nutritive activity, as in growing cells. The causes leading to diminished oxidation of the fat may be general or local. The general causes are—*first*, interference with the absorption of oxygen in consequence of disease of the respiratory organs; *second*, diminution of the hæmoglobin of the blood; and *third*, agencies which check the normal oxidizing processes. Diseases of the respiratory organs in themselves rarely, if ever, lead to fatty metamorphosis, but they increase the efficiency of other causes. The various agencies which diminish the number or impair the quality of the red blood-corpuscles, these being the carriers of oxygen, are important causes of fatty degeneration. In this group of causes are to be reckoned progressive pernicious anæmia, icterus gravis, and other diseases attended by profound anæmia, as leucocythæmia, chlorosis, chronic pulmonary tuberculosis, and in less degree other chronic cachexiæ. Phosphorus and some other poisons lead to destruction of the red blood-corpuscles and consequent fatty metamorphoses. There is some reason to believe that an insufficient supply of oxygen favors rapid tissue-metamorphosis, with the formation of fat. Especially in phosphorus-poisoning does there seem to be an excessive formation of fat as well as diminished oxidation. The use of alcohol favors the accumulation of fat by diminishing its normal oxidation. The parts which are most subject to fatty degeneration from general causes are the liver, the kidneys, the muscles, especially the cardiac muscle, and the coats of the small arteries.

Local fatty degeneration can be attributed generally to an insufficient supply of oxygenated blood. It is to be remembered that complete cessation of the blood-current leads to necrosis, and not to fatty degeneration. In cirrhotic livers the hepatic cells are often fatty from the interference with the circulation through the organ. Pus-cells are usually fatty from imperfect nutrition. For the same reason other inflammatory exudations undergo fatty degeneration. The opaque ring so often observed in the outer margin of the cornea of old people, called *arcus senilis*, is due to fatty degeneration of the corneal corpuscles.

A fatty organ is generally anæmic, of a yellowish color, and of diminished consistence. In fatty degeneration of muscles the fibres contain a large number of molecules and small drops of oil. The striæ are obscured, and may disappear. In the fatty infiltration of muscles the fat is deposited always in large drops in the cells of the interstitial tissue, and not in the muscular fibres. In fatty degeneration the cell may be completely destroyed, leaving only a fatty detritus. The description of the gross and microscopical appearances in the fatty metamorphosis of different organs belongs to Part II. of this work, where, especially, the sections devoted to fatty heart and to fatty liver may be consulted.

Mucoid and Colloid Degenerations.

Mucoid degeneration consists in the transformation of the albuminous constituents of cells or of intercellular substance into mucin, which gives the tissues a gelatinous, translucent appearance. Mucin is an albuminoid substance devoid of sulphur, held in solution only by an alkali, and therefore precipitated by acetic acid, in an excess of which it is insoluble. It is normally secreted by epithelial cells of mucous membranes and of certain glands, and it is widely distributed in the embryonic tissues. Mucoid degeneration may affect either cells or intercellular substance. It occurs occasionally in cartilage, the marrow of bone, and especially in tumors. In the myxomata the intercellular tissue is composed chiefly of a substance containing mucin.

The *colloid metamorphosis* is allied to the mucoid. It affects chiefly cells. The colloid material appears in the form of drops, either free or within cells, and it may appear more diffusely. It is of firmer consistence than mucin, contains sulphur, and is not precipitated by acetic acid. In its gelatinous, homogeneous appearance it resembles mucin. The colloid material accumulates in great amount in the follicles of the thyroid gland in goitre. The colloid change may also affect the cells in tumors.

The causes of these metamorphoses are not understood.

Amyloid or Waxy Degeneration.

The degeneration called waxy, amyloid, or lardaceous is characterized by the appearance, especially in the walls of the vessels, but also elsewhere, of a homogeneous, firm, inelastic, translucent substance similar in its composition to the albuminous matters, but more resistant to putrefaction and to the action of the gastric juice. The most important property of this new substance is its reaction with certain coloring agents, which are therefore employed for its detection. Treated with iodine, it is stained mahogany-brown; with iodine and sulphuric acid, often blue; and with methyl-violet, red. The substance was called amyloid from its supposed resemblance to starch, but it is now known to be a nitrogenous substance. For detecting the amyloid degeneration with the naked eye, a solution of iodine alone (diluted Lugol's solution) is most applicable.

The waxy degeneration may be local, but it generally affects a number of organs. The parts most frequently involved are the spleen, liver, kidneys, lymphatic glands, and the intestinal mucous membrane. Other parts may be affected, as the large vessels, the heart-muscle, the suprarenal capsules, and the small vessels in most organs. If the degeneration be well marked, the affected part presents a characteristic translucent, grayish appearance, and is usually more or less swollen and hard in consistence. The waxy material is exceptionally deposited in large nodules, constituting the waxy tumors. Waxy degeneration has been observed as a purely local change in tracheomatous granulations of the conjunctiva, and rarely in other parts. As a rule, the degeneration appears first in the walls of the small arteries and about the capillaries; thence it may extend to the surrounding tissue, which in some cases appears to be primarily involved. It has usually been held that a waxy degeneration may invade cells, connective tissue, and, in fact, nearly all the histological elements. It appears, however, to be confined to the basement-substance between the cells. The so-called structureless membranes, such as the membrana propria of the uriniferous tubules, may undergo the change. The waxy substance is often deposited in irregular clumps. The cells are compressed and atrophied. The vascular walls become converted into a hyaline, swollen mass which encroaches upon the lumen. The endothelium is for a long time preserved, and the circulation continues until the vessel is nearly obliterated. The anatomical peculiarities presented in the amyloid degeneration of different organs will be described in Part II.

The causes of waxy degeneration are chronic suppurations, particularly of bone, and certain cachexiæ, especially the syphilitic, the tuberculous, and, in a less degree, the cancerous, the gouty, and the malarial. In rare instances no apparent cause can be assigned. The degeneration may be developed within a few months from the onset of suppurative processes. It appears, as a rule, first in the spleen. It is difficult to tell exactly what symptoms are referable to waxy degeneration, as it is associated with severe wasting diseases. In the kidneys it seems to be always associated with more or less change in the interstitial tissue and in the epithelium. Waxy degeneration of the intestines leads to chronic diarrhœa.

The source of the waxy material, whether from the blood or from the albuminous substances in the tissues, is not known.

There appear in various parts of the body, both normally and pathologically, spherical or irregular masses which usually present a concentric arrangement, and which are called *corpora amylacea.* They often assume a blue color when treated with iodine or with iodine and sulphuric acid. They are not known to have any relation to waxy degeneration. So far as ascertained, they have no pathological importance. They occur especially in the prostate gland and in the central nervous system, in the latter being particularly abundant in chronic inflammatory processes.

Hyaline Degeneration.

Hyaline degeneration is the transformation of the tissues into a homogeneous, translucent, glistening material which stains deeply with eosin and carmine and is unaffected by acids. This substance, which much resembles the amyloid material, is distinguished from it by the absence of the iodine reaction. Von Recklinghausen attributes hyaline degeneration to the formation in the cells and the deposit in the tissues of a substance which he calls hyalin. This hyaline transformation is not unusual in the walls of small blood-vessels. According to Von Recklinghausen, hyaline degeneration plays an important rôle in many pathological processes. Changes which others attribute to colloid metamorphosis, to coagulation necrosis, or to fibrinoid degeneration, he refers to the development of hyalin. Hyalin may occur in the form of thrombi in the vessels.

Calcareous Degeneration.

An infiltration of the tissues with the phosphate and carbonate of lime, mingled usually with traces of the same magnesia salts, constitutes what is called *calcareous degeneration*, *calcification*, or *cretefaction*. This change is not to be confounded with *ossification*, although the latter term is often applied to it erroneously. In ossification there is a formation of true bone. In calcification the salts of lime are deposited at first in the form of irregularly scattered granules which appear dark by transmitted and bright by reflected light. Large calcareous masses may be formed by increase in number and size of the granules. Calcified tissues are recognized by their hard, sometimes stony, consistence, and by the solubility of the earthy salts in strong acids, usually with the evolution of bubbles of gas. The salts are deposited both in the intercellular substance and in the cells.

Virchow has described, under the designation *lime metastases*, the deposit of the salts of lime in various parts of the body, particularly in the arteries, lungs, and stomach, in cases of extensive caries and of cancerous disease of bone. This calcification he attributes to the presence in the blood of an absolute excess of lime salts derived by absorption from the diseased bone. With the exception of these cases, to which Virchow has called attention, it does not appear that calcareous degeneration can be referred to an abnormal quantity of lime in the blood.

In the vast majority of cases of calcification the lime is deposited in tissues previously diseased. Especially prone to calcification are tissues which have undergone fatty degeneration or coagulation necrosis. Thus, Litten observed calcareous particles in renal epithelial cells, in which coagulation necrosis had developed, in twenty-four hours after temporary ligation of the renal artery. We find calcific deposits in cheesy masses, in old inflammatory products, in desiccated pus, in tumors, and in and about parasites. Of great practical importance is the calcification of the cardiac valves in chronic endocarditis and of the arteries, either with or without inflammatory changes. Calcification of the internal and middle coats of the arteries belongs among the usual changes in old age. What is usually called calcification of cartilage is ossification or a change preparatory to ossification. Calcification may be a conservative process, as when it checks the growth of tumors, or it may be detrimental, as when it affects the arteries or cardiac valves.

Pigmentation.

Abnormal pigment in the body may be derived from the coloring matter of the blood, or it may be elaborated by the protoplasm of cells, or it may be introduced from outside of the body.

The hæmoglobin of extravasated red blood-corpuscles may be converted into yellow or brown pigment-granules, or into red rhombic crystals called hæmatoidin or bilirubin. The crystalline hæmatoidin differs from the granular pigment in the absence of iron. According to Langhans, the transformation into hæmatoidin occurs, not from diffused blood-coloring matter, but from red blood-corpuscles or their fragments which have been taken up by cells, especially leucocytes. The yellowish-brown pigment-granules in the blood and in various organs in the melanæmia of severe malarial intoxication are derived from the destruction of red blood-corpuscles.

The pigment found in melanotic tumors, in freckles, in chloasma, in the skin and certain mucous membranes in Addison's disease, and that present in atrophied muscle (brown atrophy), is produced in the interior of cells—whether from blood-coloring matter or not is uncertain. Fatty molecules are sometimes deeply stained with yellow or brown pigment, the source of which is not known (lipochrome).

Examples of pigment introduced from without are the black particles of carbon always found in the lungs of adults, inhaled iron particles, pigment in the skin and lymphatic glands resulting from tattooing, and particles of silver deposited in the tissues after the prolonged use of nitrate of silver.

Melanin is a name which is applied to most of the black pigments found in the body, without regard to their origin.

CHAPTER VI.

GENERAL PATHOLOGY OF THE BLOOD.

Plethora—Anæmia—Hydræmia—Anhydræmia—Hyperinosis—Hypinosis—Leucocytosis—Alterations in the Gases of the Blood—Increase of Oxygen—Effects of Breathing Compressed Air—Deficiency of Oxygen—Apnœa—Changes in the Albumen, Fat, and Inorganic Salts.

THE morbid conditions to be now considered embrace alterations in the quantity or in the quality of the entire mass of the blood. The means for regulating the composition of the blood in health are such that the mass of the blood is easily kept within physiological limits, notwithstanding changes in diet and in physical surroundings. Disturbances of this physiological regulation are generally referable to morbid processes in the solids, and hence the changes embraced in the general pathology of the blood are not independent affections of the blood itself, as they were regarded by the humoral pathologists, but they result usually from disease of the solid tissues, including the blood-forming organs. There are some alterations of the blood in which this connection has not been traced, and without doubt morbific agents may exercise their injurious influence primarily upon the blood as well as upon other tissues; for the blood is sometimes, although not very appropriately, regarded as a tissue with cells and fluid intercellular substance. The so-called *dyscrasiæ* depend upon anomalies in the composition of the blood of a chronic nature, and usually, if not always, are secondary to diseases of the solid parts. The physiological relations of the blood to the solid parts are so intimate and important as to render it intelligible that morbid alterations in its composition lead secondarily to changes in the solids, as well as that diseases of the solids affect the composition of the blood.

Alterations in the blood may affect, severally or combined, the corpuscular elements, the water, the organic ingredients of the plasma—namely, albumen and fibrin-generators, the gases, and the inorganic salts. Morbid conditions of the blood, due to the introduction of substances not entering into its normal composition, are also to be included in the consideration of its general pathology.

Plethora.

Plethora signifies an increase either in the total amount of the blood or in the number of the red blood-corpuscles beyond the healthy limit. The former of these conditions constitutes *polyæmia*, the latter *polycythæmia*. Individuals with a certain group of symptoms have, since remote times, been designated as plethoric, upon the assumption that the symptoms were due to an excess of blood. The symptoms referred to are abnormal redness of the face and mucous membranes; fulness of the pulse; increase of the heart's impulse, with tendency to palpitations; a sensation of warmth and of fulness, especially in the head and chest; and sometimes epistaxis and hemorrhages from different mucous membranes. Constitutional tendency, overfeeding, idleness and luxurious habits, and the arrest of periodical or habitual hemorrhages, are the leading causes assigned for this condition of supposed plethora. Bloodletting, reduced diet, and increased exercise often relieve the symptoms. While the symptoms and the means of relief certainly suggest an excess of blood, yet an increase in the amount of blood or of red blood-corpuscles in this condition has not been proven, and recent experiments on animals render improbable the existence of a permanent polyæmia.

Temporary plethora may be produced by *transfusion* of blood from one animal into another of the same species. In this way the amount of blood in a dog may be doubled without evident discomfort to the animal after the injection, and with only a brief rise in the blood-pressure during the operation. It is thus shown that the blood-vessels are capable of holding much more than the normal amount of blood. It is an interesting fact, however, that the transfusion of this large amount impairs so much the elasticity of the vessels that they cannot at once accommodate themselves even to the normal quantity of blood, and the animal dies upon the immediate withdrawal of a quantity of blood even less than that injected. Within a few hours after the transfusion the quantity of blood-plasma is considerably lessened, and after two or three days it returns to the normal amount. This reduction is effected chiefly by increase in the urinary secretion. The excess in red blood-corpuscles persists somewhat longer. A true polycythæmia exists. But in a few days, at the most in from two to four weeks, the number of red blood-corpuscles is also reduced to the normal quantity. This destruction of red blood-corpuscles is indicated by increased excretion of urea. If blood be injected equal in amount to 150 per cent. of the total quantity of blood in the animal, death ensues with symptoms of exhaustion, vomiting, and hæmaturia.

If the blood of one species of animal be injected into that of another species, the effects are entirely different. If by transfusion of lamb's blood into the veins of a dog the quantity be increased only one-fifth, the death of the dog ensues, as a rule, on the first or second day. Even half of that quantity produces severe symptoms, especially hæmoglobinuria and a general tendency to hemorrhages. The injurious effects of the transfusion of foreign blood may be explained, in part, by the fact that the serum of the blood of certain species dissolves the red corpuscles of other species.

It has been discovered by Köhler[1] that the injection into the jugular vein of a small quantity of serum rendered rich in fibrin ferment by spontaneous coagulation of the blood, and subsequently straining through linen, causes often, although not invariably, the rapid death of the animal from coagulation of the blood in the right cavities of the heart, the pulmonary artery, and its branches. The transfusion of blood which has been defibrinated by stirring does not produce these harmful effects.

It is apparent that these experiments on animals throw much doubt upon the existence of true plethora in man, without absolutely disproving the possibility of its occurrence as a permanent condition. There is certainly no proof that the symptoms hitherto embraced under the name plethora depend upon either polyæmia or polycythæmia. It has been suggested that their cause is to be found in impaired function of the vaso-motor nerves, the regulators of the circulation.

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[1] *Ueber Thrombose u. Transfusion*, Dorpat, 1877.

Anæmia.

Local anæmia or ischæmia dependent upon local disturbances of the circulation has been already described (vide p. 25). Under the name general anæmia are included diminution in the mass of blood, or *oligæmia;* diminution in the number of red blood-corpuscles, or *oligocythæmia;* and diminution in the amount of hæmoglobin in the red blood-blood corpuscles, or *achroiocythæmia.* In many cases of anæmia some of the red corpuscles are much reduced in size. These small corpuscles are called *microcytes.*[1] These microcytes are regarded by some as red blood-corpuscles in process of formation, by others as atrophied or degenerated red corpuscles. *Hydræmia*, or diminution of the solid ingredients of the plasma, especially the albumen, is also an element in most forms of anæmia. But the essential element in anæmia is diminution in the hæmoglobin of the blood. It is to this loss of the coloring matter of the blood that the most obvious and characteristic symptom of anæmia—namely, the pallor—is due. It was formerly believed that the quantity of hæmoglobin in the blood is directly proportional to the number of red blood-corpuscles, but recent observations have shown that red blood-corpuscles in disease vary in the percentage of hæmoglobin which they contain, so that reduction in the amount of hæmoglobin in the blood does not necessarily involve a corresponding diminution in the number of red corpuscles. Still, a greater or less loss of red blood-corpuscles is an almost constant change in anæmia, and the cases are exceptional in which the extent of this loss is not an approximately correct index of the degree of anæmia.

In anæmia, therefore, we may have changes in the *quality* as well as in the *quantity* of red blood-corpuscles. On the one hand, the amount of hæmoglobin in individual corpuscles may be diminished, or, on the other hand, it may be increased. The normal amount of hæmoglobin (oxyhæmoglobin) in the blood is estimated at from 12 to 14 per cent. The amount may be reduced in anæmia even as low as to 2 per cent. Cases of chlorosis have been observed with the normal number of red corpuscles and with less than half the proper amount of hæmoglobin (achroiocythæmia). Instances of oligocythæmia have been recorded in which the hæmoglobin was not reduced in proportion to the number of red blood-corpuscles. In such cases the condition of the blood is better than would be indicated by simply counting the number of red blood-corpuscles.

In health the number of red blood-corpuscles in a cubic millimeter of blood is estimated at from four and a half to five millions. This number in anæmia is often reduced one-half, and in extreme anæmia it may fall even below half a million. The average diameter of the human red blood-corpuscles in health is between seven and eight micromillimeters. There are in health some corpuscles smaller and some larger than the average. In anæmia there is often a large number of corpuscles, called microcytes, whose size is between two and six micromillimeters, and on the other hand there may be some corpuscles measuring twelve to fifteen micromillimeters in diameter (megalocytes). The large corpuscles are found especially, although not exclusively, in pernicious anæmia. The shape of the corpuscles in anæmia may also deviate from that of the normal biconcave discs with round contours. The name *poikilocytosis* is used to designate the condition of blood in which the corpuscles present manifold variations of shape. Nucleated red blood-corpuscles are sometimes found in the blood in anæmia.

The total volume of blood is not necessarily reduced in anæmia out of proportion to the loss of weight of the body, except in the case of acute anæmia immediately following hemorrhage. It may, however, be disproportionately lessened. Thus in two cases of pernicious anæmia reported by Quincke the quantity of blood was estimated to be from 4 to 5 per cent. of the weight of the body, instead of 8 per cent., the normal proportion.

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[1] Some understand by microcytes only small, spherical red blood-corpuscles.

If the anæmia be severe and chronic, secondary changes are produced in the organs and tissues of the body. Of these changes none is more important or more characteristic than fatty degeneration of the heart, as has been emphasized especially by Ponfick. Fatty degeneration of the walls of the vessels, of the liver, the kidneys, and sometimes of the voluntary muscles, may also be induced, but in less degree. Ecchymoses and a hemorrhagic diathesis may be the result of profound anæmia. Atrophy of different organs of the body is a natural result of long-continued anæmia. The nervous centres suffer the least in their nutrition. An interesting change in the marrow of the bones has been observed in profound chronic anæmia, and is probably to be regarded as secondary. This change consists in the appearance of red in the place of the yellow medulla which is normally present in the long bones of adults. The fat disappears, the number of medullary cells is greatly increased, and a considerable number of nucleated red blood-corpuscles make their appearance. This alteration is a return of the yellow marrow to the condition it presents in fœtal and infantile life. Some think that this change in the osseous medulla has to do with the regeneration of red blood-corpuscles.

Although it has long been known that if the cause of anæmia be removed the red corpuscles are renewed in a comparatively short time, we possess little positive knowledge as to the mode of regeneration. Experiment and pathological observation point especially to the spleen and marrow of the bones, perhaps also to the lymphatic glands, as organs in which red as well as white blood-corpuscles may be formed. There are three leading views as to the new formation of human red blood-corpuscles in the adult. According to one view (Hayem's), they are formed from little bodies identical with the blood-plates of Bizzozero, and called by Hayem hæmatoblasts, which are found in the circulating blood; according to a second view (Neumann's), they are formed in the marrow of the bones from leucocytes by a transformation of the protoplasm into hæmoglobin and a subsequent disappearance of the nucleus; according to another view (Bizzozero's), they are formed in the marrow of the bones by indirect division of nucleated red blood-corpuscles. According to the two last views, which are more probable than the first, nucleated red blood-corpuscles represent the young or embryonic blood-corpuscles.

Anæmia may be divided into an acute and a chronic form. The best example of acute anæmia is that produced by copious hemorrhages. This also affords the purest example of oligæmia. A considerable amount of blood—in dogs over one-quarter of the entire mass—may be withdrawn from healthy individuals without permanent damage. The loss of one-half of the entire volume of blood is usually fatal. Females, as a rule, are more tolerant of loss of blood than males. Infants are especially susceptible to the evil effects of the withdrawal of blood. A large quantity of blood—in dogs over a quarter of the whole volume—may be removed without more than a temporary lowering of the blood-pressure. Under the influence of the vaso-motor nerves, which are affected by anæmia as by an irritant, the vessels adapt themselves within very wide limits to varying amounts of blood without permanent alteration of the blood-pressure. In healthy individuals a very considerable loss of blood is repaired within a short time, at the most in three or four weeks. The blood is very soon restored to its normal volume by the absorption of water. As the red blood-corpuscles and the albumen are not so easily renewed, the specific gravity of the blood is now diminished. A condition of hydræmia has replaced the oligæmia. The white blood-corpuscles are sooner restored than the red. In proportion to their number, there are fewer white than red corpuscles lost by hemorrhage. The former are of less weight, more viscid, and do not so readily escape from the vessels. Then the lymph, the flow of which is increased by severe hemorrhages, constantly brings new leucocytes to the blood, so that soon after a considerable hemorrhage the number of white corpuscles, in proportion to the red, is increased. A temporary relative leucocytosis is thus produced. The food restores to the blood its normal amount of albumen. The red-blood-corpuscles are also regenerated, although more slowly.

The symptoms of acute anæmia are of course grave in proportion to the amount of blood lost. They are weakness, pallor, coldness of the surface, feeble and rapid pulse, dimness of vision, dyspnœa, muscular spasms, especially in the calves of the legs, and, if the loss of blood be sufficient, unconsciousness and epileptiform convulsions from anæmia of the brain. Death results from hemorrhage, not in consequence of the loss of red blood-corpuscles or of any particular constituent of the blood, but from the diminution in the total volume of blood. To this diminished volume the heart and the blood-vessels cannot at once adjust themselves. The symptoms are more marked when the hemorrhage occurs in persons previously healthy than in those already enfeebled. The immediate treatment consists in means to arrest the hemorrhage, in maintenance of the recumbent posture with the head lower than the feet, and in the administration of stimulants. If transfusion be necessary, the same purpose is accomplished by the infusion of dilute solution of common salt (0.6 per cent.) as by transfusion of blood, and the former procedure is simpler and less dangerous than the latter.

The most important causes of chronic anæmia are the following: 1, repeated hemorrhages, as menorrhagia, or hæmatemesis; 2, the loss of essential constituents of the plasma, particularly albumen, as in chronic albuminuria, prolonged lactation, chronic dysentery and diarrhœa, or prolonged suppurations; 3, defective supply of nutriment (anæmia of inanition); 4, affections occasioning indigestion and vomiting, as chronic gastritis, or ulcer of the stomach; 5, chronic wasting diseases, particularly tuberculosis and cancer; 6, the poisons of chronic infectious diseases, as malaria, or syphilis; 7, certain mineral poisons, especially lead and mercury. Anæmia more acute in its development may attend acute infectious diseases, as yellow fever, typhoid fever, and the entrance into the blood of poisons which rapidly destroy the red corpuscles, as phosphorus and the biliary salts. There is, further, an important class of anæmias for which, with our present knowledge, no cause can be assigned. This class embraces leucocythæmia, pseudo-leucocythæmia, Addison's disease, chlorosis, and pernicious anæmia. Profound and often fatal anæmia, without apparent cause or obvious structural changes in the body, exclusive of those which may be secondary to the anæmia, is known as primary, idiopathic, essential, or pernicious anæmia. The most reasonable hypothesis as to these anæmias without known cause is that they depend upon disturbances in the blood-forming organs, which may be embraced under the generic name hæmatopoietic system. As these forms of anæmia constitute, for the most part, well-defined diseases, susceptible usually of diagnosis, it will be convenient to treat of them in Part II. of this work under the heading "Diseases of the Hæmatopoietic System," although, as has already been stated, our knowledge of the formation of blood-corpuscles after birth is incomplete.

As is evident from the enumeration of its causes, symptomatic anæmia is incident to a variety of diseases, in connection with which it will hereafter be referred to. It is proper here to mention the common and most diagnostic symptoms, although they may be modified by the primary disease. Such symptoms are pallor of the face and mucous membranes, impairment of muscular and mental energy, functional disorders of the nervous system, especially neuralgia and so-called spinal irritation, coldness of the surface, dyspnœa on exertion, impaired digestion, palpitation of the heart, a pulse either small or full, but compressible. It is important to note that in persons who have an unusual vascularity of the face anæmia may exist not only without notable pallor, but even with a rosy complexion. Of special importance in the diagnosis of anæmia is the presence of certain physical signs. There is often present in anæmia a soft bellows murmur accompanying the first sound of the heart, and heard most distinctly at the base of the heart and in the larger arteries, the carotid, the subclavian, etc. To constitute evidence of anæmia there must be wanting the signs of organic lesion of the heart and large vessels. In conjunction with this murmur a continuous humming sound, sometimes musical, is heard when the stethoscope is applied over the veins of the neck. This sound, due to the movement of blood in the veins, is called the venous hum, or, after the French, *bruit de diable*. These murmurs are distinguished from those denoting lesions, as inorganic, hæmic, or anæmic murmurs. The arterial murmur is present in only a certain proportion of cases, but the venous hum may be heard almost invariably.

Symptomatic anæmia is sometimes amenable to treatment, at other times not, according to the nature of the primary disease. It may be noted that anæmia is not incompatible with embonpoint. The first indication is to ascertain and remove if possible the cause or causes on which the anæmia depends. The most important measures in restoring the quality and quantity of red globules are—*first*, a nutritious alimentation, into which meat should enter largely; *second*, iron as a special remedy, the effect of which in increasing the amount of hæmoglobin and the number of corpuscles is often remarkable; *third*, other tonics, as arsenic, quinine, strychnine, and stimulants to render the digestive functions more active; and *fourth*, a regimen calculated to increase the energy of the assimilative functions, consisting in exercise in the open air, change of climate, recreation, etc.

Hydræmia.

By *hydræmia* is understood a relative or an absolute increase in the amount of water in the blood in proportion to the solid ingredients. It is the diminution in the amount of albumen which forms the chief element in hydræmia. Less emphasis is laid in this connection upon the loss of blood-corpuscles, although, as has been mentioned already, anæmia and hydræmia are usually associated. The salts and extractive matters of the blood are left out of consideration. Hydræmia is essentially hypalbuminosis, relative or absolute. There are three possible forms of hydræmia: in the *first*, the amount of water is normal, but the solids are diminished; in the *second*, the solids are normal in quantity, but the amount of water is increased; in the *third*, the amount of solids is diminished, and that of the water is increased. In the third form the highest degree of hydræmia is reached. The various causes of anæmia, involving loss of the solid constituents of the blood, are also causes of hydræmia. The most extreme hydræmia is that produced in many cases of chronic nephritis or Bright's disease, in which there is not only a continual drain of albumen from the blood, but the excretion of water by the kidneys is lessened. The conditions are here present for the production of the third form of hydræmia. In Bright's disease the specific gravity of the blood-serum has been known to sink from 1030 to 1013, the percentage of albumen from 8 to 4, corresponding to an increase in the amount of water from 90 to 95 per cent.

General œdema has long been considered to be a most characteristic symptom of hydræmia. The abundant serous transudations in various parts of the body in Bright's disease have been referred directly to the extreme hydræmia of this disease. It was naturally supposed that the less concentrated the plasma, the more readily it would transude through the walls of the vessels. The experiments of Cohnheim and Lichtheim,[1] however, have demonstrated that hydræmia, even when present in a degree never met with in man, is not a direct cause of general œdema, but that this œdema follows only when the nutrition of the vascular walls is impaired and they are thereby rendered more permeable. Inasmuch, however, as the hydræmia favors this weakening of the vessel-walls, it still must be considered an important indirect cause of dropsy.

Anhydræmia.

A too concentrated state of the blood in consequence of an absolute increase in its solid constituents is not a recognized pathological condition. We know nothing of absolute *hyperalbuminosis* as a morbid state of the blood. *Anhydræmia*, or a thickened condition of the blood from loss of water, is familiar to us in man only as a change resulting from excessive serous discharges from the intestine, particularly as a result of cholera. In cholera the blood may become so concentrated as to flow with difficulty and to present an almost tarry consistence. As there is an effort to repair the loss of water by absorption, the organs and tissues become shrunken and dry, the secretions are diminished or checked, the circulation is slow, the blood-pressure is reduced, and the pulse becomes feeble or imperceptible. The appearance of the salts of potassium in the plasma indicates that red blood-corpuscles are destroyed. If reaction ensue after the choleraic discharges cease, the blood is rapidly restored to its normal percentage of water by the absorption of fluids taken into the system.

Hyperinosis; Hypinosis.

An abnormal increase in the amount of fibrin in the blood constitutes *hyperinosis;* an abnormal diminution of the fibrin is called *hypinosis.* In former times, when venesection was more frequently practised, physicians paid much attention to the amount of fibrin in the blood, to the rapidity with which it coagulates, and to the appearance of the coagulum. It is now recognized that many fallacies existed in the former methods of analysis, and that estimates of the fibrin do not have the diagnostic value formerly attributed to them. The statements formerly made as to increase or diminution of fibrin in the blood were based upon the amount of coagulum formed after death or in blood withdrawn by venesection, or upon the rapidity with which coagulation occurred. Fibrin, however, does not exist preformed in the blood. The quantity of fibrin which coagulates from the blood is believed to depend less upon the amount of fibrin-forming substance in the blood than upon the presence or absence of conditions which favor or impede the coagulation.

Imperfect coagulation of the blood, or hypinosis in the old sense, has been observed in some acute infectious diseases, in acute icterus, in death from asphyxia, and in death from certain poisons, such as sulphuretted hydrogen and hydrocyanic acid. Increase of fibrin, or hyperinosis, occurs in inflammations, in pneumonia, in acute rheumatism, and in erysipelas.

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[1] *Virchow's Archiv*, Bd. 69, p. 106.

The so-called *buffy coat*, or *crusta inflammatoria*, is due to the slow coagulation of the fibrin, so that the red corpuscles have time to sink and leave the upper layers of the coagulum uncolored. This appearance in blood removed by venesection is not characteristic of inflammation and has no diagnostic value.

Leucocytosis.

According to the nomenclature proposed by Virchow, a temporary increase in the number of white corpuscles in the blood is called *leucocytosis;* a permanent and usually much greater increase constitutes the disease called *leucocythæmia* or *leukæmia.* The number of white corpuscles in the blood is subject to much greater variation, within normal limits, than is that of the red corpuscles. The proportion is usually estimated as 1 white to 350–500 red. Leucocytosis, indicated by a moderate increase in the number of white corpuscles, has been observed, as a physiological condition, during digestion and in pregnancy. Leucocytosis is present in inflammations attended with profuse suppuration. It is also observed in fevers. A relative, and sometimes an absolute, leucocytosis is often present in anæmic conditions, as already mentioned. The increase in white corpuscles in these conditions rarely approaches in degree that found in the disease leucocythæmia, in which the proportion of white to red may be as 1 to 10, 1 to 3, and even in one case 3 white were present to 2 red. In leucocythæmia the number of red corpuscles is diminished, and the symptoms are mainly those of anæmia. This disease will be considered in Part II. of this work, in the section treating of diseases of the hæmatopoietic system.

Alterations in the Gases of the Blood.

The gases contained in the blood are oxygen, carbonic acid, and nitrogen. The oxygen is, for the most part, in unstable combination with hæmoglobin in the form of oxyhæmoglobin. There may be also a small amount of oxygen simply dissolved or absorbed in the plasma. The oxygen may be easily driven out of the blood by certain other gases, as carbonic oxide, or by a vacuum. Carbonic acid gas exists in the plasma and in the red corpuscles of the blood only in combination with alkalies or alkaline salts; none is held in simple solution (P. Bert). Nitrogen is simply absorbed. Of course, the stable combinations of these gases in the blood do not here enter into consideration.

Probably the only influence which can *increase* the quantity of oxygen in the blood sufficiently to give rise to morbid symptoms is the inhalation of compressed air; and here the evil effects are due less to the increased quantity than to the increased tension of the oxygen in the blood.[1] Death in compressed air may result as well from excess of carbonic acid in the blood as from excess of oxygen. The effects of changes in the barometric pressure have been ably studied by Paul Bert,[2] who investigated the influence of compressed and of rarefied air upon men and animals. He found that evil effects are felt under a pressure of six atmospheres; that in animals convulsions appear when the pressure reaches twenty atmospheres; and that death rapidly ensues when the pressure equals twenty-five atmospheres. As the hæmoglobin in normal arterial blood is nearly or quite saturated with oxygen (Herter), it is mainly the dissolved oxygen of the plasma which is augmented by inhaling compressed air. According to Bert, death ensues when the proportion of oxygen in the arterial blood is augmented one-third. Under the influence of this superoxygenation of the blood the oxidation of the tissues is diminished, the production of carbonic acid, the excretion of urea, and the destruction of sugar in the blood are lessened, and as a result the temperature is lowered.

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[1] G. v. Liebig (*Münchener ärztl. Intelligenzbl.*, No. 19, 1879) explains the injurious influence of compressed air by the mechanical hindrance which it offers to expiration.

[2] *La Pression barométrique, etc.*, Paris, 1878.

The study of the effects of high atmospheric pressure upon man has acquired especial interest in consequence of the extensive use made of compressed air in engineering operations. In diving-bells, in mines in which compressed air is used to keep water from flowing into the shafts, in the caissons employed in the sinking of piers, men are subjected to the influence of compressed air, but the degree of compression rarely suffices in itself to produce grave symptoms. Some inconvenience may be felt from pain in the ears and sometimes in the frontal and maxillary sinuses. There is, however, danger in passing rapidly from highly-compressed air into the normal atmosphere if the stay in the former have been long. A number of fatal accidents have occurred from ignorance or negligence in this respect. Hoppe-Seyler, and subsequently Bert, found that if the pressure have been considerable and it be too rapidly removed, nitrogen is set free in the blood in the form of gaseous bubbles, which, collecting in the right side of the heart, stop the circulation and cause sudden death. Nitrogen is absorbed by the blood in accordance with Dalton's law for the absorption of gases by fluids, and consequently accumulates in the blood in considerable quantity in compressed air. There have been observed occasionally to follow the removal of the pressure certain symptoms, to which A. H. Smith[1] has given the name of the *caisson disease*, and the explanation of which is not established. The most frequent and important of these symptoms are pain in one or more of the extremities, and sometimes in the trunk, epigastric pain and vomiting, paralysis more or less complete, general or local, most frequently confined to the lower half of the body, headache, and vertigo. According to Smith, the duration of the caisson disease varies from three or four hours to six or eight days. The paralysis may disappear within twelve hours or may continue for weeks. Death occurs only in cases which are severe from the first. Smith refers the symptoms, at least in severe cases, to congestion of the brain and spinal cord. It is, however, not improbable that at least some of the symptoms may be due to the liberation of bubbles of nitrogen in moderate amount or in situations not immediately endangering life. Morphine, atropine, and especially ergot, were found useful in relieving the pain. The main reliance, when the symptoms are urgent, and especially when life is endangered soon after removal of the pressure, is return to compressed air. Bert has proposed the inhalation of oxygen to displace the free nitrogen from the blood by diffusion.

Air compressed not more than from one to five atmospheres has been employed as a therapeutical agent, chiefly in certain pulmonary disorders. The influence of moderately-compressed air is to diminish the number of pulsations of the heart and to increase the capacity of the lungs by compression of the intestinal gases. According to Bert, the maximum of oxidation takes place under a pressure of three atmospheres.

A condition attributed to excess of oxygen in the blood is a temporary cessation of the respiratory acts which is observed to follow violent and full inspirations, and which has been studied chiefly by physiologists on animals, the condition being induced by vigorous artificial respiration. To the cessation of breathing as thus produced the term *apnœa* is limited by some German writers; but this term, as usually applied, embraces the various conditions which occasion dyspnœa. In the latter comprehensive sense, inclusive of serious disturbances of hæmatosis, whether accompanied or not by a sense of the want of breath, or dyspnœa, the term will be used in this work.

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[1] *The Effects of High Atmospheric Pressure*, Brooklyn, 1873.

Deficiency of oxygen in the blood is of much greater pathological importance than its increase. If the quantity of oxygen in the blood be greatly diminished, there follows a group of symptoms to which the names *suffocation*, *asphyxia*, and *cyanosis* are applied. Causes which reduce the amount of oxygen in the blood sufficiently to produce suffocative symptoms are—1, diminution in the supply of oxygen to the pulmonary capillaries, as from breathing in rarefied air and in small closed spaces; also from obstruction or compression of the air-passages, as in croup; from diseases which lessen the respiratory surface of the lungs or interfere with their function, as pneumonia, phthisis, pneumothorax, asthma, œdema; from interference with the respiratory centre, as in certain affections of the central nervous system and in narcotic poisoning; 2, obstruction to the circulation in the lungs, as from embolism of the pulmonary artery and in valvular lesions of the heart; 3, diminution in the amount of hæmoglobin in the blood, such as results from profuse hemorrhages; 4, displacement of oxygen from its combination with hæmoglobin by other gases which enter into a firmer combination, as carbonic oxide, from which many intentional and accidental cases of poisoning have resulted (nitric oxide also displaces the oxygen, but being irrespirable need not here be considered); 5, rapid reduction of oxyhæmoglobin by gases which seize the oxygen, such as sulphuretted hydrogen, and probably also phosphuretted, arseniuretted, and antimoniuretted hydrogen, which further decompose the blood. Mention here should also be made of the asphyxia of new-born children (asphyxia neonatorum), which is due to separation of the placenta or to closure of the umbilical vessels before birth. After death from rapidly-produced suffocation the blood is of a dark color, and usually, although not always, fluid or imperfectly coagulated; the right cavities of the heart are, as a rule, distended with blood, the mucous membrane of the larynx and trachea is congested; and ecchymoses are frequently present beneath the pleura and pericardium. In consequence of the fluid condition of the blood there is hypostatic congestion of most organs, and unusually marked livid spots (*livores mortis*) on the most dependent parts of the surface of the body. Chemical analysis of the blood just before death shows the oxygen to be nearly or entirely absent and the carbonic acid to be more or less increased in amount. After poisoning by carbonic oxide, the blood is bright red instead of dark, unless this gas has been partly or wholly converted into carbonic acid.

The symptoms of suffocation are, at first, dyspnœa, as indicated by increase in the rapidity or in the depth of the respiratory movements, or in both; convulsions, which are usually absent when the suffocation is not rapidly induced, and which even in rapid suffocation may fail, as in drowning; lowering of the temperature; elevation of the blood-pressure; at first slowing of the pulse from irritation of the vagi, then increased rapidity from paralysis; dilatation of the pupils; protrusion of the eyeballs (exophthalmos), and a dark blue, so-called cyanotic hue of the surface of the body. At a late stage these symptoms may become considerably modified. This is the asphyctic stage proper. In it the color becomes less dark, the dyspnœa is lessened or disappears, the pulse is small and frequent, reflex excitability is lessened, and finally unconsciousness, anæsthesia, and death follow. Sugar may appear in the urine in cases of suffocation. The symptoms which have been enumerated are modified in individual cases, especially by the rapidity with which the oxygen in the blood is lessened in amount. Dyspnœa, with more or less marked suffocative symptoms, is an important element in many diseases which will be considered in Part II. of this work. There has been much dispute as to whether the symptoms of suffocation, particularly dyspnœa, are due to the loss of oxygen in the blood or to the excess of carbonic acid. It is now quite certain that in rapid suffocation the dyspnœa is due chiefly to the diminished quantity of oxygen. It is possible to produce all of the symptoms of asphyxia in animals by lessening the amount of oxygen without increasing the amount of carbonic acid in the blood (Pflüger), and although a large accumulation of carbonic acid in the blood also causes dyspnœa, the amount of this gas found in the blood in most cases of asphyxia is not sufficient to explain the symptoms. The treatment consists in removal of the cause, if possible, and in efforts to increase the quantity of oxygen in the blood. This increase is best effected by the employment of artificial respiration and by the inhalation of oxygen. In poisoning by carbonic oxide the transfusion of blood has proved successful. Special therapeutical indications will be considered in the second part of this work.

Although, as has been mentioned, in most cases of suffocation there is an excessive amount of carbonic acid in the blood, this excess does not usually suffice to produce dyspnœa. It is, however, possible to produce this symptom by inhaling air containing a large quantity of carbonic acid, even if there be no diminution of oxygen. The asphyxia is developed more slowly than when resulting from lack of oxygen. The experiments of Raoult[1] have shown that the presence of carbonic acid in the inspired air diminishes the quantity of carbonic acid produced in the body, and especially the amount of the oxygen consumed. The experiments of Friedländer and Herter[2] upon animals demonstrate that if the quantity of oxygen be normal, inhalation of air containing an excess, but less than 20 per cent., of carbonic acid produces symptoms of irritation, such as increased frequency of respiration and increased blood-pressure, but no really poisonous effects; but if the amount of carbonic acid be increased to about 30 per cent., symptoms of depression follow, the breathing becomes slower and weaker, the blood-pressure sinks, voluntary and reflex movements are weakened and finally abolished, the temperature falls, and the animal dies in the course of a few hours. If the quantity of carbonic acid be increased to the maximum, the symptoms of depression follow very rapidly, and death occurs frequently within half an hour. According to Bert, symptoms of carbonic-acid poisoning do not appear until all of the alkali in the blood has been saturated with carbonic acid and the gas begins to be dissolved in the plasma.

Changes in the Albumen, Fat, and Inorganic Salts of the Blood.

These changes can be dismissed with a few words. Enough has already been said in connection with hydræmia and anhydræmia concerning alterations in the quantity of albumen. Fat is probably always present in the blood-plasma in health. Its average amount is estimated at 2 to 3 parts per 1000, but the quantity varies considerably within normal limits. The amount of fat in the blood is increased during the digestion of fatty substances, when the blood may acquire a milky or chylous appearance from the molecules of fat. In diabetes the blood often has a slightly milky appearance from an increased amount of fat. This condition of the blood is called *lipæmia.* It is said that in certain diseases of the liver, in phthisis, and in chronic alcoholismus there is increased amount of fat in the blood. This chylous condition of the blood is, of course, not to be confounded with the entrance of oil-drops into the blood after fractures and in certain morbid conditions. This fatty embolism has already been described. (See *Embolism and Thrombosis.*) We are not able to assign any pathological importance to a chylous condition of the blood.[1]

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[1] *Comptes rendus*, t. 82, p. 1101.
[2] *Zeitschr. f. Physiol. Chemie*, Bd. 2, p. 99, 1878.

Although the inorganic salts are present only in small amount in the blood (8 parts to 1000, of which about one-half is chloride of sodium), they undoubtedly have an important part in the vital processes. The effects of their withdrawal from the food are described in works on physiology and can hardly claim pathological importance. Some writers have attributed the symptoms of scorbutus to a deficiency of alkaline carbonates or to a lack of potash salts in the blood, but we possess no very satisfactory chemical analysis of the blood of scorbutic individuals. There seems to be no diminution in the chlorides of the blood in pneumonia and in the fevers, notwithstanding sometimes an entire absence of chlorides in the urine in these affections. The diseases rachitis and osteomalacia are supposed by many to depend upon an insufficient amount of the salts of lime in the blood. According to C. Schmidt, there is a definite relation between the quantity of albumen and that of salts in the blood, diminution in the albumen being attended by increase in the salts.

CHAPTER VII.

GENERAL PATHOLOGY OF THE BLOOD (CONCLUDED).

Glycohæmia—Acetonæmia—Uræmia—Ammoniæmia—Uricæmia or Lithæmia—Cholæmia—Cholesteræmia—Melanæmia—Septicæmia—Pyæmia.

Glycohæmia.

GLYCOHÆMIA signifies the presence of sugar in the blood. A small amount of grape-sugar exists normally in the plasma of the blood. This normal amount is estimated at from 1 to 2½ parts per 1000. The quantity of sugar is about the same in venous and in arterial blood, and does not seem to be essentially influenced by the diet, beyond the fact that sugar is absorbed from the intestine by the portal vein.

If the quantity of sugar in the blood exceed a certain amount (2½–3 parts per 1000, according to Bernard), sugar appears in the urine, constituting the condition called *glycosuria*. Many physiologists indeed believe that a trace of sugar is present in the normal urine, the quantity being so small as to be unrecognizable by the ordinary tests. Glycosuria may be a temporary condition in health and in disease, or it may be long continued. Prolonged glycosuria is attended by a group of characteristic symptoms, and is called *diabetes mellitus*, a disease which will be considered in Part II. of this work. Certain considerations pertaining to the pathogeny of glycohæmia as a morbid state find their place appropriately under the general pathology of the blood. The amount of sugar in the blood of diabetic individuals may be 9 parts to 1000 of serum (Hoppe-Seyler). There has been found in diabetes increased decomposition of the nitrogenous constituents of the body, as indicated by an excess in the excretion of urea out of proportion even to the large amount of ingesta. Quincke found an increased amount of iron in various organs of the body, indicating increased destruction of red blood-corpuscles. In the milder cases of diabetes the sugar may be made to disappear from the urine by withholding saccharine and amylaceous articles of food; in the more severe cases the sugar remains, although in diminished amount, during a diet of strictly animal food. Permanent glycosuria may appear as a symptom of certain affections of the central nervous system, particularly tumors and hemorrhages involving the medulla oblongata. With this occasional exception there are found in diabetes mellitus no pathological changes to which the increase of sugar in the blood can be referred with any certainty.

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[1] Sanders and Hamilton (*Edin. Med. Journal*, vol. ii. p. 47, 1879), however, consider lipæmia and the resulting fatty emboli as the cause of the dyspnœa and other symptoms of diabetic coma.

There are many theories as to the nature of diabetes mellitus, but none has yet obtained general acceptance. Most of these theories are little more than conjectures based chiefly on physiological experiments, the results of many of which are still in controversy, or, with our present knowledge, neither admit of interpretation nor shed much light upon diabetes as it occurs in man. A temporary glycosuria can be produced in animals in a great variety of ways, of which the longest known and most celebrated is puncture of the floor of the fourth ventricle, the *piqûre* of Bernard. This is thought to cause vaso-motor paralysis of the blood-vessels of the liver, and to hasten thereby the circulation through the organ. Among other methods of producing glycosuria experimentally, may be mentioned injuries of various parts of the brain and of the spinal cord, destruction of sympathetic ganglia or division of sympathetic nerves in different situations, irritation of the depressor nerve, poisoning with curare, carbonic oxide, and most narcotics, injection of large quantities of solutions of common salt into the blood, and, in cats, simple fixation and tracheotomy.

There are two possibilities as to the accumulation of sugar in the blood. One is that there is increased access of sugar to the blood; the other, that there is diminished consumption of sugar in the system. An increased amount of sugar has been supposed to come from different sources—from the liver, from the intestine, from the muscles, or from the tissues in general. The theory of the *hepatogenous origin* of diabetes mellitus is the one which has been most widely accepted since the researches of Bernard upon the glycogenic function of the liver. There are two modifications of the theory that in diabetes an increased quantity of sugar passes from the liver into the blood. In order to understand these two forms of the hepatogenous theory, it is necessary to know that it is thought not only that glycogen is formed out of sugar, but that it is also converted into sugar. According to one view, in diabetes the production of sugar in the liver is abnormally great. This may be due to increased formation of glycogen or of sugar-ferment, or it may be the result of too rapid passage of glycogen from the hepatic cells into the blood, where it is converted into sugar. Pavy supposes that the liver produces sugar in diabetes, but not in health. Another form of the hepatogenous theory is that the sugar which is brought to the liver by the portal vein, whether in normal or in abnormal quantity, is not converted into glycogen, but passes unchanged through the liver into the hepatic vein. According to the former view, there is an increase of the sugar-forming function of the liver; according to the latter, its glycogen-forming function is interfered with. Many of the advocates of each view hold that the primary change is increased rapidity of the portal circulation in the liver. This assumption is based upon experiments intended to show that temporary glycosuria is produced in animals by paralytic distension of the blood-vessels of the liver. The theory that diabetes depends upon an increased production of sugar in the liver is founded on the doctrine that the formation of sugar from glycogen is a normal function of the liver. This theory has been opposed by investigations which have resulted in a denial of the existence of a sugar-forming function in the liver.[1]

In favor of the theory that diabetes mellitus is due to the non-conversion of sugar into glycogen in the liver, the following experiments have been adduced: If sugar be injected into the jugular or the crural vein of an animal, it soon appears in the urine, whereas a larger amount may be injected into the mesenteric vein without causing glycosuria. Cases are reported in which the ingestion of sugar or of amylaceous substances has induced glycosuria in cirrhosis of the liver, a disease in which a considerable part of the hepatic parenchyma is destroyed and the portal circulation is obstructed. Bernard produced glycosuria by feeding with sugar and amylaceous substances animals in which the portal vein was closed. On the other hand, it has been shown that puncture of the fourth ventricle does not cause glycosuria if the animal has been poisoned with arsenic or phosphorus, substances which induce fatty metamorphosis of the liver.

The theory of Huppert is perhaps the best presentation of the view that diabetes mellitus depends upon diminished oxidation of the sugar. The researches of Pettenkofer and Voit regarding the nutritive changes in a diabetic patient led them to advocate this theory. According to these investigators, diabetes mellitus is due to a general disturbance of nutrition, in consequence of which the albuminous constituents of the body undergo abnormally rapid metamorphosis. Proof of this they find in the amount of urea excreted, which exceeds that produced from the ingesta. Hence diabetic individuals rapidly emaciate. Sugar, like urea, they consider to be a normal product of the decomposition of albuminous substances. In health this sugar is rapidly oxidized. In diabetes it is claimed that less oxygen is absorbed than normal, notwithstanding the increased amount of oxidizable substances in the blood. This diminution in the absorption of oxygen is attributed to destruction of red blood-corpuscles, or to their abnormal constitution in consequence of their participation in the general malnutrition of the body. Inasmuch as the sugar produced in increased amount by metamorphosis of albumen does not find the proper quantity of oxygen for its combustion, it accumulates in the blood and causes abnormal glycohæmia. The observations of Quincke[2] are not in harmony with Pettenkofer and Voit's explanation of the diminished absorption of oxygen. Quincke found not only no reduction in the amount of hæmoglobin in the blood in diabetes, but in one case even an actual increase. The diminished consumption of the sugar in diabetes has been attributed also to a reduction of the alkalinity of the blood, and to the absence of some ferment which is supposed under normal conditions to disintegrate the sugar.

Dickinson[3] believes that diabetes mellitus is always the result of changes in the central nervous system. The most important of these changes he considers to be dilatation of the arteries and of the perivascular spaces, and minute hemorrhages with disintegration of nerve-elements. Changes similar to those described by Dickinson, however, have been found in other diseases, and are not constant in diabetes. While it is true, as has been mentioned, that in certain cases of diabetes mellitus marked changes have been found in the brain, especially in the pons and medulla oblongata, there is not sufficient evidence for believing that all cases of diabetes are of nervous origin.

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[1] Abeles, *Œstr. med. Jahrb.*, 1875, p. 269; Von Mering, *Arch. f. Anat. u. Phys.*, 1877, p. 379; Bleile, *ibid.*, 1879, p. 59.

[2] *Virchow's Archiv*, Bd. 54, p. 542.

[3] *Med. Times and Gaz.*, March 9, 1870, and *Treatise on Diabetes*, 1875.

It can serve no useful purpose to describe other theories which have been proposed or to attempt to criticise further those which have been cited. They all rest upon too uncertain a foundation. Until we have more positive knowledge as to the source of sugar in the healthy organism, and as to its metamorphoses, we cannot hope for any satisfactory explanation of the abnormal accumulation of sugar in the blood and in the urine in diabetes mellitus.

The transient appearance of a small quantity of sugar in the urine in various diseases has no more pathological importance than the occasional appearance of a trace of albumen.

Acetonæmia.

Attention was first called to acetonæmia by the recognition, in the urine and in the expired air of diabetic patients, of some substance which imparts an aromatic, fruity, chloroform-like odor resembling that of acetone. Acetone, or some substance yielding acetone, was proven to be frequently present in the urine in cases of diabetic coma. The theory of poisoning of the blood by acetone was constructed to explain these cases of coma. In many of the cases there was found in the urine a substance which imparted to it a burgundy-red color upon the addition of a solution of ferric chloride (Gerhardt's reaction). This reaction was considered proof of the presence of acetone or of an acetone-yielding substance.

There is no proof that acetonæmia is a condition of any pathological importance. The usual tests for the recognition of acetone in the urine are not very satisfactory. As to the occurrence of acetone in the urine, however, in a variety of conditions, there can be no doubt. It is even claimed by Von Jaksch that acetone is a normal product of tissue-metamorphosis, and that a minute quantity is constantly present in the urine. Pathological acetonuria he finds in fevers, diabetes, cancer, and various diseases of the gastro-intestinal tract. The substance which responds to the ferric-chloride test is not acetone, but it is probably diacetic acid.

There is no proof that acetone, diacetic acid, or ethyl-diacetate possesses toxic properties in the human organism. The coma and other disturbances attributed to them may occur without their presence, and, on the other hand, acetonuria and diaceturia may exist without any characteristic symptoms.

Uræmia.

By *uræmia* is understood the accumulation in the blood of excrementitious substances of the urine. The term is usually applied to a group of symptoms which appear when the function of the kidneys is interrupted or much impaired. The most important of the uræmic symptoms are coma and epileptiform convulsions, preceded often by headache, vomiting, and diarrhœa. The convulsions usually precede the coma, but either may appear alone. Amaurosis, dyspnœa, and maniacal delirium are also to be included among the occasional manifestations of uræmia. In the majority of cases uræmia is the result of some form of acute or chronic diffuse inflammation of the kidneys (Bright's disease). It may attend any disease in which the excretion of urine is more or less completely suspended, as double hydronephrosis, stricture of the urethra, cystitis, etc. In uræmia, as a rule, the quantity of urine, and especially the quantity of urea excreted, are diminished, the urine is albuminous and contains casts, and dropsy is present or it has preceded the attack. There are, however, exceptions, especially as regards the quantity of urine and the presence of dropsy. We cannot account for the fact that in one case uræmia appears, and in another it fails, although the conditions are apparently the same in both. Notwithstanding much research, no thoroughly satisfactory explanation of the uræmic phenomena has yet been reached.

The oldest and still most prevalent theory is that uræmia is due to the accumulation of urea in the blood. The arguments which have been adduced in support of this view are that urea, introduced into the blood of animals, produces symptoms similar to those of uræmia in man, and that in uræmia urea is in excess in the blood. In opposition to this view, however, the majority of experimenters agree that the injection even of large quantities of urea into the blood of healthy animals is harmless.[1] Urea may be fed freely to dogs without injurious effects if the animals be allowed to drink water. Inasmuch as urea is rapidly eliminated by healthy kidneys, these experiments do not prove that urea is innocuous when its excretion from the body is checked. In order to determine the effects of urea under conditions in which it cannot so readily be eliminated, it has been injected into the blood of animals whose kidneys have been removed. Uræmic symptoms develop in nephrotomized animals which survive the operation, in every case sooner or later. That the appearance of these symptoms is hastened by the injection of urea has not been established. Voit found that symptoms resembling those of uræmia are produced if the rapid elimination of urea fed to dogs be prevented by withholding water. The same effects were observed after feeding in the same way some other substances, such as benzoate of soda. Voit, therefore, does not attribute specific poisonous properties to urea, but thinks that uræmic symptoms appear when the accumulation in the blood of a variety of substances, including urea, uric acid, potash salts, creatinin, and extractive matters, becomes so great that the products of tissue-metamorphosis cannot be carried off. These waste products collect not only in the blood, but in the tissues, and here check that interchange between the tissue-elements and the nutritive fluids which is essential to the proper performance of cell-function. The results hitherto obtained by experimentation with urea cannot serve as a basis of support for the doctrine that urea is the sole poisonous agent in uræmia.

The blood of uræmic individuals has been analyzed with the view of determining the injurious ingredients. The blood of animals in which the kidneys have been extirpated or the ureters tied has been found to contain urea in tenfold its normal amount if the animals survive the operation from two to four days. These experiments show that urea is not formed exclusively in the kidneys, but that it is produced elsewhere and conveyed to these organs by the blood. Normal blood contains 0.01 to 0.08 per cent. of urea. The quantity of urea has been found much increased in the blood of uræmic patients. Bartels mentions a case in which 0.8 per cent. of urea was found. Hoppe-Seyler found 0.127 per cent. These amounts are larger than have usually been found in uræmia. But other excrementitious substances as well as urea are present in excess. A notable increase of extractive matters (0.86 per cent., Hoppe-Seyler) has been found, the importance of which is not to be underestimated because they have hitherto defied chemical analysis. There is one factor which disturbs the certainty with which urea accumulates in the blood when the function of the kidneys is suspended—namely, the vicarious elimination of urea by other organs. Urea may be excreted vicariously by the stomach, intestine, and skin. It has also been found in larger percentage in dropsical effusions than in the blood. In the stomach and intestine urea is readily converted into carbonate of ammonia. Urea has repeatedly been observed in uræmia as a crystalline deposit upon the skin. The bile has also been found to contain an abnormal amount of urea.

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[1] Feltz and Ritter found that when urea in large doses produced convulsions it was always contaminated with ammonium salts. It never produced convulsions when pure (*Compt. rendus*, t. 86, No. 15, 1878).

Picard recently finds, in opposition to most observers, that the rapid injection of concentrated solutions of urea into the jugular vein of dogs produces uræmic symptoms with fatal termination. He does not attribute the symptoms to the direct action of the urea, but to suspension of the renal excretion (*Gaz. méd. de Paris*, 1879, No. 5).

While urea has been found in excess in uræmic blood, there have been also cases of uræmia in which the amount of urea in the blood has been slightly, if at all, increased. In one case Jacobsen[1] found the quantity of urea too small to admit of quantitative estimation. On the other hand, a large quantity of urea has been found in the blood without the coincidence of uræmic symptoms. Unless in these analyses there has been some error, it seems quite certain that there are cases of uræmia in which the symptoms are not due to the retention of urea in the blood.

A theory of uræmia elaborated by Frerichs with great skill, which obtained at one time considerable credence, is now generally discredited, at least as regards its applicability to most cases. This theory is that uræmic symptoms are due to the presence in the blood of carbonate of ammonia produced by decomposition from the urea. There is no evidence for Frerichs's assumption that urea is decomposed into carbonate of ammonia in the blood. This decomposition, however, takes place in the stomach and intestine, carbonate of ammonia having been recognized often in the vomit of uræmic patients. There is no doubt that carbonate of ammonia may be absorbed by the blood from the intestine, and that in certain cases, which are rarer than was once supposed, it may be detected in the breath. It is true that the injection of carbonate of ammonia into the blood of animals produces more marked symptoms than does the introduction of urea. The most constant of these symptoms are convulsions, followed often by coma, with infrequency of the pulse and respiration. But while carbonate of ammonia always produces one and the same group of symptoms, mainly those of irritation, on the other hand the manifestations of uræmia are various, and only in a certain proportion of cases are they analogous to those produced by the ammonia salt. Carbonate of ammonia has been detected in small quantity in the blood in uræmia, but it has also been found to be absent in many cases.

Traube referred the uræmic phenomena to cerebral œdema and consequent cerebral anæmia. The œdema he attributed to hydræmia and high arterial pressure—conditions which are often, but not invariably, present in uræmia. Œdema and anæmia of the brain are found when no uræmic symptoms have been manifested during life, and they are by no means constantly present after death from uræmia. Œdema of the brain may often be regarded with more propriety as the result rather than as the cause of convulsions.

It is held by some writers that uræmia is not always produced in the same way, but that in different cases the causes are different. Bartels was an eminent advocate of this eclectic view. Jaccoud attempts even to diagnosticate between uræmia due to toxæmia and that due to œdema of the brain. But no essential differences have been detected, as regards symptoms, between cases of uræmia in which the blood was loaded with urea and those in which no excess could be determined; between those in which carbonate of ammonia was found in the blood and those in which it was absent; or between cases accompanied with hydræmia and those without this pathological condition.

It is only established with regard to the cause of uræmia that this condition is the result of an abatement of the function of the kidneys, and that this abatement is followed by an accumulation of excrementitious substances in the body. It is inconceivable that the accumulation in the blood and tissues of waste products should not have an injurious effect. Perhaps the following explanation of the uræmic phenomena is most in harmony with clinical and experimental observations: The uræmic symptoms are the result of the retention in the system of excrementitious materials. It is the entire mass of waste products, and not any single element, which is the source of trouble. The nature of these waste materials is very imperfectly known. They probably consist mainly of nitrogenous substances in different conditions of the metamorphosis of which urea is the final stage. The non-elimination of the excretory products causes an incomplete metamorphosis of the waste nitrogenous elements. These are retained, not only in the blood, but finally in the tissues. The nutritive processes are thereby disturbed. As the uræmic symptoms are chiefly of nervous origin, it is necessary to emphasize especially the disturbance of nutrition of the nervous system.

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[1] *Ziemssen's Cyclopædia of the Practice of Medicine*, Eng. trans., vol. xv. p 130.

Ammoniæmia.

The occasional absorption of carbonate of ammonia produced from urea excreted by the intestine has been mentioned already. Whether the amount of carbonate of ammonia which thus gains access to the blood ever suffices to produce poisonous symptoms is uncertain. There is, at least, not sufficient ground for believing that uræmia is, in reality, ammoniæmia. An ammoniacal state of the blood has also been referred to the absorption of carbonate of ammonia formed in decomposed urine in cases of retention of urine and of cystitis, from stricture of the urethra, enlarged prostate, paralysis of the bladder, pyelitis, etc. Musculus succeeded in isolating from the urine, in a case of cystitis, a ferment capable in a short time of transforming urea into carbonate of ammonia. Pasteur and Joubert found this ferment only when bacteria were present, and they believe that it is produced by these organisms. Leube and Graser have isolated no less than five different forms of bacteria, some from ammoniacal urine and others from the air, which are capable of transforming urea into carbonate of ammonia. The organisms which cause ammoniacal decomposition of the urine gain access to the urine from outside of the body, chiefly by catheterization. Some writers have considered ammoniæmia as the cause of the symptoms often observed in the late stages of cystitis and of pyelitis. These symptoms are irregular chills, fever, dryness of the mucous membranes exposed to the air, vomiting, diarrhœa, delirium, somnolence, and coma. Ammoniacal exhalations from the lungs and the skin are sometimes observed. As suggested by Rosenstein,[1] these symptoms are rather those of septic infection than of ammoniæmia. They do not at all resemble the symptoms induced in animals by the injection of carbonate of ammonia. Convulsions are among the most constant and prominent effects of poisoning with carbonate of ammonia, but they are absent in this so-called ammoniæmia.

Uricæmia—Lithæmia.

Uric acid exists in minute quantity in the blood in health. Its abnormal accumulation constitutes the condition to which, in the first edition of this work (1866), was applied the name uricæmia. Subsequently the name lithæmia was applied to it by Murchison. Garrod has shown that an abnormally large quantity of uric acid is present in the blood in gout.[2] It may also exist in increased amount in chronic lead-poisoning, leucocythæmia, and some other conditions. The exact form in which uric acid exists in the blood is not known, but it is probably the neutral urate of soda. The acid urate of soda is much less soluble than the neutral salt. The accumulation of uric acid in the blood is a constant attendant of gout, and is regarded by Garrod as the cause of most of the gouty symptoms. The most characteristic of these symptoms is the deposition, in various situations, of concretions of urates, accompanied often by inflammatory processes. The gouty concretions—or tophi, as they are called—consist of urate of soda, probably as an acid salt, combined often with urate of magnesia and of lime, and carbonate and phosphate of lime. These deposits are found in and about the joints, especially the small joints of the foot and hand, in the cartilage of the pinna of the ear, in the pyramidal portion of the kidney, in tendon, in the skin, nerves, vessel-walls, in the membranes of the cord, and in the spongy substance of bone. According to Ebstein, the urates are deposited only in necrotic foci. The concretions consist partly of acicular crystals and partly of granules. True gouty inflammation is always accompanied with a deposition of urate of soda in the inflamed part. The kidneys are often diseased in the late stages of gout, being small and granular, with increase of connective tissue, deposits of urates in the pyramids, and frequently waxy degeneration of the glomeruli and vessels. The urinary secretion in gout was found by Garrod to be deficient in uric acid, especially just before a paroxysm. Stockvis found at the onset of a gouty paroxysm the excretion of urea and of the earthy phosphates diminished. Others have found the excretion of phosphoric acid augmented during the attack. In the intervals between the paroxysms the excretion of uric acid may be either normal or diminished. The causes which have been assigned for an excess of uric acid in the blood are the insufficient excretion of the acid, its increased formation in the system, and diminished alkalinity of the blood or lymph, whereby the neutral urates are converted into the less soluble acid urates. Upon the assumption that uric acid is converted in the system into urea by oxidation the theory has been advanced that the uric-acid diathesis is the result of incomplete oxidation of the nitrogenous products of metamorphosis. A deficiency of oxygen, however, has not been shown to exist in the uric-acid diathesis. It will be impossible to give any satisfactory explanation of uricæmia until we learn more of the origin of uric acid in the body and of its metamorphoses. The most important elements in the etiology of the gouty diathesis are heredity, the use of wines and malt liquors, and a diet rich in albuminous and fatty substances. Over-indulgence in these articles of diet is believed to cause an increase in the proportion of acids and of acid salts, and so to favor the precipitation of acid urates. Digestive disturbances, which are frequent in gout, may likewise be attended by the generation of organic acids, the absorption of which may reduce the alkalinity of the blood. It seems probable that the deposit of urate concretions depends not only upon increased formation and diminished excretion of uric acid, but also upon lessened solvent power of the blood and other fluids of the body.

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[1] "Ueber Ammoniæmia," *Deutsche Zeitschr. f. Prac. Med.*, No. 20, 1874.

[2] Garrod, *On the Nature and Treatment of Gout and Rheumatic Gout*, London, 1859.

The designation "uric-acid diathesis" is used by some physicians in a rather indefinite way to describe various morbid states which may not at any time be accompanied by deposits of urates, and in which there is no proof of an excess of uric acid in the blood.[1] In the cases here referred to the urine is habitually highly colored, excessively acid, and it often deposits urates, uric acid, and oxalate of lime. The patients generally live luxuriously, and have a tendency to *embonpoint*. They suffer from digestive disturbances, and are sometimes hypochondriacal. There is a tendency to the formation of uric-acid gravel and calculus. In some of the patients here described the symptoms of small granular kidney, with increased arterial tension, subsequently develop.

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[1] Murchison on *Diseases of the Liver*, New York, 1877; DaCosta in *Am. Journ. Med. Sciences*, Oct., 1881; more recent contributions have been made by McBride, Lyman, Hudson, and Potter (vide paper by Landon Carter Gray in the *New York Med. Journ.*, Jan. 16, 1886).

Cholæmia—Cholesteræmia.

The presence of the essential constituents of the bile in the blood constitutes the morbid condition called *cholæmia*. The constituents which come chiefly into consideration are the biliary coloring matters, bilirubin and biliverdin, and the salts of the biliary acids—namely, glycocholate and taurocholate of soda. If the coloring matter of the bile be present in the blood in sufficient amount, it appears after a time (forty to sixty hours) in the urine and in the tissues, to which it gives a yellow color. This condition is called *icterus*, or *jaundice*. The biliary coloring matter may appear in nearly all the tissues. The central and peripheral nervous system and cartilage, however, are very slightly, if at all, stained. All of the fluids except the saliva,[1] tears, and mucus may be colored yellow. The biliary coloring matter may be easily recognized by Gmelin's test with nitroso-nitric acid. The biliary coloring matters and the biliary salts are formed in the liver, and do not pre-exist in the blood. There is reason to believe that under pathological conditions bilirubin may be formed in the blood from dissolved hæmoglobin. A number of substances, such as water, the biliary acids and salts, ether, chloroform, ammonia, and certain acids, when injected into the blood cause the destruction of some of the red blood-corpuscles, and the appearance first of hæmoglobin, and afterward of biliary coloring matter, in the urine. Certain poisons and infectious principles are believed to act in the same way in human blood. Icterus as thus induced by changes in the blood itself is called *hæmatogenous*, in distinction from the usual form, which is designated as *hepatogenous*. Hepatogenous cholæmia is dependent upon the absorption of the biliary coloring matter and of the biliary salts after their formation in the liver. The bile is probably absorbed, not directly by the blood-vessels, but by the lymphatics of the liver, and is conveyed by the thoracic duct to the blood. The causes of hepatogenous jaundice are—*first*, obstruction to the passage of bile, either in the small or large ducts; *second*, interference with the respiratory movements of the diaphragm which aid in the propulsion of the bile; *third*, lowering of the blood-pressure in the portal vessels. As yet, no certain differential symptoms between hæmatogenous and hepatogenous icterus have been established. Some pathologists are even unwilling to admit the existence of hæmatogenous jaundice. The biliary salts are found in small quantity in the urine in hepatogenous icterus, but it is not easy to detect them. Their absence cannot be considered, with our present knowledge, a sure sign of hæmatogenous icterus. These salts are known to undergo certain modifications in the blood, so that only a small quantity of those absorbed appears unchanged in the urine. In jaundice the biliary coloring matter exists, for the most part, dissolved in the fluids or diffusely infiltrated in the tissues, but it may present itself in the form of brown granules or of rhombic crystals of bilirubin, especially in the liver and in the kidneys. The influence of biliary salts in the blood has been experimentally studied upon animals. The injection of glycocholate of soda causes destruction of a certain number of red blood-corpuscles, diminution in the rapidity of the pulse, slowing of the respiration, sinking of the temperature, ecchymoses, and a weakness of the voluntary muscles. Apathy and a certain degree of somnolence are present even in the milder grades of icterus.

All clinical observers have met with exceptional cases of cholæmia in which the bile appears to have an intensely noxious influence on the nervous system, causing convulsions, coma, and death. The hæmic condition to which these effects are referable has been called "cholæmic intoxication." If uræmia be excluded, this condition denotes the presence in the blood of a constituent or of constituents of bile other than the bile-pigment or the biliary salts, for the latter are present in cases of ordinary hepatogenous jaundice in which no grave effects are manifested. Facts, physiological and pathological, point to the accumulation in the blood of cholesterin as affording a rational interpretation of the phenomena of cholæmic intoxication, or, as it has been also called, grave jaundice. In 1862, A. Flint, Jr., demonstrated that the cholesterin in bile is a result of an excretory function of the liver not previously recognized, and that it is an excrementitious principle derived chiefly from the disassimilation of nervous tissue. He also made examinations showing no increase of cholesterin in the blood in a case of ordinary hepatogenous jaundice, but a notable increase in a fatal case of jaundice with cirrhosis of the liver and ascites. To a morbid increase of cholesterin in the blood he applied the name cholesteræmia.[1] Subsequent observations have tended to confirm the pathological conclusions based on these investigations. In 1873, Müller produced, by the injection of cholesterin into the blood of dogs, phenomena resembling those of the so-called grave jaundice.[2] In 1872, Picot ascertained the existence of cholesteræmia in a fatal case of grave jaundice.[3] It is believed by some that there are excrementitious substances other than cholesterin which may be retained in the blood when the secretion of bile is arrested by extensive destruction of the hepatic parenchyma or otherwise. The non-secretion of bile is called *acholia*. It is to be borne in mind that acholia does not necessarily involve cholæmia. The toxical effects of an accumulation of cholesterin and of other excrementitious principles may occur without as well as with jaundice. It is not improbable that a deficient elimination of cholesterin may occasion more or less of those indefinite ailments which are commonly embraced under the name *biliousness*, and which are relieved by remedies supposed to increase the functional activity of the liver. It should be added that with regard to the pathological importance and relations of cholesteræmia there is not unanimity of opinions, and that experiments have been reported which contradict those of Müller.[4]

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[1] According to Fenwick, bile may appear also in the saliva (*Lancet*, Sept. 1, 1877).

Melanæmia.

In melanæmia the blood contains black granular pigment in the form of small roundish or angular granules. This condition is a result of malaria. It is most marked in the pernicious forms of intermittent and of remittent fever, and is present only in slight degree in the milder grades of intermittent fever. It may be found also in chronic malarial cachexia. The pigment may be found in any vascular part. In the spleen, liver, marrow of the bones, and lymphatic glands the pigment is not only within, but frequently outside of, the blood-vessels. In other parts the pigment is mostly intravascular. The pigmented organs may assume a dark, slate-like color. The pigment may be contained in leucocytes, in red blood-corpuscles, and in fusiform cells such as exist normally in the spleen. Often the pigment-granules appear to be free, but careful examination will then usually show that the pigment is contained in little round or oval pale hyaline or slightly granular bodies.

It is generally agreed that the pigment of melanæmia is formed out of the coloring matter of red blood-corpuscles, a certain number of which are destroyed in the process. There are different views as to the situation and manner in which the pigment is formed.

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[1] Vide "Experimental Researches upon a New Excretory Function of the Liver, etc.," *Am. Journ. of Med. Sciences*, Oct., 1862; also *Textbook of Physiology*, 1876.

[2] *Archiv für Experimentelle Pathologie und Pharmakologie*, Leipzig, 1873.

[3] *Journal de l'Anatomie et de la Physiologie*, par M. Robin, Mai et Juin, 1872.

[4] Vide Krusenstern, "Zur Frage über das Cholestearin," *Virchow's Archiv*, Bd. 65, p. 410, 1875.

The oldest view is that of Virchow and Frerichs, who believe that the pigment is formed in the spleen out of red blood-corpuscles which have either escaped from the blood-vessels or are contained in venous thrombi, and that it is thence conveyed by the portal vein to the liver, where it accumulates in considerable quantity, and whence it passes into the circulation. The chief support of this view is that the spleen is much enlarged, and is more deeply pigmented than any other organ.

Another and perhaps now more generally accepted view is that the pigment is formed in the circulating blood out of red blood-corpuscles which are destroyed by the malarial poison. According to this theory, the accumulation of pigment in the spleen is explained by the retention of pigment there in the same manner as fine particles of any kind, such as cinnabar or carmine, when injected into the blood, accumulate in this organ. The formation of malarial pigment in the blood occurs mainly during a febrile malarial paroxysm. The pigment often disappears from the circulating blood in the course of two or three days after a malarial paroxysm, but melanæmia may continue for months after the cessation of the attack.

Laveran, and Marchiafava and Celli, have found in some of the red blood-corpuscles of malarial patients, especially during the paroxysm, round, oval, or fusiform bodies which stain with methylene-blue and some other staining dyes. These bodies may be very minute or they may occupy most of the corpuscle. Some of them are endowed with amœboid movement. They may occur outside of the corpuscles. They often contain malarial pigment. Councilman has found that the apparently free pigment is in reality contained in little pale bodies which seem to be identical with those described by Laveran and by Marchiafava and Celli. The latter authors believe that these bodies, which first appear in the interior of red blood-corpuscles, are parasites, and that they elaborate the pigment out of the hæmoglobin. Satisfactory proof as to the nature of these bodies has not been furnished. They will be further considered in the article on intermittent fever.

In melanæmia the number of red blood-corpuscles is diminished, either with or without an increase of the white corpuscles.

Septicæmia and Pyæmia.

The diseases septicæmia and pyæmia will be considered briefly, as they belong to the domain of surgical rather than of medical pathology. The study of the nature of these diseases involves extremely complicated and unsettled questions. A variety of affections of a grave nature, which may follow wounds, are designated as septic. Our knowledge of the causes of these septic affections is too imperfect to enable us to classify them etiologically. An old and still prevalent theory is that septicæmia, as the name implies, is due to putrid infection, and pyæmia to purulent infection. There can, however, be little doubt that under the generic name septicæmia are included different morbid conditions. and that not all of these are referable to putrid infection. So much confusion has arisen as to the nature of the septic diseases that some prefer to group them all under the name septicæmia or under the name pyæmia. The distinction, however, which is generally used as a basis of classification is the presence or absence of metastatic abscesses. As this distinction does not rest upon an etiological basis, it is an unsatisfactory ground of classification, but perhaps it is the most useful one at present.

Septicæmia.—Most of the experiments and pathological investigations intended to elucidate the nature of septicæmia are based on the assumption that septicæmia is due to infection with some poison or poisons contained in putrefying substances. It has been found that if animals be inoculated with fluid containing the products of decomposed albuminous substances, symptoms and pathological changes are induced analogous to those observed in certain forms of septicæmia in man. It has been established by the researches of Pasteur that putrefaction is due to the action of bacteria. The question therefore arises whether putrid infection is produced by the absorption of chemical substances generated by bacteria or by the entrance and growth of the bacteria of putrefaction in the circulating blood. A number of chemical substances of an extremely poisonous nature have been isolated from putrid material. Some of these substances resemble alkaloids in their properties and composition, and have received the name of ptomaïnes. Undoubtedly, therefore, when a focus of putrefaction exists in the body, then ptomaïnes may be absorbed and poison the system. A certain number of cases of septicæmia are to be explained by the absorption of ptomaïnes. For such cases the names putrid intoxication, sapræmia, and ptomaïnæmia have been proposed. Of course in these cases of putrid intoxication the intensity of the symptoms depends upon the quantity and the virulence of the noxious substances absorbed, and if the putrefying material be seasonably removed the symptoms will disappear.

Whether septicæmia is ever produced by the entrance and development in the blood of the ordinary bacteria of putrefaction is uncertain. Only a small number of these bacteria have been isolated, and these do not seem to possess marked pathogenic properties.

Many, probably most, cases of septicæmia are caused by infection with micro-organisms the characters of which are very imperfectly known. The name septicæmia is given to a considerable number of diseases which have been produced experimentally in animals by inoculation with different kinds of bacteria. There is no evidence that these different forms of experimental septicæmia are in any instance identical with human septicæmia. In septicæmia in man different forms of bacteria have been found in the blood both during life and after death. The forms most frequently found are micrococci, and of these at least some seem to be identical with micrococci observed in pyæmia.

Septicæmia is observed most frequently after wounds, especially those complicated by injuries of bone and by contusion or laceration of the soft parts. Its first symptoms appear usually in from two to four days after the infliction of the wound and before suppuration has been fairly established. The changes which render a wound a source of infection are believed to be those of decomposition brought about by the agency of bacteria. The discharge from the wound is thin and ichorous; the surrounding parts are œdematous and sometimes gangrenous. Septicæmia may be caused also by gangrene, by ill-conditioned abscesses, and by inflammations in general. Usually, although not always, the focus of infection communicates with the open air. The hectic fever of phthisis, the suppurative fever of smallpox and certain other secondary fevers, are considered by some as due to septicæmia. There is some reason to believe that the healthy respiratory and intestinal mucous membranes can absorb septic poison generated outside of the body.

The changes found after death from septicæmia are mostly of a negative character. Decomposition sets in rapidly. The blood is dark-colored and coagulates imperfectly. Ecchymoses may be found in the serous membranes and in other parts. The spleen is often swollen and soft. Parenchymatous degeneration of the liver, heart, and kidneys is present. The solitary follicles and Peyer's patches may be swollen. Enteritis is less common in the septicæmia of man than in that induced in animals. Multiple abscesses are absent. There may be inflammation of the pleura or of other serous membranes.

Septicæmia may be ushered in by a chill, but this is not constant, and repeated chills do not occur. There is, as a rule, a continuous, usually high, fever without distinct type. The skin is dry and hot, or at times there may be profuse perspiration. There is usually a yellowish hue of the skin, but the icterus is not intense. The pulse is small and frequent. The tongue and lips are dry. Diarrhœa is inconstant. The urine is scanty and high-colored. It may contain albumen. From the onset the patient is indifferent and apathetic. There may be low delirium. Death is preceded by stupor, and frequently by decline of temperature. The duration may vary from a day or two to several weeks. It is usually about a week. It is supposed that septic poisoning in a mild or moderate degree not infrequently occurs, and ends in recovery; but in the cases in which the symptoms denote a grave affection the proportion ending fatally is very large.

The principles involved in the prevention of septicæmia embrace especially the treatment of wounds, for which works on surgery must be consulted. The different modes of antiseptic dressing and the open treatment of wounds are the means which have been most successful. After the symptoms have developed, large doses of quinine and of salicylic acid have been administered, on the ground that they possess antiseptic properties. Stimulants are required to support the strength of the patient.

Pyæmia.—As the name denotes, pyæmia was originally supposed to be caused by the entrance of pus into the blood. The disease, however, is no longer attributed to the direct absorption of pus-corpuscles. The leucocytosis frequently observed in pyæmia indicates increase of white blood-corpuscles. It may be considered as established that pyæmia is due to infection by certain micro-organisms. The leading anatomical characteristic of pyæmia is the presence of metastatic abscesses in different parts of the body. We now know that suppurative inflammations are almost always caused by the invasion of bacteria. It is probable that severe chemical irritants are also capable of causing suppuration, but such irritants rarely come into consideration in the etiology of suppurations in human beings. Of the different kinds of bacteria which have been found in acute abscesses, those most frequently met with, and most important, are various forms of *Staphylococcus pyogenes* and the *Streptococcus pyogenes*. The question arises whether the bacteria found in pyæmia are peculiar to this disease or are identical with the bacteria of ordinary pus. Contrary to what might naturally be supposed, the evidence thus far goes to show that the bacteria found in the blood and in the pus of pyæmic patients are the same as the bacteria observed in ordinary abscesses. The *Streptococcus pyogenes*, either alone or associated with *Staphylococcus pyogenes*, is the organism which has been most frequently found in pyæmia. We must therefore believe that the same organisms which cause comparatively harmless local abscesses may also cause pyæmia when they gain access to the blood in sufficient numbers.

Wounds and surgical operations, especially those involving bone, are the most common causes of pyæmia. The latter does not occur usually until after suppuration is established. The discharge from the wound becomes thin and scanty, and there is œdema of the surrounding parts. Pyæmia follows also suppurative periostitis and osteomyelitis, puerperal endometritis, and rarely other internal suppurations. The affections included under the name puerperal fever embrace both pyæmia and septicæmia. Pyæmia develops most frequently in hospital wards and in uncleanly surroundings. Sometimes no primary focus of suppuration or wound can be detected. The disease is then distinguished as primary or idiopathic pyæmia.

Especially characteristic of pyæmia is the development of thrombosis of the veins, either within or in the neighborhood of the parts affected with suppurative inflammation. These thrombi do not organize, but soften and give rise to infectious emboli. The thrombosis may be accompanied by phlebitis or inflammation of the wall of the vein, but this is not necessary. The emboli (except when the thrombosis is in some of the rootlets of the portal vein) are carried first to the lungs, where they cause abscesses which are frequently surrounded by hemorrhagic infarction or lobular pneumonia. Abscesses may be present also in the spleen, the kidneys, the heart, the liver, the muscles, and other parts of the body. These abscesses may be produced by emboli derived either from the primary thrombus or from secondary thrombi formed in the veins near the pulmonary abscesses. There is also reason to believe that secondary abscesses may be formed, independently of thrombi, by the agency of white blood-corpuscles and of micrococci which accumulate in small vessels of the affected part. Suppurative inflammation of the joints and of serous membranes is usually present in pyæmia. Mycotic endocarditis is sometimes found. There may be inflammation of the lymphatic vessels (lymphangitis) and swelling of the lymphatic glands near the primary source of infection. The spleen is usually swollen, and the same parenchymatous degenerations are present as in other acute infectious diseases. Micrococci have been repeatedly observed in the blood and in the abscesses. Colonies of micrococci in the vessels and in the uriniferous tubes are easily recognized, as a rule, in the early stages of the abscesses in the kidney. The blood usually is well coagulated, and there is less tendency to rapid decomposition than in septicæmia.

The clinical history of pyæmia is characterized by repeatedly-recurring chills, more or less pronounced, occurring irregularly and followed by profuse sweating. Fever is persistent, varying, as shown by the thermometer, in different cases, the greatest amount of heat being directly after the chill, the mercury rising at this time from 104° to 108°. The skin becomes sallow and not infrequently distinctly jaundiced. The intelligence is but little or not at all disturbed. There may be pain in the chest, with cough and frequent respiration, and physical exploration may show pleuritis with effusion, or bronchial râles with broncho-vesicular respiration at different points on either side of the chest or on both sides. Pain may be felt in the knee and other joints, and effusion within the synovial membrane may be discovered. At length persistent collections, not preceded by the usual local symptoms of phlegmonous inflammation—" cold abscesses," as they are sometimes called—may be found in different situations, sometimes in one or more of the limbs and sometimes on the trunk. The formation of purulent matter in these situations is sometimes extremely large. Suppurative inflammation of the eye, leading to sloughing and the evacuation of the humors, is an occasional event. This has been observed chiefly in cases of pyæmia following parturition. The affection in most cases runs a rapid course, the duration being from eight to ten days. With less intensity the disease may continue from two to four weeks, and occasionally it becomes chronic, lasting for several months. In the latter case the duration depends on the secondary abscesses, which by their continuance become properly sequels of the pyæmia. In the vast majority of cases the affection ends fatally. Death seems, in some cases, to be attributable to asthenia incident to the morbid condition of the blood, and sometimes to the secondary affections of the viscera. In the cases which recover the local effects consist chiefly of subcutaneous purulent collections and arthritic suppuration. The foregoing enumeration of the leading symptomatic events embraces points by means of which, generally, the discrimination of pyæmia can be made without difficulty.

The indications for treatment in pyæmia are essentially the same as in septicæmia.

The prophylaxis embraces disinfectant measures of vast importance, with reference to which surgical treatises are to be consulted.

CHAPTER VIII.

THE CAUSES OF DISEASE, OR ETIOLOGY.

Etiology a branch of General and Special Pathology—Its Importance—Internal or Intrinsic and External or Extrinsic Causes—Ordinary and Special or Specific Causes—The terms Contagion, Infection, Miasm, Virus, Venom defined—Other Poisons belonging to Toxicology—Traumatic Causes—Spontaneous Diseases—Primary and Secondary Causes—Complications and Intercurrent Diseases—Predisposing and Exciting Causes—Congenital and Inherited Predispositions to Disease—Co-operating or Accessory Causes—Diathesis—Cachexia or Dyscrasia—Endemic Diseases dependent on Emanations from the Soil—Epidemic Diseases produced by Causes existing in the Atmosphere—The Causes of Contagious Diseases exclusively derived from those affected with these Diseases—Fomites—Portability of the Special Causes of Diseases not Contagious—The Causes of Contagious and Infectious Diseases probably Living Germs or Organisms—Vegetable and Animal Parasites.

ETIOLOGY is that branch of medicine which treats of the causes of disease. As one of the divisions of the Principles of Medicine, or General Pathology, it treats of causes with reference to diseases considered, not individually, but in groups, or to disease in general. The consideration of the causes of individual diseases forms an important part of Special Pathology, or the Practice of Medicine. Etiology therefore enters into the range of topics belonging to the first and to the second part of the work. Following the general plan pursued in the foregoing chapters, I shall limit myself here to certain distinctions and definitions which are to be understood as preliminary to entering on the study of individual diseases, deviating from this plan only in making some remarks on the causation of diseases by living organisms.

Knowledge of the causes of disease is important as the basis of prophylaxis, or the prevention of disease. It is obvious that in proportion as we are able to trace diseases to their sources we may expect to extinguish causes or obviate their morbific influence. Knowledge of causes also is important as entering into the management of diseases, for it not infrequently happens that causes continue to be operative after disease has been produced, and their removal is of course a prime object in order to effect a cure. It will be apparent, when we come to inquire into the causes of individual diseases, that the amount of our present knowledge in this direction is limited. Modern researches, however, have developed much information, and not a little progress has been made in the prevention of diseases in consequence of increased knowledge of etiology; but there yet remains a wide field for further developments.

Causes are *intrinsic* or *internal*, and *extrinsic* or *external*. Examples of external or extrinsic causes are infectious miasms, viruses, different poisons, together with the many agencies of disease by means of wounds or injuries. On the other hand, examples of internal or intrinsic causes are an accumulation of urea in the blood, an excess of uric acid in gout, a morbific principle in rheumatism, supposed to be lactic acid, etc.

Among the internal causes belong those which originate in the mind; and these play an important part in the production of morbid phenomena. Over-exertion of the faculties of the mind, mental application unduly prolonged, and intense or long-continued emotional excitement lead to disorders of the nervous system, together with more or less impairment of the vital forces. Insanity and minor forms of mental derangement may not infrequently be traced to these causes. The bodily functions are weakened, partly from direct influences pertaining to the nervous system, and in part because the operation of these causes involves neglect of hygienic conditions of health which relate to diet, muscular exercise, recreation, etc. Long-continued mental depression, whether produced by the causes just named or other causes, often stands in a causative relation to disorders of digestion and anæmia. An absorbing sentiment or passion may induce disorders by leading to neglect of the means of preserving health. Finally, a concentration of the attention on the bodily functions, over-anxiety concerning health, and a craving for the interest or sympathy which illness is expected to awaken in others are important elements entering into many cases of disease, especially in women. The knowledge and the tact of the physician are often called into requisition in estimating the agency of the various causes referable to the mind.

Causes may be distinguished as *ordinary* and *special* or *specific*. Ordinary causes are those to which all persons are more or less exposed. Atmospheric vicissitudes are ordinary causes. They are generally supposed to be involved very frequently in the causation of disease and to give rise to a great variety of diseases. Special or specific causes are often extraordinary in their occurrence; that is, persons are exposed to them only at certain times or in certain situations, and each cause gives rise invariably to one form of disease. The causes of typhus, smallpox, etc. are examples of special or specific causes. The cause which produces periodical fever is not capable of producing typhus, that producing typhus will never give rise to periodical fever, and the same is true of other causes belonging in this category. The existence of a special cause may be inferred from the specific character of a disease, although the nature and source of the causative agent are not understood. It is certain, for instance, that diseases possessing phenomena and laws so distinctive and uniform as those which belong to the clinical history of epidemic cholera, typhoid fever, yellow fever, etc. must each have its own special cause.

In this connection may be defined the terms contagion and infection. The term contagion denotes a causative agent (a contagium) produced in a person affected with a particular disease, and capable of giving rise to the same disease when introduced in the body of another person. A contagium may be contained either in a palpable morbid product—for example, the contents of a variolous vesicle or pustule—or in the impalpable emanations from the body. In the former it is said to be *fixed*, and in the latter it is said to be *volatile*. A morbid product containing a fixed contagium is called a *virus*. An impalpable emanation containing a contagium is, with etymological propriety, called a *miasm*, but the term miasm is restricted by writers at the present day chiefly to those impalpable causes of disease which originate without the body, and the diseases to which they give rise are distinguished as miasmatic diseases. A familiar example of a miasm is the special cause of the periodical fevers, called malaria; and these fevers are miasmatic diseases. The term *infection* denotes any causative agent which, under certain conditions, is capable of unlimited increase or multiplication. With this definition an infectious disease may or may not be communicable by means of a contagium. If communicable, it is an infectious contagious disease; if not communicable, it is an infectious miasmatic disease. Liebermeister proposes the name *miasmatic contagious diseases* for those which, as is supposed, require a causative agent produced within the bodies of persons affected, but an agent which it is requisite should undergo certain changes after having been thrown off from the body before becoming capable of giving rise to disease in others. Examples of diseases which are thus distinguished are typhoid fever and epidemic cholera. The doctrine that the causative agent in contagious diseases is a living germ or organism (contagium vivum), and that this is also true of miasmatic infections and miasmatic contagious diseases, will be referred to in the concluding portion of this chapter.

A contagious disease may be derived from an inferior animal. Rabies, for example, is derived from the dog or cat, and glanders from the horse. Disease in man may also be produced by the introduction into the system of a product from an inferior animal which, as regards the animal furnishing it, is a normal product. Such a causative agent is a *venom*. The so-called venomous animals produce, physiologically, an agent which, received into the system of man or of another animal, gives rise to disease. Thus, certain serpents and insects have special glands furnishing secretions which cause disease and even death when introduced into the human body. These secretions, normal as regards the animals which furnish them, are venomous.

Various morbific substances which are neither viruses nor venoms, not giving rise to either contagious or miasmatic diseases, are embraced under the name poisons. Their morbid effects are proportionate to the quantity of poison received into the system. Here is a striking difference as contrasted with viruses, the morbid effects of the latter bearing little or no proportion to the quantity received. The number of these poisons is great. Their study constitutes an important branch called *toxicology*. Examples are arsenic, lead, prussic acid, etc. etc. They offer differences as regards the gravity of their morbid effects. Some are quickly fatal in a small quantity, as prussic acid, strychnine, aconite, etc.; others act more slowly and with less virulence, as mercury, lead, etc. None of these poisons increase within the body, and hence the diseases to which they give rise are not communicable.

The effects of these poisons are manifested in different parts of the body. Certain poisons exert effects on particular parts. Thus, alcohol and opium exert their effects on the brain, arsenic on the intestinal tract, strychnia on the spinal cord, mercury on the mouth, phosphorus on the liver, aconite on the heart, etc. Many, however, act simultaneously on a greater or less number of parts. As regards their effects on the blood, some are merely mixed or in solution, and act directly on parts to which they are carried in the circulation. Oxalic acid, arsenic, mercury, lead, prussic acid, alcohol, etc. have been discovered in the blood. In other instances chemical combinations take place in the blood. A striking illustration of this is afforded by one of the experiments of Bernard. Emulsin and amygdalin are not poisonous separately, but they combine and form prussic acid. Injected separately into the veins in different animals, they do no harm. If, however, they be successively injected into the veins of the same animal, they combine in the blood to form prussic acid, and the animal dies as if struck by lightning. On the other hand, the blood prevents certain combinations which take place readily out of the body. An experiment of Bernard illustrates this fact. Cyanuret of potassium and lactate of iron in combination form Prussian blue. Injected successively in the veins, they do not combine in the blood, but the combination takes place and Prussian blue is formed after both have entered the urine or within the intestinal canal.

It may be added that the terms contagion, miasm, virus, poison, are often used interchangeably, and not according to the strict definitions which are given in the foregoing paragraphs.

Certain causes are distinguished as *traumatic;* and this name is applied to distinguish the diseases produced by this class of causes. A traumatic cause gives rise to a local disease by acting in an appreciable manner on the part affected. Traumatic diseases are those thus produced. Anything which occasions an injury or wound of a part, and consequently disease of that part, is a traumatic cause. The name signifies a wound. A bronchitis caused by the inhalation of an irritating vapor is produced traumatically. A calculus in the pelvis of the kidney which gives rise to pyelitis, and a stone in the bladder which occasions cystitis, are traumatic causes. The causes thus distinguished, therefore, may be either internal or external, although in the great majority of cases they belong to the latter class.

Diseases not traumatic, and which do not proceed from any appreciable causative agency, are said to be *spontaneous* or *idiopathic.* The former term, as thus applied, is to be taken in a conventional, not a literal, sense. It is obviously incorrect to say of any disease that it originated spontaneously. Every disease must have its adequate determining cause. But many diseases are developed without our being able, in the existing state of knowledge, to refer them to their causes; and the term spontaneous, applied to those diseases, means simply that they proceed from causes which are unknown. Pleuritis, pericarditis, and other affections developed frequently in the course of Bright's disease were formerly considered as spontaneous; but now that their development is attributable to the action of excrementitious principles accumulating in the blood, the term has ceased to be applicable to them. The number of so-called spontaneous diseases will diminish in proportion as our knowledge of etiology advances.

Causes are distinguished as *primary* and *secondary.* A secondary cause is an effect of disease. Urea, for example, accumulating in the blood, gives rise to various local affections—pleuritis, pericarditis, etc. But the accumulation of urea is an effect of some disease of the kidneys which interrupts the eliminative function of these organs. Urea in excess in the blood is thus a secondary cause of disease. A primary cause, on the other hand, does not proceed from a prior disease. All external traumatic causes, for instance, are primary. The same distinction is made among diseases; that is, some are primary and others secondary—protopathic and deuteropathic. Primary diseases proceed from primary causes, and those called secondary are due to the effects of pre-existing diseases; in other words, to secondary causes. A large proportion of the diseases the existence of which is first ascertained are secondary. They involve in their causations antecedent affections. Not infrequently a number of diseases are thus linked together in a consecutive series. In the examination of patients, therefore, it is not sufficient to have discovered a disease, but, having discovered one disease, it remains to be ascertained whether other diseases do not exist sustaining to the disease first discovered a relation either of cause or effect. Clinical researches have developed important facts with respect to the causative relations existing between different diseases, and these facts are of great utility in medical practice.

Secondary and subordinate diseases are distinguished as *complications.* A complicating disease is one which occurs in the course of another disease with which it is supposed to have some pathological connection. Pneumonitis, for example, developed in the course of typhoid or typhus fever, is a complication of the latter. Pneumonitis generally has a complication—namely, circumscribed pleurisy. A disease is said to be *intercurrent* when it occurs during the progress of another disease, without necessarily having any dependence on the latter. Lobar pneumonitis, for example, occurring in a patient affected with pulmonary phthisis, is an intercurrent affection.

As just stated, causes of disease may be themselves effects of disease, so that certain diseases may be said to stand in a causative relation to other diseases. On the other hand, some diseases afford protection against others. As an illustration, emphysema of the lungs and pulmonary phthisis may be cited. The latter disease occurs in patients affected with the former so rarely as to show that it exerts a protective influence. Another illustration is afforded by disease of the heart and pulmonary phthisis. A phthisical affection occurs so rarely when cardiac disease occasions much disturbance of the circulation that the latter must be considered as affording more or less exemption from the former.

Another division is into *predisposing* and *exciting* causes. Predisposing causes induce a liability or tendency to certain forms of disease. Exciting causes, acting on persons already predisposed to certain diseases, determine their occurrence. Predisposing causes may be alone sufficient to give rise to disease, or they may only suffice to place the system in a condition favorable for the occurrence of disease. In the latter case the disease occurs when exciting are superadded to the predisposing causes. To cite an instance: A person under the influence of predisposing causes is in a condition favorable for the occurrence of acute rheumatism; in this condition he is exposed to atmospherical vicissitudes, and the latter determine an attack of this disease. The exciting cause—namely, the atmospherical vicissitudes—would not, alone, have been adequate to give rise to an attack of rheumatism; and the former—namely, the predisposing causes—alone might have been inadequate, but conjointly they occasion this disease. Exciting causes act the part of the match when everything is ready for an explosion. It is thus seen that predisposing causes determine the nature of the disease, and exciting causes the date of its occurrence.

A predisposition to some form of disease may be inherent in the constitution, or congenital. Some persons have inborn tendencies to certain diseases. This fact is strikingly illustrated by the occurrence of phthisis successively in different members of the same family, a large number of brothers and sisters being carried off by this disease. This congenital predisposition may remain completely latent until the period of life in which the disease is most liable to be developed; and we sometimes see a whole family of children, one after the other, fall victims to this disease when they severally reach a certain age. A predisposition to certain forms of disease may not only be congenital, but inherited, and a constitution involving a tendency to disease is transmitted from parent to offspring. Among the diseases perpetuated in this way are phthisis, gout, cancer, asthma, and syphilis.

The predisposition to a disease, whether congenital or acquired, may be strong or feeble. Various circumstances may act as *co-operating* or *accessory causes;* that is, acting in conjunction with the constitutional tendency. Thus, in a person predisposed to phthisis the development of this disease may be greatly promoted by unfavorable hygienic circumstances, such as sedentary habits, deficient ventilation, and inadequate alimentation. These co-operating causes, in conjunction with an existing predisposition, may serve to develop the disease when, without their aid, the predisposition might not have been sufficient. There is reason to believe that persons with a feeble predisposition to this disease often escape if they be exempt from the operation of co-operating causes. The latter, moreover, are to a greater or less extent controllable, while the predisposition, especially if it be congenital, is beyond control. Diseases thus are preventable, nothwithstanding a predisposition to them, in so far as they depend on the union of co-operating causes. Herein lies a truth of great practical importance.

Diseases which originate from special causes often appear to require for their production co-operating causes. Facts seem to show, for example, that yellow fever, periodical fevers, and epidemic cholera, which undoubtedly involve the agency of special causes, would in many cases not have occurred had not other than the special causes contributed to their production. By eradicating, as far as possible, all unfavorable hygienic influences, special causes may be rendered, to a great extent or perhaps completely, inoperative. The special causes we may not be able to remove; but co-operating causes are to a great extent within our control, and by removing the latter the diseases are rendered preventable.

A constitutional predisposition to a particular form of disease constitutes what is called a *diathesis*. A diathesis, therefore, may be either congenital or acquired; and the diseases which are considered as generally, if not always, involving a constitutional predisposition, or diathesis, are sometimes distinguished as *diathetic* diseases. A *cachexia* or *dyscrasia* involves a diathesis, and sometimes more. These terms denote not merely a constitutional predisposition to disease, but that condition of the system which exists when the disease is actually developed. A person born with a tendency to phthisis, for example, has the phthisical diathesis, and this diathesis eventuates in the tuberculous cachexia when the person becomes affected with phthisis.

The term *vulnerability* has been of late applied to a condition of the system favorable for the morbific operation of any causes, either ordinary or specific. The sense of this term differs from that of predisposition, the latter denoting a tendency to a particular form of disease, whereas vulnerability denotes a susceptibility to all morbific agencies. Vulnerability thus, in contradistinction to predisposition, does not determine the nature of the diseases produced by different causes. The term vulnerability, in fact, means neither more nor less than a general susceptibility to the causes of disease.

Our present knowledge, for the most part, of the special or specific causes of disease is based on logical inference rather than on demonstration; but certain conclusions respecting their origin and diffusion may be logically determined. One of these conclusions is that some morbific agents (miasms) emanate from the soil. This may be inferred with respect to the special causes giving rise to diseases called *endemic*. Endemic diseases are those which prevail within circumscribed territorial limits; that is, their prevalence does not extend beyond sectional boundaries. Now, this fact is sufficient for the inference that the source of the causative miasms is in the soil, because, of the elements which enter into climatic influences, those peculiar to any particular district are terrestrial. Again, in the diseases called *epidemic*—namely, those which prevail successively or simultaneously, at variable intervals, in different and often widespread territorial districts—the morbific agents, whatever may be their source, must be transported through the atmosphere or brought in some way from situations more or less distant. The causes of epidemic disease are migratory. In some instances they traverse successively almost every portion of the habitable globe. This is true of epidemic bronchitis, or influenza, and of epidemic cholera. It is altogether improbable that the special causes in these and other epidemics originate in the different sections of country over which their prevalence extends. Diseases are said to be *pandemic* when they are spread simultaneously over a whole country or population. The special causes which give rise to contagious diseases are derived from the bodies of those affected with these diseases; and with respect to certain of these diseases it is probable that the special causes are exclusively thus derived. It may fairly be doubted whether smallpox ever originates from any other source, notwithstanding the occurrence of cases in which it cannot be traced to contagion. Yet the first case of smallpox which ever existed must, of course, have been an exception to this law, and it is possible that there are still occasional exceptions.

Diseases are communicated not only by coming into contact with, or in proximity to, patients, but by means of what are called *fomites*. This term is applied to inanimate substances, such as clothing or articles of merchandise, to which contagious matter has adhered. In this way certain diseases, such as smallpox and scarlatina, may be disseminated at points far distant from the source of their special causes. The term fomites is generally restricted to the transmission of the matter of contagion. But it is certain that other special causes of disease may be transmitted in the same way. Facts, for example, show that the special cause of yellow fever is capable of transportation. And if this be true, it is correct to say that a disease may be *portable* although not communicable.

Parasites.

The study of parasites has acquired great interest and prominence since it has been proven that many and important diseases are of parasitic origin. It has long been a logical inference that the infectious diseases are caused by the invasion of micro-organisms. The hypothesis of a living contagion was most skilfully advocated upon *a priori* grounds by Sir Henry Holland, Henle, and J. K. Mitchell. Only upon this hypothesis can be satisfactorily explained such characteristic features of infectious diseases as the period of incubation, the development of the disease from the reception of an infinitesimal quantity of virus, and the almost limitless multiplication of the virus, so that the minute quantity received by a single individual, by its proliferation, may suffice to infect an entire community. It was once urged against the theory of animate contagion that the causation of infectious diseases might be explained by the action of chemical ferments; but this argument has turned to the advantage of the germ doctrine, since it has been proven that the ferments either are, or are produced by, living organisms.

The purely deductive argument as to the causation of infectious diseases by living germs has been supported during the last twenty, and especially during the last ten, years by a mass of trustworthy observations and experiments which render the doctrine of a living contagium no longer a theory, but an established fact for a number of infectious diseases. Upon this basis of facts the argument by analogy becomes of great force in support of the germ-origin of other infectious diseases in which causative organisms have not yet been discovered.

What is necessary in order to prove that a given disease is caused by a special micro-organism? This proof is absolute and complete when for a given disease three conditions are fulfilled: first, the determination of the constant presence of a special micro-organism associated with the lesions of the disease; second, the isolation of this organism by a series of pure cultures; and third, the production of the disease by the inoculation of the isolated organism. This rigid proof has been fulfilled for splenic fever, tuberculosis, glanders, erysipelas, and a number of diseases of inferior animals. Hardly less complete is the proof that the *spirochæte Obermeieri* is the cause of relapsing fever. Here the organism is of a peculiar character, is constantly present in the blood in the paroxysms of the disease, and the inoculation of blood containing the parasite causes the disease. Inasmuch as one or both of the last two conditions of the absolute proof cannot always be fulfilled—since it is not possible in all cases to find a suitable medium for the cultivation of suspected organisms, and inoculation experiments upon animals yield negative results when the animals are not susceptible to the disease—the question arises as to the value to be attached to the mere presence of micro-organisms in the lesions of a given disease. The answer is, that when a special form of micro-organism is constantly found associated with the lesions of a disease in situations where foreign organisms do not normally occur, there can be very little doubt that the organism is the cause of the disease. In opposition to this view it has been and is still urged that the organism is the result, and not the cause, of the disease—that the disease simply affords suitable conditions for the lodgment and growth of the organism. This argument was not without some plausibility in the early period of bacterial investigation, but the force of the argument has been greatly impaired by the failure to establish its correctness in any single instance; while, on the other hand, whenever it has been possible to cultivate and inoculate a special form of micro-organism constantly associated with the lesions of a disease in situations where foreign organisms do not occur, the suspected organism has been proven to be the cause of the disease. Thus, it is reasonably certain that the bacteria which are found constantly in the internal lesions of typhoid fever are the cause of this disease, although no animal may be found in whom it it possible to produce this disease experimentally.

Especial caution is required in drawing conclusions from the presence of organisms upon surfaces in communication with the exterior of the body, as upon the various mucous membranes; for upon many of these surfaces bacteria are normally present in large number. But even here, when it can be established, as in the case of the bacterium of Asiatic cholera, that an organism with distinctive characters is constantly associated with the lesions of a disease, and is never found in the body except in connection with that disease, it may be assumed that this organism is the cause of the disease.

Bacteria.—The living organisms which have thus far been proven to be the causes of infectious diseases belong for the most part to the class of schizomycetes or bacteria. The term *bacteria* is used, although not with etymological correctness, to include the whole class of organisms more scientifically designated as schizomycetes (fissure-fungi). It is not necessary to suppose that all infectious diseases are due to bacteria; it may be that other organisms, such as some of the Protozoa, are concerned in the causation of certain diseases.

The bacteria are simple, microscopic, vegetable cells, usually very minute, composed of protoplasm and perhaps an enveloping membrane. In shape the bacteria are round, rod-like, or spiral. They occur singly, in pairs, in chains, or in clumps. Clumps of bacteria imbedded in a gelatinous substance are called colonies or zoogl\u0153a, and they present an extremely characteristic appearance. Bacteria multiply, often with great rapidity, by transverse division or by the formation of spores. The spores are vastly more resistant to destructive influences than the bacteria themselves. Both bacteria and spores are destroyed by steam at a temperature of 100° C. (212° F.).

It is not possible at present to make any satisfactory classification of bacteria. For purposes of description Cohn's classification is convenient, viz.: *first*, round bacteria, called *sphæro-bacteria* or *micrococci; second*, short, rod-shaped bacteria, called *micro-bacteria; third*, longer rod-shaped bacteria, called *desmo-bacteria* or *bacilli;* and *fourth*, spiral bacteria, called spirillæ and spirochætæ. These four classes may be reduced to three by eliminating the term micro-bacteria, and calling, as is now customary, all rod-shaped bacteria, whether long or short, bacilli.

In order to determine the species of a particular kind of bacterium it is necessary to know not only its form, but the manner and conditions of its growth and its physiological properties. Nothing could be more erroneous than to suppose that because two bacteria resemble each other, or are even identical in form, they are therefore of the same species. One of these bacteria may be perfectly harmless and the other a deadly poison. The criteria which are usually employed, as far as possible, in determining the characters of any form of bacterium are—its morphology, the peculiarities of its growth in different culture-media, such as nutrient gelatin, agar-agar, blood-serum, steamed potato, bouillon, and its physiological properties, such as presence or absence of injurious effects by inoculation in animals, and presence or absence of fermentative changes by inoculation in suitable fluids.

According to their biological properties bacteria are described as *chromogenous*, or color-producing; *zymogenous*, or fermentative; and *pathogenic*, or disease-producing. The only kinds of bacteria which will be considered in this work are the pathogenic, and of these only such as are concerned in the causation of diseases in human beings. Some of these bacteria will be further considered in Part II. of this work in connection with the diseases in which they are found.

The *Micrococcus gonorrhœæ*, sometimes called gonococcus, is constantly present in gonorrhœal secretions, whether derived from the urethra, vagina, uterus, or conjunctiva. Usually two micrococci are closely joined together, forming a diplococcus. The diplococcus may resemble a single biscuit-shaped micrococcus of large size. The gonorrhœal micrococci may be found between the cells, but their most characteristic morphological property is their appearance in the interior of pus and epithelial cells. According to Bumm, the gonococcus grows slowly at a temperature of 30° to 34° C. upon gelatinized blood-serum. He succeeded in producing gonorrhœa by inoculation of the urethra with a pure culture of the organism. Further inoculation and cultivation experiments are needed, although there can be little doubt that the organism is the cause of gonorrhœa.

The *micrococci of pus* occur singly or in irregular clusters called staphylococci or in chains called streptococci. A variety of species have been distinguished, chiefly by the appearances of their cultures. The forms most frequently found in the pus of acute abscesses are the *Staphylococcus pyogenes aureus* and the *Staphylococcus pyogenes albus*. Next in frequency is the *Streptococcus pyogenes*. No less than nine different species of micrococci in pus have been distinguished. Frequently, two or more forms of micrococci are associated in the same abscess. The streptococci seem to occur especially with the more severe phlegmonous inflammations, such as those with an erysipelatous tendency. The micrococci of pus can be cultivated in gelatin and in agar-agar. By the inoculation of some of the pure cultures in animals and in human beings abscesses have been produced.

The *micrococcus of erysipelas* is a streptococcus which can be cultivated in nutrient gelatin. Inoculation of its pure cultures in man and in animals causes erysipelas. According to Passet, a streptococcus found in abscesses cannot be distinguished from that of erysipelas.

The *micrococci of osteomyelitis* seem to be identical with the staphylococci and the streptococci of ordinary abscesses. As has already been mentioned (p. 81), the same is true of the micrococci found in pyæmic abscesses.

In fetid pus, bacilli are usually found as well as micrococci. In malignant endocarditis, micrococci apparently identical with *Staphylococcus pyogenes* and *Streptococcus pyogenes* are found.

In many infectious diseases, such as scarlatina, variola, morbilli, diphtheria, typhoid fever, and acute yellow atrophy of the liver, micrococci are found occasionally, but not constantly. There is no evidence that these micrococci are the cause of the diseases. These septic micrococci, when they occur in the internal organs, are probably the result of a secondary or mixed infection which seems to be common in this class of diseases.

The *bacillus anthracis* occurs in the form of immobile rods 5 to 20 μ long and 1 μ broad, with flattened ends, and often arranged in chains. Within the body the rods multiply solely by transverse division, but outside of the body they may grow into long threads in which glistening spores are developed. The spores are very much more resistant to destructive agents than the bacilli. The bacilli and spores are incapable of development at a temperature below 15° C. (59° F.) or above 43° C. (110° F.). They require oxygen for their growth. The *bacilli anthracis* are constantly found in the body in splenic fever. They can be readily cultivated outside of the body in nutrient gelatin and in various other media. Inoculation of the pure cultures in animals susceptible to splenic fever always produces the disease. By cultivation of the bacilli of splenic fever at a temperature of 42° to 43° C. (107.5° to 109.5° F.), their virulence may be weakened or entirely destroyed, although the organisms still retain their normal appearance and capacity of development.

The *bacillus of typhoid fever* is a short, thick bacillus with rounded ends. It is found in colonies in the intestinal lesions, the swollen mesenteric glands, the spleen, and sometimes in other organs. It can be readily cultivated in nutrient gelatin, agar-agar, blood-serum, and on potatoes. This organism will be fully considered in the article on Typhoid Fever.

The *bacillus of malignant œdema* resembles the *bacillus anthracis*, but can be distinguished from it by its mobility and by slight differences of form. Its spores are widely distributed, occurring in earth, hay-dust, and putrefying fluids. It has been found in a few instances in man as the cause of a fatal inflammatory œdema. This bacillus can be cultivated in gelatin, blood-serum, agar-agar, and in potatoes under circumstances where the entrance of oxygen is excluded. Inoculation of the cultures in animals causes the characteristic disease. The growth of the organism is attended by the development of gas and fetid substances, which probably poison the system.

The *bacillus of rhinoscleroma* is a short bacillus constantly found in the peculiar hard growths of this disease, which has been observed especially in South-eastern Europe. Cultivation and inoculation of the bacillus have not yet succeeded.

The so-called *bacilli of croupous pneumonia* are elliptical bacteria which were first described by Friedländer as micrococci. A characteristic but not peculiar feature of these bacilli is the presence around them of a gelatinous capsule which can be stained by certain aniline dyes. These bacilli are frequently but not constantly present in the exudation of lobar pneumonia. They grow in gelatin-cultures in masses compared in shape to a nail. Pneumonia, usually of the lobular variety, has been produced in animals by inhalation of the organisms and by inoculation in the pleura and lungs. The inoculation experiments, however, are not very conclusive. In view of this fact, and in view of the inconstancy in the presence of these bacilli, there is much doubt as to the etiological significance of the organisms described by Friedländer. The micrococcus of *sputum septicæmia* (Sternberg) and other forms of bacteria have also been assigned as the cause of pneumonia.

Under the heading of Infectious Tumors (p. 42) mention has already been made of the *bacilli* of *tuberculosis*, of *leprosy*, of *syphilis*, and of *glanders*. The tubercle bacilli will be also considered in connection with Pulmonary Phthisis. The *bacilli of cholera*, which by some are considered to be spirilla or vibrios, will be described in connection with Asiatic cholera in Part II.

The *spirochæte Obermeieri* is a spiral bacterium 16 to 40 μ long. It is constantly present in the blood during the attacks of relapsing fever, and is absent in the intervals. In the fresh blood it is in rapid motion. The inoculation of blood containing the organism produces relapsing fever. The spirochæte has not been satisfactorily cultivated. (For further details see the article on Relapsing Fever in Part II.)

The peculiar organisms found in *actinomycosis* have already been described (p. 47). Mention has also been made of the bodies found by Laveran, and by Marchiafava and Celli, in the blood of malarial patients, which will be further considered in the article on Intermittent Fever.

There are various possibilities as to the mode of action of bacteria in the system, but we possess very little positive information upon the subject. It has been suggested that pathogenic bacteria may produce their injurious effects by withdrawing nutriment from the cells and tissues of the body, by acting as ferments which cause abnormal metabolism and produce noxious substances (ptomaïnes), and by acting as chemical or as mechanical irritants.

Fungi.—*Mould-fungi* belonging to the common species, mucor, aspergillus, and penicillium, are not infrequently found growing in necrotic tissues exposed to the air. The spores of certain varieties of mucor and of aspergillus when injected into the blood of animals are capable of growing in certain organs into mycelium, but the development never reaches the stage of fructification. No instances are known of a similar invasion of the human body with the mould-fungi. In a number of diseases of the skin, fungi have been discovered which constitute an essential pathological element in these affections. These fungi are *Achorion Schoenleinii* in favus, *Tricophyton tonsurans* in herpes tonsurans, and *Microsporon furfur* in pityriasis versicolor. The botanical position of these fungi is not known. The fungus found in aphthæ or thrush is called *Oidium albicans*, but there is doubt as to its being a true oidium.

The *yeast-fungi*, called *saccharomyces*, are often found in the fermenting contents of the stomach in various diseases of this organ. These fungi are concerned in alcoholic fermentation.

Animal Parasites.—The animal parasites belong to the three divisions *Protozoa*, *Arthropoda*, and *Vermes*.

1. The Protozoa are the most lowly organized members of the animal kingdom. The *Infusoria*, which form a class of Protozoa, have been observed frequently in the contents of the intestine, especially in cholera and in inflammatory conditions of the mucous membrane. It has not been proven that they are in any way associated with the cause of disease. The Infusoria most frequently found are *Cercomonas intestinalis*, *Trichomonas vaginalis*, and *Paramœcium coli*. *Amœbæ* have been found occasionally in the intestinal contents in dysentery. The *Psorospermiæ*, which have been found in the liver, in pleuritic exudations, and in the intestinal mucous membrane, are usually classified among the Protozoa, but their exact nature is problematical. They have been recently described as Coccidia.

2. The arthropod parasites include animals from the subdivisions *Arachnida* and *Insecta*, which infest usually the skin. To the Arachnida belong the *Acarus folliculorum*, frequently found in comedones, but without pathological importance; the *Acarus* or *Sarcoptes scabiei*, which is the cause of the disease known as scabies or itch; the *Pentastomum constrictum* and the *Pentastomum denticulatum*, which have been frequently discovered in the liver. Under Insecta are included the different species of lice—viz. *Pediculus capitis*, *Pediculus pubis*, and *Pediculus vestimenti*.

3. Animals of the division Vermes, or worms, are the most frequent of all animal parasites. The parasitic worms are embraced under the subdivisions *Nematoda* or round-worms, *Trematoda* or suctorial worms, and *Cestoda* or *Tæ-*

niada, which include the tape-worms and the old order of cystic worms. The Nematoda are *Ascaris lumbricoides*, found often in the small intestine; *Oxyuris vermicularis*, vulgarly known as the pin-worm, found in the large intestine; *Tricocephalus dispar*, in the cæcum; *Strongylus gigas*, in the pelvis of the kidney; *Anchylostoma duodenale*, in the duodenum; *Anguillula stercoralis*, in the intestine and bile-ducts in an endemic diarrhœa which prevails in Cochin-China; *Trichina spiralis*, in the muscles; *Filaria medinensis*, or Guinea-worm, in the areolar tissue, where it causes painful abscesses (in tropical climates); *Filaria sanguinis*, found in the tropics in large numbers in the blood, where it may cause chyluria and hæmaturia. Under Tremadota are included *Distoma hepaticum* in the biliary ducts; *Distoma lanceolatum*, or liver-fluke, in the same situation; and *Distoma hæmatobium* (of Bilharz) in the portal vein, and, together with its eggs, in other situations, especially the kidneys, bladder, and intestine, where it may cause severe affections. The *Distoma hæmatobium* is a frequent parasite in Egypt and in Abyssinia. The Cestoda include the fully-developed tape-worms and their larvæ or scolices. The immature cestoid worms or scolices which are parasitic in man are *Cysticercus cellulosæ*, the scolex of *Tænia solium;* and the *echinococcus* or hydatid, the scolex of *Tænia echinococcus*, a small tape-worm which infests the dog. *Cysticercus cellulosæ* may be present in any part of the body. It is of most importance usually in the brain. The echinococcus is most frequent in the liver, but it may exist in other parts. The fully-developed tape-worms in man are *Tænia solium*, *Tænia medio-canellata*, and *Bothriocephalus latus*, or Swiss tape-worm. Rare forms are *Tænia nana*, *Tænia flavo-punctata*, and *Tænia cucumerina*. Most of these worms receive consideration in Part II. of this work.

CHAPTER IX.

SYMPTOMATOLOGY.—DIAGNOSIS.—PROGNOSIS.

Pathognomonic Symptoms—Diagnostic Symptoms—Subjective and Objective Symptoms—Signs—Clinical History of Diseases and the Different Modes by which it is Obtained—Stages of Disease—Precursory or Prodromic Events—Sequels—Diagnosis—Reasoning by Way of Exclusion—Differential Diagnosis.—Prognosis—Prognostics.

THE study of the symptoms of disease forms a division of the Principles of Medicine, or General Pathology, called *Symptomatology* or *Semeiology*. The consideration of symptoms will occupy a large proportion of the space to be devoted to each individual disease in treating of the Practice of Medicine, or Special Pathology, in the second part of this work. It will suffice to present in this chapter certain distinctions and definitions which should be understood before entering on the study of individual diseases.

Under the name symptoms are embraced all appreciable morbid phenomena occurring in connection with disease. The name signifies "to fall together," and denotes concurrence of events. To repeat the often-quoted comparison by Galen, symptoms accompany diseases as the shadow follows the substance. By means of symptoms the existence of disease is made apparent and the character and seat of different affections are ascertained.

Certain symptoms are distinguished as *pathognomonic*. A pathognomonic symptom is one which denotes invariably the existence of a particular disease. It derives this significance from the fact that it occurs only in connection with one disease. Whenever such a symptom, therefore, is present, it represents that disease, and in itself suffices for its recognition. The number of pathognomonic symptoms is quite small. As an illustration, a semi-transparent, viscid, rusty-colored matter of expectoration belongs exclusively to pneumonitis. It is therefore a pathognomonic symptom, always denoting the existence of that disease. In like manner the crepitant râle obtained by auscultation is so distinctive of the same disease as to be nearly pathognomonic. It would be difficult to cite many additional examples.

A symptom is said to be *diagnostic* when it occurs more frequently in connection with a particular disease than with other diseases. Just in proportion as it is restricted to a few diseases is its diagnostic import marked; that is, it denotes the presence of some one of these diseases, and its value in diagnosis is the greater the smaller their number. Different symptoms differ as regards their respective diagnostic value. Some are highly diagnostic; in other words, they point strongly to the existence of particular diseases. Others are moderately, and again others slightly, diagnostic. The measure of diagnostic significance depends on the frequency of concurrence with a particular disease and the infrequency of concurrence with other diseases.

Symptoms are either *subjective* or *objective*. Subjective symptoms are those which relate to the sensations or feelings of the patient, and which, consequently, the physician can appreciate only through the patient's description of them. Objective symptoms, on the other hand, are those which the physician observes for himself, and for a correct appreciation of which he is in no wise dependent on the patient. As regards subjective symptoms, the physician is exposed to error in consequence of inability on the part of the patient to describe them clearly. In young children this class of symptoms is not available. This is true also of idiots, the insane, and in the delirium incident to different diseases. As regards these symptoms, too, the physician is liable to be deceived by intentional misrepresentations on the part of patients; and he is liable to be deceived unintentionally by a tendency on the one hand to exaggeration, and on the other hand to depreciation of morbid sensations or feelings. Owing to these sources of error much tact is often requisite in placing a proper estimate on subjective symptoms. Objective symptoms are not open to the same liabilities to error. The physician judges of these by the exercise of his own senses, and if he fail to discover or place a proper estimate upon them the fault consists in his own want of attention, judgment, or skill. Objective symptoms are thus much more reliable than subjective in the investigation of cases of disease.

The exaggeration of pain or suffering of any kind and of morbid sensations is common—sometimes because patients are led to form an exaggerated idea of their subjective symptoms; sometimes with a view to awaken interest or sympathy; sometimes for malingering purposes; and sometimes to secure greater attention on the part of the physician and more efficient treatment. Factitious and exaggerated ailments enter pretty largely into the cases which the practitioner is called upon to treat.

A symptom, as already defined, is any manifest morbid phenomenon or event. Occurring concurrently with a disease, it may or may not have a pathological connection with that disease. Its pathological connection with the disease may be such as to render it a pathognomonic symptom or more or less diagnostic; and, on the other hand, it may be present as a mere coincidence. If the latter be true, it has no pathological connection with the disease, and is devoid of diagnostic significance. This difference among symptoms has been expressed by calling them *signs* when they are either pathognomonic or more or less diagnostic. According to this distinction, a symptom becomes a sign whenever it denotes, in a greater or less degree, the existence of a particular disease. The term signs, however, has come conventionally to be used in a different sense. It is usual to apply this term to the phenomena obtained by certain methods of investigation distinguished as physical—namely, percussion, auscultation, etc. These phenomena are called physical signs, but for convenience the word physical is often omitted, and is considered as understood when the term sign is alone used. In this sense of the term signs it is used in contradistinction to symptoms, the latter embracing all other phenomena manifested in connection with disease. The phenomena thus included under the name signs are of course objective, and will be found to be the most reliable of those by means of which the existence, seat, and character of certain diseases are ascertained.

The symptoms and signs which occur during the course of a disease are the events which make up its *clinical history*. This phrase is applied to a description of the manifest phenomena taking place from the beginning to the end of a disease, together with the order of their succession and the laws which govern them. Or, instead of clinical history, it is proper to say the *natural history* of a disease when the disease is allowed to pursue its course unaffected by disturbing influences of any kind, and when no attempt is made to arrest, abridge, or modify its course by therapeutical measures.

How is the clinical or natural history of a disease to be obtained? There are three methods which may be employed. One method consists in selecting, as types of the disease, a few well-marked cases, and basing the description of the disease on the events observed in these cases. This method is defective, because some symptoms belonging to the disease might be wanting, and some symptoms not belonging to the disease might be present in these few representative or typical cases. Moreover, this method is inadequate to show the relative importance of particular symptoms. Another method is to make out a history from recollection after having observed a greater or less number of cases. A history thus obtained must be imperfect from the obvious inability to retain in the memory all the events which occurred while the cases were under observation, together with the order and relative frequency of their occurrence. The third method is the only one by means of which an accurate clinical history of a disease is to be obtained. It consists, *first*, in the accumulation of a certain number of cases fully and carefully recorded; and *second*, in an analysis of the recorded cases with reference to the symptomatic phenomena which occurred, the relative frequency of their occurrence, the order of their succession, the number of deaths and recoveries, the duration of the disease, etc. By this method are ascertained events which are constantly present, and therefore have an essential relation to the disease; events which are incidental to the disease, being present more or less frequently; and events the presence of which is purely accidental. There are many diseases which have not, as yet, been sufficiently studied after this method, and here, therefore, is ample scope for further clinical labor. Among the diseases which have been studied after this method may be mentioned typhoid and typhus fever and acute lobar pneumonitis. The knowledge of the clinical history of these and some other diseases obtained by means of this method within late years exemplifies its advantages.

The clinical history of a disease is divided into several distinct periods or stadia, which are commonly called *stages*. The first stage may be distinguished as the invasion or access of the disease. This period dates from the first morbid manifestations, and extends to the time when the disease may be considered as fully developed or established. The duration of this stage varies in different diseases and in different cases of the same disease. Generally, the duration is governed by certain laws proper to different diseases. Some diseases are characterized by the abruptness of their development, the stage of invasion being very short or altogether wanting. The *career* or *course* of a disease extends from the date of its development—that is, from the end of the stage of invasion—to the time when the disease may be considered as ended. This period is frequently subdivided into two or more stages. The stage of convalescence embraces the period between the end of the disease and the complete restoration of health.

The clinical history of a disease properly embraces phenomena which may precede the stage of invasion or access. They are distinguished as *precursory* or *prodromic* events; and in some diseases is to be included a period which is called the period of *incubation*. The latter term, signifying hatching, relates to the time which elapses after the reception of certain special causes of disease before any morbid manifestations take place. For example, after inoculation with the virus of smallpox a certain number of days elapse before any morbid phenomena make their appearance. But in the mean time occult operations are going on in the system, and when these are sufficiently advanced the period of invasion or access begins. The significance of the term incubation is thus apparent. It is rendered especially significant by the germ doctrine of the causation of many diseases. The existence of a period of incubation is one of the points in evidence of the truth of this doctrine. Again, the clinical history of a disease should include phenomena which may occur after recovery from a disease, provided they be dependent upon it. Certain diseases are liable to be followed by certain consequences, which are called *sequelæ* or *sequels.* Thus, after recovery from scarlatina, albuminuria and general dropsy occur in a certain number of cases. These are results of this disease, and hence belong to its history as sequels. In like manner, phthisis is likely to be a sequel of measles, motor paralysis follows diphtheria, etc.

As a general statement, diseases, in respect of the phenomena and laws belonging to their natural history, present in different times and places great uniformity; they are essentially the same in different parts of the world, and they undergo little or no material changes from age to age. This statement is believed to be correct, although it is in opposition to views somewhat prevalent. The different phases and modifications which the same diseases may present, not only at different times and places, but in different cases at the same time and place, are, for the most part, to be explained by the coexistence of affections which may have been antecedent or which are developed as complications.

Diagnosis.

One of the most important of the aspects under which individual diseases are to be considered is in their *Diagnosis*. The term diagnosis means the discrimination of diseases; that is, ascertaining their character and seat. The practical importance of a correct discrimination of diseases with reference to their management is sufficiently obvious. How is it possible to bring to bear upon a case of disease established principles of treatment until the character and seat of the disease are ascertained? Diagnosis and treatment are, in fact, the two practical ends of the study of medicine. Diagnosis is also important with reference to obtaining knowledge of the clinical history of diseases. In collecting cases for analysis, if different diseases be confounded the results of the analysis will be vitiated. In like manner, correctness of diagnosis is the basis of therapeutical experience. Conclusions drawn from the effects of measures of treatment in a series of cases will, of course, prove fallacious if the series include cases of different diseases which have been confounded.

Here is a source of not a little of what has been called false experience in practical medicine. Diseases which were formerly confounded are now known to be distinct, and can be readily discriminated from each other. For example, the eruptive fevers, measles, scarlet fever, and smallpox, were once considered as different varieties of one disease. Physicians did not undertake until recent times to discriminate between pleuritis, pneumonitis, and bronchitis; now the diagnostic characters of each are well ascertained, and are sufficient for its recognition. Still more recently, typhus and typhoid fever have been shown to be essentially distinct diseases.

Differences in skill and tact among practitioners of medicine relate especially to diagnosis. In medical consultations it is here chiefly that points for discussion and disagreement arise. It is here that the want of aid and counsel is oftenest felt by the physician. The diagnosis involves more embarrassment than the management of diseases. This fact is not appreciated by the people at large, many of whom undertake to decide respecting the nature and seat of the disease whenever their friends are ill.

The diagnosis of a disease may be based on the presence of a pathognomonic event. Thus, the characteristic expectoration or the crepitant râle denotes the existence of pneumonitis. But, as already stated, there are but few symptomatic phenomena which are truly pathognomonic. Moreover, they are not present in all cases of the diseases which they denote; hence their absence is by no means proof against the existence of the diseases. The characteristic expectoration and the crepitant râle, for example, are wanting in not a very small proportion of cases of pneumonitis. It is evident, therefore, that the diagnosis of a disease cannot often be based on pathognomonic events. And next in importance as regards the basis of diagnosis is the presence of diagnostic symptoms. As a rule, in proportion to the degree of diagnostic significance belonging to the symptoms present and to their number, is the diagnosis of a disease easy and positive. In these respects different diseases and different cases of the same disease differ widely; in some instances the diagnostic symptoms present are few in number and weak, while in other instances they are many and strong. It is with special reference to the relative diagnostic value of the different events which make up the clinical history of a disease that this history is important to the physician.

The laws of disease are often of importance in relation to diagnosis. For example, typhoid fever rarely affects persons over fifty years of age. When, therefore, persons beyond this period of life are said to have typhoid fever, the chances are that an error has been made in diagnosis. Certain diseases, as a rule, occur but once in the same person. The probabilities, therefore, in a case of doubtful diagnosis are against the existence of a disease of this class if it be ascertained that the patient has already had the disease. Certain physical signs situated within a circumscribed space at the summit of the chest denote phthisis, while the same signs situated elsewhere might point to some other affection, owing to the law that a phthisical affection, in the vast majority of cases, first occurs at or near the apex of the lung. Similar illustrations might be multiplied: the laws regulating the development of a disease, the succession of events, its duration, its sequels, etc., often furnish valuable aid in its diagnosis. Here, too, the practical importance of knowledge of the clinical history of diseases is apparent.

A highly effective method of reaching the diagnosis of a disease is called "reasoning by way of exclusion." In a case of doubt as regards diagnosis the problem is generally to decide between a certain number of diseases. The existing disease is one of two, three, or more diseases which may be suspected to exist. Now, if it be difficult to decide which one of these is the existing disease from the positive proof relating to diagnostic events and laws, it may be practicable to decide that there is insufficient evidence of the existence of one or more, and therefore the latter are excluded. By this process of elimination the number of diseases is diminished, and may be reduced even to one disease, the diagnosis thus being made on the principle of exclusion.

In discriminating a disease from one of several diseases with which it has more or less symptomatic phenomena in common, the amount of positive evidence in behalf of its existence is to be considered, and also the evidence against the existence of the diseases from which it is to be discriminated. This comparison of one disease with other diseases, with reference to the positive and negative evidence of its existence, constitutes what is called *differential diagnosis;* and to discriminate a disease in the manner just described is to differentiate it. Thus, typhus and typhoid fever have many symptoms in common, but differ from each other in essential points. The differential diagnosis of these diseases has reference to the circumstances which denote the presence of the one and the absence of the other.

Diagnostic skill is required not alone in differentiating diseases, but in the discrimination of cases in which diseases are either simulated or their existence fancied. The knowledge and tact of the physician are here not infrequently called into requisition. The judgment must rest mainly on deviations from the established laws of different diseases as regards symptomatic phenomena, etc., and the absence of objective phenomena which in certain diseases are invariably present. It follows that the practitioner, other things being equal, will be successful as a diagnostician in such cases in proportion to the accuracy and extent of his knowledge of the phenomena and laws of different diseases. Diseases are simulated for the attainment of certain ends. Thus, soldiers feign diseases with a view to escape active duty or to obtain a discharge from the service; prisoners feign diseases to secure pardon or the comforts of a hospital; and cases are not rare of persons simulating various affections to excite sympathy and become objects of interest. Feigning diseases for these or other ends is called malingering, and persons attempting the deception are malingerers. One mode of malingering is to pretend that diseases which actually exist are more severe than they really are. Among the diseases which malingerers are apt to feign are epilepsy, paralysis, phthisis, aphonia, amaurosis, and insanity.

Practical skill in diagnosis requires mental qualifications which all do not possess in equal measure. Aside from these, the amount of skill which may be acquired will be proportionate to the knowledge of, and familiar acquaintance with, different diseases as regards their clinical histories. It would be absurd to suppose that diseases are to be recognized by an intuitive perception, and that some persons have a peculiar innate gift in this direction. A diagnosis not based on adequate pathological information is always open to more or less of the chances of error which pertain to guessing. The mental qualifications of the skilful diagnostician do not differ essentially from those required for other than medical investigations—namely, accuracy of observation, the ability to reason correctly, sound judgment, and good sense.

Prognosis.

The division of medicine called *Prognosis* treats of the means by which the course and termination of diseases may be foreseen. It is often important to form a correct judgment concerning the way in which diseases may be expected to end, with reference to communications with patients and their friends. They may desire—and if so it is proper they should receive—information on this point. Nor should the physician be unmindful of the fact that persons in general are likely to estimate his knowledge and ability by the correctness of his judgment in this regard. Recollecting this fact, the protection of his own interests, in addition to other considerations, will dictate a certain amount of reserve in his predictions. But a correct judgment as regards prognosis may be important with reference to treatment. Therapeutical measures are sometimes to be withheld in view of the complete hopelessness of the condition of the patient, and under these circumstances persistence in the use of remedies is not only superfluous, but likely to do harm. On the other hand, therapeutical measures may be employed with reference to the danger of death without regard to the nature or seat of the disease. A disease equal in all respects in different cases may destroy the lives of some, while others recover, owing to the diversity which exists in different persons as regards the ability to sustain and overcome disease; and in the management of diseases it is often the chief aim of the physician, in the language of Cullen, to obviate the tendency to death.

Prognostics are those circumstances on which a prognosis is based. They relate, of course, mainly to the symptoms of disease. Formerly, before the nature, seat, anatomical changes, etc. of different forms of disease were as well understood as now, all the circumstances connected with diseases were considered with direct and special reference to their significance as prognostics. The consideration of these is appropriate in treating of the Principles of Medicine, or General Pathology, but it will suffice to consider them incidentally in connection with individual diseases. A few points which relate to the physiognomy or obvious appearances, and which render the prognosis unfavorable, may here be enumerated by way of illustration.

Notable emaciation and pallor, existing in connection with any chronic affection, are unfavorable prognostics. Lividity of the prolabia and face, if persisting, generally denotes great danger. Great frequency and feebleness of the pulse, exclusive of diseases of the heart, and especially in acute diseases, point to a fatal termination. Great prostration is usually an alarming symptom. Spasmodic inspiration, if not dependent on pulmonary disease, precedes the development of coma, unless it be referable to hysteria. Coma, if it last more than one or two days, will be likely to end in death. Paralysis of the sphincters, leading to involuntary evacuations, and loss of the muscular power of deglutition, often denote impending dissolution. Impaired capability of feeling the presence of liquid in the air-passages, and inability to make the effort requisite for its removal by expectoration, are forerunners of the moribund state. A notable increase of the animal heat, as ascertained by the thermometer, if persisting, is to be reckoned among the prognostics denoting imminent danger. Finally, the characters pertaining to the physiognomy which constitutes the *Hippocratic countenance*—so called because they were described by the ancient Father of Medicine—denote the moribund state. These characters are marked pallor with more or less lividity, pinching of the nostrils, sinking of the eyes, hollowness of the temples, coldness and transparency of the ears, dropping of the lower jaw, etc. The change in physiognomy in this state is so great that persons are sometimes hardly recognizable. The features, to use a French expression, are decomposed, and a glance at the face often suffices to show that death is nigh at hand.

CHAPTER X.

PROPHYLAXIS.—GENERAL THERAPEUTICS.

Experience the Basis of Therapeutical Knowledge—Two Sources of Experience: Analysis of Recorded Cases with Reference to the Duration or Termination of Diseases, and Observation of the Immediate Effects of Therapeutical Measures—Rational Inference as a Means of Arriving at Principles of Therapeutics—Importance of Knowing the Intrinsic Tendencies of Diseases toward Recovery, or otherwise—Active Measures to be Employed only when clearly indicated—The Expectant Plan of Treatment—Conservative Medicine—Importance of Hygienic Measures of Treatment—Modes of Dying resolvable into Apnœa and Asthenia—Classification of Therapeutical Measures, according to the Objects to be accomplished, into Prophylactic, Abortive, Curative, Palliative, Hygienic, and Sustaining Measures.

Prophylaxis.

THE prevention of disease is an object connected with pathological studies. It constitutes a division of medicine called *Prophylaxis*. The name "preventive medicine" has of late been applied to it. It is one of the several aspects under which individual diseases are to be considered. As regards the beneficent fruits of knowledge, it holds a place not inferior to that of therapeutics. Many diseases are preventable by removing or obviating their causes; and it is with reference especially to this application that the study of causes, or etiology, is of great practical importance. The knowledge already acquired of the causation of certain diseases has proved of much value. A striking illustration of this is afforded by our present acquaintance with the poisonous effects of lead. It is only within late years that certain diseases have been traced to the action of this poison. The diseases thus produced were doubtless of frequent occurrence before their causation was understood. The physician is now able sometimes to determine that diseases are thus produced, although he may not be able to ascertain in what manner lead has been introduced into the system; characters pertaining to the disease show that they have originated from this source. This statement is true of that form of paralysis called wrist-drop and of the form of neuralgia known as saturnine colic.

The recognition of the germ doctrine as affording the most rational explanation of certain of the special or specific causes of disease has led to the employment, with great effect for the prevention of infectious diseases, of measures the efficacy of which apparently depends on their agency in destroying the low forms of vegetable life. In this agency consists the efficiency of certain of the so-called disinfectants. The prevention of infectious diseases will be more and more efficient in proportion to the increase of knowledge of disease-producing organisms, together with the conditions under which they are multiplied. An essential etiological factor in the causation of certain infectious diseases, in addition to the presence of specific organisms, is the existence of peculiar local conditions. These conditions constitute the predisposition to certain diseases. They may be distinguished as diathetic conditions. They are as essential to the development and multiplication of pathogenetic organisms as the presence of the latter. Their removal is not less effectual for the prevention of infectious diseases than measures which are directly destructive to the organisms. Preventive medicine may therefore accomplish as much by finding means for the removal of these conditions as by the discovery of disinfectants; and it may be that the time will come when the means will have been discovered of so sterilizing the human soil that it will be incapable of giving productiveness to the germs of disease.[1] Unknown special causes may be indirectly controlled, to a certain extent, by removing accessory or co-operating causes. In this way sanitary measures are efficient in protecting against epidemic and endemic diseases. Much has been accomplished in behalf of prophylaxis, but much remains to be accomplished. Sanitary reforms relating to ventilation, sewerage, cleanliness, etc., there is reason to believe, do very much toward the prevention of disease, and hence these are among the most important of the modes by which philanthropic efforts may be rendered useful to mankind. The prospect of diminishing the mortality from certain diseases relates more to prophylaxis than to therapeutics.

General Therapeutics.

The division of medicine devoted to the treatment of disease is called *Therapeutics*. The consideration of therapeutical measures, as regards the evidence of their usefulness, the rationale of their operation, the indications for their employment, etc., constitutes *general therapeutics*. The general principles of therapeutics are usually considered in connection with the Principles of Medicine, or General Pathology. The consideration of the treatment of individual diseases enters into Special Pathology, or the Practice of Medicine, and constitutes *special therapeutics*. It will suffice here to offer a few remarks relating to general therapeutics, reserving the different therapeutical measures for consideration in connection with the treatment of individual diseases in the second part of this work.

It is needless to offer any remarks on the importance of therapeutics. The treatment of disease, next to its prevention, is the great end of all studies relating to pathology, general and special. It is, however, the most difficult of all the branches of medicine for the teacher. It is so, not alone from uncertainty as regards the correctness of therapeutical principles, but because principles undoubtedly correct in their general application are to be greatly modified in adapting them to the varied circumstances pertaining to individual cases of disease. The same disease, under different circumstances, may claim not only widely different modifications of treatment, but therapeutical measures directly opposite in character. There are abundant occasions for citing illustrations of this fact in treating of individual diseases. It is impossible to formulate rules for the application of therapeutical measures. If this were possible, the practice of medicine would be a mechanical, not a rational, art. All that the teacher and author can hope to do is to present general principles, together with the more important considerations involved in their application; and their successful application requires not only knowledge, but reasoning powers, judgment, good sense, and practical tact.

How are the facts and principles of therapeutics obtained? Mainly by experience. The question then arises, How are the results of experience, as regards the utility of therapeutical measures, obtained? There are two sources of experimental evidence in behalf of the utility of a particular method of treatment in any disease. One source of evidence relates to the termination and duration of the disease in a series of cases in which the method of treatment has been employed. The superiority of a method is shown by a larger number of recoveries and an average duration shorter in such a series than in other series of cases treated otherwise. This statistical investigation is open to certain fallacies. No two series of cases of a disease are in all particulars exactly alike. Cases differ in the degree of severity and extent of disease, in the constitutional condition of patients, in the existence or otherwise of complications, and in a great variety of circumstances pertaining to season, climate, age, habits, etc. The results of a comparison of different series of cases in respect of termination and duration are to be accepted as the basis of experience only on the ground that the differences in the different series mutually compensate for each other. Hence the number of cases embraced in a series should be large, and the results of the comparison should be confirmed or corrected by being repeated with successive series of cases collected at different times and places and by different observers. Moreover, pains should be taken to group together cases resembling each other as closely as possible. Observing carefully all proper precautions, the results obtained by this kind of investigation are of great value if deduced from data faithfully recorded by honest observers and competent diagnosticians. The memory is wholly inadequate for such an investigation. Moreover, recorded facts afford the only security against prejudice and bias. Unrecorded experience is by no means valueless, but it is of little relative value as compared with the results of the analysis of recorded data.

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[1] Vide article by Dr. Ezra M. Hunt in the *Philadelphia Medical News*, Sept. 15, 1883.

Another source of experimental evidence relates to the immediate effects of therapeutical measures irrespective of the termination or duration of diseases. The usefulness of remedial measures may be apparent from their immediate effects, although there may be no proof that the disease has been abridged or although it may end fatally. A method of treatment may be useful in a certain number of the cases in a series, although in the majority of cases it may be injurious. For example, let it be assumed that in the larger number of cases of lobar pneumonitis bloodletting does harm—it may nevertheless do good in a small number of cases; and, conversely, if it be assumed that this remedy is useful in the majority of cases, it may be pernicious in some cases. So with regard to other measures in other diseases.

It is thus evident that after the utility of particular therapeutical measures in certain diseases, as the rule, has been ascertained by means of a sufficient number of facts derived from the first source of experimental evidence, it is vastly important for the physician to exercise discrimination in the application of these measures to individual cases. And it is to be borne in mind that in the practice of medicine the physician deals with cases of disease separately, not in aggregates. The general principles of therapeutics have a relation to individual cases of disease not unlike that of the rates of life insurance to the liability to death of particular persons. It is easy to calculate the probabilities of the duration of life in a large number of persons of a given age, and to determine exactly what it is worth to insure the lives of a large number, but no one thinks of applying to an insurance office to know how long he is to live.

Rational inference is a means of arriving at therapeutical principles. If, for example, it be known that a person has swallowed a corrosive poison, reason and common sense at once dictate the propriety of endeavoring either to expel it or to neutralize it by an appropriate antidote. But in such a case experience must be resorted to to know whether either of these objects be practicable, which of the two is to be preferred, and the best means of effecting each. Analogical and *a-priori* reasoning may lead to the employment of measures in the treatment of diseases, but the results of experience are necessary to establish their therapeutical value. Talent and genius may be shown in originating methods of cure, but the practical test of their efficacy is afforded by experience. It is true that false experience has abounded in medicine, but if experience be open to fallacies, this is true to a far greater extent of theoretical or speculative reasoning. It is worthy of note that our knowledge of the most important remedies has been acquired wholly by experience, without any explanation of their *modus operandi*. Examples are the salts of quinia as a remedy for periodical fever, mercury and iodine in certain forms of syphilis, etc.

The intrinsic tendency of a disease to recovery or otherwise is to be considered with reference to the value of therapeutical measures. Knowledge of the natural history of diseases is important as the true point of departure for therapeutics. Much has been acquired within late years in this branch of knowledge, but much remains to be acquired. Diseases which were formerly supposed to tend to a fatal result if not treated by means of active remedies are now known to end generally in recovery if left alone. Examples are acute pleuritis and uncomplicated lobar pneumonitis. The importance of increasing our knowledge of the natural history of diseases by collecting cases which have pursued their course without active treatment is to be kept in mind by those desirous of contributing to the advancement of therapeutics; and opportunities of accumulating such cases should not be neglected, always provided that the welfare of the patient be not compromised by withholding measures which there are good grounds for believing will prove useful.

It may be laid down as a golden rule in therapeutics that active measures of treatment are only to be employed in cases in which they seem to the physician to be clearly indicated. The severity of disease and the danger of the patient, be they never so great, do not alone constitute grounds for the employment of active measures. If they be not useful they are likely to do harm. Therapeutical measures, in proportion to their power, are powerful for either good or harm, and must therefore be either indicated or contraindicated. In the language of another, the physician "should be content with doing nothing when ignorant how to do good."

The term *expectant*, as applied to the treatment of disease, is often used, and its true sense is not always observed in its application. The treatment of a disease by expectation consists in watching carefully its progress, and in meeting with appropriate measures unfavorable events as they arise. This plan is not inconsistent with the employment of active measures of treatment; but these are resorted to not simply because the disease exists, but with reference to events connected with the disease. In this sense of the term the treatment of a disease is expectant whenever the physician does not attempt to abridge or arrest it, but strives to aid in conducting it to a favorable termination.

The progress of medicine within late years as regards therapeutics is characterized by the development of a principle which may be distinguished as *conservatism*. This term may be applied to medical as to surgical practice, and denotes an object which has not heretofore been sufficiently considered—namely, the avoidance of useless and injurious therapeutical measures. Conservative medicine does not interdict the use of the most potential of remedial agencies, but it enforces discrimination and circumspection in their use, recognizing fully their potency for evil as well as for good. The conservative physician is by no means a mere spectator of the progress of disease, but in wielding the resources of therapeutics he is careful to preserve the powers of life, following the injunction of Chomel, not to treat disease, but patients affected with disease.[1]

The management of cases of disease involves not only the exercise of judgment in the employment or otherwise of therapeutic measures, but attention to hygienic regulations. And in a better appreciation of these the practice of medicine at the present time offers a striking contrast with the past. It may perhaps safely be said that the greater success attending the management of diseases now than heretofore is due as much to improvements as regards diet, ventilation, etc. as to the more judicious use of medicinal agencies.

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[1] Vide A. Flint, *Essays on Conservative Medicine and Kindred Topics.*

In the treatment of diseases involving danger to life the mode of dying is to be considered. It is always a useful inquiry at the bedside, By what mode of dying will the case under treatment end if the patient do not recover? The answer to this question will often furnish the indications for treatment by means of which the danger may be forestalled; and here, it is plain, is the source of indications under circumstances in which it is the aim of the physician to "obviate the tendency to death." It suffices for all practical purposes to reduce the different modes of dying to two—namely, by *apnœa* and by *asthenia*.

Death by apnœa is produced by interruption of the function of respiration. The type of this mode of dying is furnished in cases of strangulation. Death by this mode occurs when produced by affections involving obstruction to the passage of air to the air-cells of the lungs, such as acute laryngitis, œdema of the glottis, foreign bodies in the air-passages, and capillary bronchitis. It occurs sometimes when a considerable portion of the pulmonary organs is rendered incompetent to receive air, as in pleuritis with large effusion, pneumonitis affecting both lungs, and extensive pulmonary phthisis. It also occurs when the respiratory movements are arrested by an affection of the medulla oblongata involving the suspension of the influence by which these movements are sustained. When death occurs from coma the mode of dying is by apnœa. In this mode of dying the embarrassment of respiration is out of proportion to the disturbance of the circulation, and when death takes place purely by this mode the action of the heart continues after the cessation of respiration.

Death by asthenia, on the other hand, is due to the failure of the circulation. The action of the heart may be suddenly arrested by paralysis from over-distension of the ventricles, by the pressure of blood from an aneurism opening into the pericardium, by pericardial effusion, and by the coagulation of blood within the cavities. But in all diseases which prove fatal by adynamia or exhaustion the mode of dying is by asthenia, and when death takes place purely by this mode respiration continues for a greater or less period after the pulse has ceased to be appreciable at the wrist. Apnœa, however, is frequently combined with asthenia in producing death; the diminished muscular power renders the patient unable to prevent the accumulation of liquid in the air-passages, and the respiratory function also may be interfered with by congestion and œdema of the lungs due to exhaustion. So, also, asthenia is frequently combined with apnœa in fatal affections of the organs of respiration. In fact, in a large proportion of cases the two modes of dying are combined, either the one or the other mode predominating.

With a view to reference in the second part of this work, a classification of methods of treatment based on the general objects which the physician aims to accomplish in the management of individual diseases will be convenient. These objects are embraced in the following classes:

1. *Prophylactic or Preventive Measures.*—Vaccination is a preventive measure with respect to smallpox. Quinia, given to protect against intermittent fever, is a measure belonging to this class. Measures to eliminate excrementitious principles from the blood when their deficiency in the urine is ascertained, before the manifestations of uræmia take place, is an example. The ejection of poisons from the stomach and the employment of antidotal remedies belong in the same category.

These prophylactics apply to individual cases. A wider range of prophylactic measures takes in the destruction of the special causes of disease by disinfectants, the avoidance of exposure to these causes by removal or isolation, the observance of the laws of health as regards habits of life, etc.; in short, everything belonging to public and private hygiene. Preventive medicine, with this comprehensive scope of application, cannot be here considered. The prevention of particular diseases will, however, enter into their consideration in the part of this work devoted to the Practice of Medicine.

2. *Abortive Measures.*—The measures in this class are those employed with a view to arrest the progress of a disease at the outset—to cut it short or strangle it. Quinia given in large doses in intermittent fever is an abortive remedy. It is not easy to cite many examples of this class. The range in which abortive measures are effective, with our present knowledge, is small. Here is abundant room for further developments in therapeutics.

3. *Curative Remedial Measures.*—Those measures which abridge the duration of a disease, diminish its severity or danger, and, in general terms, modify or influence it in a favorable manner, may be distinguished as curative. Quinia is a curative remedy in the cases of periodical fever in which it is not abortive, and also in certain cases of neuralgia. Mercury and iodine are curative in cases of syphilis. Opium is a curative remedy in peritonitis, in spasmodic affections like colic, and in sporadic cholera. Many other examples of this class might be cited.

It is probable that remedies which are curative or abortive in certain diseases act by destroying morbific germs or organisms; that is, the remedies are parasiticides. Assuming the correctness of the germ doctrine as applied to all infectious diseases, an important object in therapeutics is to ascertain what particular remedy is destructive to the germs or organisms peculiar to each of these diseases. Experience teaches that a remedy which arrests one infectious disease may have no effect upon another. For example, quinia arrests and prevents malarial fever, but has no prophylactic or curative influence upon relapsing fever; and so, in order to have controlling remedies for all infectious diseases, it may be necessary to discover as many different parasiticides as there are varieties of disease-producing organisms.

4. *Palliative Measures.*—The measures belonging to this class are those which are employed with the object of relieving pain or suffering of any kind. These measures are directed, not to a disease *per se*, but to the symptoms or effects of a disease. But they may, to a greater or less extent, exert a curative influence. It is conceivable that relief of pain or suffering may conduce to the favorable termination of a disease in some cases.

5. *Sanitary or Hygienic Measures.*—The measures of this class relate to pure air, temperature, diet, cleanliness, moral influences, etc. Medicine has certainly derived as much advantage from this class of late years as from curative remedial measures. The improvements which have taken place in the management of diseases, as regards air, water, food, exercise, etc., are very great, but there is room for still further improvement.

6. *Sustaining or Supporting Measures.*—The measures of this class consist of tonic remedies, alcohol, and alimentation. The objects are to obviate a tendency to death by asthenia and to forestall a degree of prostration dangerous to life. Sustaining measures are applicable to the treatment of all diseases which, if they prove fatal, will destroy life by asthenia, and they are indicated with an urgency corresponding to the danger of dying by this mode. These, together with palliative and hygienic measures, constitute the treatment of all diseases which cannot be arrested and which are not amenable to curative measures. Not a few diseases will be found in this category.

The terms *analeptic* and *restorative* are applied to measures relating to diet, regimen, and remedies when the object is, in general terms, to bring the system back to its normal condition. Supporting measures have reference more especially to acute diseases which threaten life. Analeptic measures are those called for when the general health is deteriorated, although there may be no disease which involves danger to life—when, in other words, the system is *below par*. They are often called *building-up* measures, and are indicated in a large majority of disorders and chronic ailments.

A very good division of therapeutical indications is into—1st, the indications which relate to the removal or abatement of causes, or Causal indications; 2d, Symptomatic indications, or those having reference to the symptoms, irrespective of the nature and seat of the disease; and 3d, Pathological indications—that is, the indications derived from a knowledge of existing diseases considered with reference to their character, situation, and intrinsic tendencies.

Measures of treatment are sometimes classified and named from their action being in opposition to a particular morbid condition; thus, formerly, the term *antiphlogistics* was much in vogue, this name being applied to measures which were considered as opposed to inflammation. Of late, certain measures the immediate effect of which is the diminution of the temperature of the body are often distinguished by the name *antipyretic*. Quinia and some other remedies are called *antiperiodic*, from their efficacy in arresting affections which are characterized by intermittency; remedies which are antagonistic to the effects of syphilis are called *antisyphilitic*, etc. It is, however, an error to suppose that it is a governing principle in therapeutics to employ remedies or measures the action of which upon the body in health is the opposite of the effects or manifestations of disease. This principle is expressed by the term *allopathy*, a term applied by Hahnemann to denote a therapeutical doctrine the reverse of *homœopathy*. Legitimate medicine recognizes no such restricted and exclusive dogma as these two terms alike denote; and hence the name allopathy is to be by no means accepted, but should be considered as a term of unmerited reproach.

An important precept in therapeutics is the observance of simplicity in prescribing remedies. As a rule, remedies differing in their effects should not be given in combination or at the same time. Different remedies should be conjoined only when they co-operate for a common object; such, for example, as a cathartic or a diuretic operation. This precept is important, not only because different remedies are likely to conflict with each other, but because it is difficult to judge of their efficacy severally. To observe the separate effects of particular remedies in individual cases is essential in order to obtain that experience which will be useful to the same patient at other times and in other cases of the same disease. The experience of the physician accustomed to prescribe together multifarious remedies must, of necessity, be confused and uncertain. Moreover, complicated prescriptions are objectionable on the score of their tending to invest the practice of medicine with an unworthy mystery. These remarks of course do not apply to uniting with a curative remedy medicines designed to correct incidental effects which it is desirable to avoid, or to diminish the repulsiveness of a remedy or to secure for it a better tolerance. These objects in the combination of medicines are important. Not the least conspicuous of the modern improvements in medical practice relates to the use of alkaloids and concentrated remedies, together with other contrivances to divest remedies of a nauseousness which was formerly, as a matter of course, associated with the idea of a drug.

In the popular mind the practice of medicine is too closely connected with the use of drugs. Not disparaging the value of these, it is desirable for the public to understand that the proper office of the physician is to observe cases of disease, and to either prescribe or withhold remedies according to the indications. To forbear medicinal interference is as important in some cases as is active treatment in other cases, and to exercise this forbearance often requires on the part of the physician much firmness, on account of not only the expectations, or it may be solicitations, on the part of patients and others, but of his own anxiety to render efficient services. It should be an object with medical men to endeavor to correct popular errors in regard to the use of drugs and other therapeutic measures.

A considerable share of medical practice consists in the treatment, not of the individual diseases which are to be taken up in the second part of this work, but of disturbances of health which have no well-defined places in medical nosology. These disturbances often proceed from the operation of morbid causes, intrinsic or extrinsic, which it should be the aim of the physician to ascertain, and, if possible, to remove. The development of serious diseases may doubtless in this way frequently be prevented. In many cases the knowledge and skill of the physician are called into requisition to determine that no serious or well-defined disease exists. It is by no means the sole office of the physician to treat diseases. To indicate the cause of disease, more especially the voluntary violations of the laws of health; to enjoin such a course of life as shall seem most likely to afford security against disease and promote physical and mental vigor; to decide in cases of disease against the employment of active therapeutical measures,—these are duties not less important than the employment of potent remedies whenever the latter are required.

In the management of disease mental influences are often of not a little importance. The physician may in many cases effect much by judicious encouragement and by stimulating the voluntary efforts of the patient. Hopefulness and a strong will are curative agencies which are frequently powerful auxiliaries to medicinal remedies, and they are sometimes more potent than drugs; on the other hand, despondency and a lack of resolution have a depressing effect which in some cases tells greatly against recovery. Certain disorders may be produced and prolonged indefinitely by mental causes, the removal of which is essential to a cure. Too much concentration of the attention on the functions of the body is a common source of disorder among persons who from want of occupation unfortunately become unduly engrossed with their physical condition; and in such cases to divert the mind from the body is an object not less important and difficult than in other cases to secure a proper degree of care for existing ailments. The ability to exert those mental influences which are suited to different cases conduces in no small measure to success in practice; and this ability requires, in addition to natural powers, knowledge of human character and a tact which is acquired by experience. A cheerful mien and manners calculated to inspire confidence are by no means beneath the attention of the physician, but, on the contrary, they are to be cultivated as highly useful professional attainments. Medicine in its practical aspect involves knowledge, judgment, and skill as regards not only the employment of the several classes of the therapeutical measures which have been enumerated, but the mental influences suited to the circumstances peculiar to individual cases of disease.

The history of medicine shows many and great mutations in the prevailing doctrines of therapeutics at different periods. The medical practice of the present day in many respects is in striking contrast to that current a half century ago, and it is by no means improbable that a comparison with the present day at the end of the next half century will reveal points of contrast not less striking. These mutations, so far from being a reproach, illustrate the progressive character of medicine. Therapeutical principles can never become unchangeably fixed until the utmost limits of attainable pathological knowledge are reached and nothing further remains to be ascertained by experience. It would be absurd to claim for medicine this perfectibility, but continued improvement in the management of disease may be reasonably expected. Future improvement will doubtless embrace the addition of new remedies and measures of treatment, more extended and precise knowledge of those already in use, and the correction of errors which probably now, as heretofore, render sometimes the efforts of the physician not only inadequate but prejudicial.

The uniformity of the laws of disease at different periods and places has been already referred to. The bearing of this on the stability of therapeutics is obvious. So also with reference to the same application, it is important to assume that the system of man is not liable to alterations which render the operation of therapeutical agencies widely different at periods not far removed from each other. The opinion held by some that such alterations have occurred within the memory of those now living does not rest on any solid basis. Admitting to the fullest extent diversities attributable to race, climate, occupation, hereditary influences, etc., these do not invalidate the statement that the human constitution remains essentially unchanged as regards the application of established therapeutical principles. Were the truth otherwise, it would strike at the root of medical experience. If changes requiring a revolution in therapeutics were liable to occur with each successive generation, it is evident there could be no permanent principles of medical practice; the fruits of experience in our day, which so many are striving to develop, would be not only useless, but an injury to those who are to come after us.

The medical student and practitioner should have a just appreciation of the relative agency of nature and of art in the recovery from disease. It is customary and convenient to personify disease, speaking of it as of an enemy to be recognized, attacked, and, if possible, vanquished. Remedies and therapeutical methods are said to be curative, as if diseases were overcome exclusively by them. The mind should not be deceived by these figurative expressions. In reality, recovery from disease is always effected by nature; that is, by the agency of processes inherent in the organism. All that art can do is to remove obstacles to the operation of natural processes and to promote their favorable operation. Take as an example a remedy which is considered as specially curative—namely, quinia in periodical fever; this remedy, strictly speaking, does not cure the disease, using the term *cure* in its conventional sense, but it removes something on which the continuance of the disease depends. This something removed, nature effects the cure. As another example, opium does not cure peritonitis, but by promoting rest of the inflamed part and tolerance of the disease it affords aid to nature in effecting the cure. In like manner, all remedies and therapeutical measures exert their so-called curative effects in one or the other of these two ways. The expression *vis medicatrix naturæ*, which has been used from time immemorial, represents the agency of nature in the cure of disease. That the physician is the servant, not the master, of nature (medicus naturæ minister, non magister est), and that, using the term *cure* in its literal sense, he has the care of diseases, but nature effects the recovery (medicus curat sed natura sanat morbos), have long been recognized truths, as these old aphorisms prove.

Let it not be considered that to attribute supreme agency to nature is to disparage art. The triumphs of the latter are made none the less beneficent and brilliant by subordination to the former. The achievements of the human mind in every direction are dependent on circumstances and laws determined by a higher power.[1]

This chapter concludes the First Part of this work, the part devoted to the Principles of Medicine, or General Pathology. A succinct account of the changes belonging to general pathological anatomy has been presented, together with certain distinctions, definitions, and general considerations relating to etiology, symptomatology, diagnosis, prognosis, prophylaxis, and therapeutics. These subdivisions of the Principles of Medicine have been considered as fully as is deemed important by way of preparation for entering on the study of individual diseases. The latter will be considered in the remainder of the work, which is to be devoted to the Practice of Medicine, or Special Pathology.

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[1] The relative agency of nature and of art in recovery from disease is clearly and cogently set forth by Alexander Harvey, M. D., in his *First Lines of Therapeutics*, 1879.

PART II.

PRACTICE OF MEDICINE, OR SPECIAL PATHOLOGY.

INTRODUCTION.

Of the Different Aspects under which Individual Diseases are to be Considered in Treating of the Practice of Medicine, or Special Pathology; namely, Anatomical Characters, Clinical History, Pathological Character, Causation, Diagnosis, Prognosis, Prevention, and Treatment—Sense of the term Individual as applied to a Disease, and the several Grounds of Individuality—Varieties of a Disease—Definition of the terms Acute, Subacute, and Chronic—Symptoms or Events incidental to Diseases sometimes, for convenience, considered as Individual Diseases—Self-limited Duration of Certain Diseases—Nosology—Nosological Arrangement adopted in this Work.

THE *Practice of Medicine, or Special Pathology*, treats of individual diseases. In this department of medical study individual diseases are considered under several different aspects. One of these aspects relates to the appreciable changes found after death. These changes belong to morbid anatomy. Some diseases are characterized by morbid appearances in the parts affected, and other diseases are not accompanied by any lesions which are appreciable. The latter are distinguished as *functional diseases*. The term *anatomical characters* of a disease expresses the changes belonging to morbid anatomy which occur in connection with that disease. This term will be adopted as the title, usually, of the first aspect under which diseases not functional will be considered.

Another aspect relates to the phenomena or events which compose the *clinical history* of a disease. Under this head will be embraced precursory or prodromic events, the symptoms which accompany the access and those which occur during the career of the disease under consideration, the order of their succession, the laws regulating the development and progress of the disease, etc. This will constitute the second of the different aspects under which individual diseases, severally, are to be considered.

A third aspect relates to the *pathological character* of a disease. This aspect embraces what is known of the nature and seat of the morbid conditions which constitute or enter into the disease under consideration.

A fourth aspect relates to *etiology*, or the *causation* of a disease. Under this head will be stated what is known of the cause or causes of each individual disease.

A fifth aspect relates to *diagnosis*, or the discrimination of diseases. The application of the methods of investigation which by means of symptoms, laws, etc. enables the physician to recognize the disease under consideration, and to distinguish it from other diseases, will be considered under this head, Physical signs, whenever these are available in the diagnosis of a disease, will be embraced under this head.

A sixth aspect relates to *prognosis.* This aspect embraces the intrinsic tendencies of diseases as regards termination in death or recovery. The circumstances which denote, on the one hand, a favorable, and, on the other hand, an unfavorable, progress of diseases will come under the head of the prognosis. And the consequences more or less remote, or sequels, may sometimes be conveniently embraced under this head.

A seventh aspect relates to the *treatment* and *prevention* of a disease. The consideration of the treatment of each disease will embrace the indications for therapeutic measures and the remedies which experience has shown to be useful in that disease. Hygienic measures will also enter into the consideration of treatment. Prophylactic measures of treatment will claim consideration in treating of different diseases, and the measures which remove or obviate the causes of disease will often be found to be not less important than the judicious application of principles of therapeutics.

Before entering upon individual diseases it may be well to inquire what is meant by this use of the term *individual,* and whence does a disease derive its individuality. An individual disease differs from other diseases sufficiently for it to be regarded as separate and distinct. The sense of the term individual in this application is analogous to that of species in natural history. The individuality of a disease is established by distinctive features pertaining to its clinical history. These distinctive features may be derived exclusively from the seat of the disease. Thus, pleuritis and pericarditis are inflammatory diseases of a serous membrane, and have essentially the same pathological character; but the different anatomical relations of the pleura and of the pericardium furnish sufficient points of distinction to constitute each of these affections an individual disease. In other instances distinctive features are derived from the processes belonging to disease. Inflammatory diseases, for example, differ essentially from those which are not inflammatory. Again, certain diseases derive each an essential distinction from its exclusive dependence on a special cause. It is sufficient to show that a disease is produced by a special cause to establish its claim to be regarded as an individual disease.

An individual disease is to be distinguished from individual cases of disease. An individual case of disease has a relation to an individual disease like that of a particular man to the human species. The study of individual cases of disease belongs to clinical medicine. Some individual diseases embrace several varieties. Empyema and pneumo-hydrothorax may be considered as varieties of pleuritis, both consisting in inflammation of the pleura, the former differing from ordinary pleuritis in the fact that the liquid contained in the cavity of the pleura is purulent, and the latter differing in the fact that air and liquid are contained in that cavity. The several types of intermittent fever distinguished as tertian, quotidian, etc. are different varieties of one disease. In the progress of medicine certain diseases have come to be considered as essentially distinct which were formerly regarded as different varieties of one disease. This is true of all the eruptive fevers. It is probable that certain diseases now considered as individual will be hereafter found to be divisible into two or more.

Differences as regards severity and duration constitute a basis of the division of a disease into varieties. The same disease may be either *acute, subacute,* or *chronic.* A disease is acute when it has a certain degree of intensity and runs a rapid career. In general, an acute disease is of sufficient gravity to confine the patient to the bed. The subacute variety of a disease has less intensity, frequently not compelling the patient to keep the bed, and perhaps not preventing him from being about or even pursuing his usual occupation. The chronic variety exists when a disease is subacute and has continued for a considerable period. Some diseases are never chronic. This term, for instance, is not applicable to typhus or typhoid fever. The division into varieties, based upon the differences just named, is especially applicable to inflammatory diseases.

The list of individual diseases embraces some, recognized as such for the sake of convenience, which in reality are only effects or symptoms of diseases. Jaundice, for example, is an effect or symptom occurring in connection with different diseases. Dropsy is in the same category. It is, however, convenient to consider jaundice and the different forms of dropsy as if they were individual diseases. In fact, if we were better acquainted with the primary or elementary forms of disease, it would probably appear that not a few of the diseases now considered as individual are only manifestions of morbid conditions at present occult, the latter constituting the true diseases.

A point of distinction among diseases, interesting and of much practical importance, relates to duration. Some diseases continue for a definite period, never exceeding certain limits in this regard. The duration of certain diseases is regulated by fixed laws; that is, they rarely end until after the lapse of a certain number of days, and never exceed a certain number of days in their continuance. The continued and eruptive fevers are examples of diseases the duration of which has definite limits. Intermittent fever, on the other hand, has no fixed or definite duration if its course be not interfered with. Diseases which tend intrinsically to end after a certain time are distinguished as *self-limited.* The laws of different diseases with respect to self-limitation are often to be taken into account in the employment of therapeutical measures; and it is obvious that these laws are to be considered in estimating the amount of curative influence exerted by methods of treatment. One of the most striking and important of the characteristics of modern medicine is the knowledge which has been acquired of the self-limitation of different diseases. Facts have shown that even pulmonary phthisis in some cases ends in recovery from an intrinsic tendency thereto.

In treating of individual diseases some classification of them is essential. The classification of diseases constitutes a division of medicine called *nosology.* The history of medicine furnishes many nosological systems, the relative merits of which it would be useless to discuss. Undoubtedly, a scientific mode of classifying diseases would be to arrange them after essential points of distinction relating to their pathological character; but with our present knowledge of the pathological character of different diseases to do this is impracticable. A nosological arrangement on this basis must be deferred for a future period, when pathology shall have advanced far beyond its present condition. The same remark is applicable to any arrangement based on differences as regards the causes of diseases. Our present knowledge of etiology is not sufficient to furnish the basis of a permanent nosology. Inasmuch as the chief object of a classification is convenience, the proper course to be pursued, evidently, is to adopt a provisional plan which will best subserve this object; and the most convenient classification at the present time is that which has of late years been adopted by the majority of authors and teachers. This consists in dividing diseases into first, *general;* and second, *local.* General diseases are those which involve more or less disturbance of the whole system, this disturbance not having been preceded by any known local affection, local affections, if they occur, being developed secondarily. To this class belong the essential fevers. Under the head of constitutional diseases it is convenient to include some in which the local manifestations have no fixed seat and sometimes differ in character. Rheumatism and gout are constitutional diseases, the chief manifestations consisting of local affections differently situated in different cases and at different periods in the same case. The local affection, however, is in each essentially the same wherever seated. Syphilis is another constitutional disease manifesting itself in local affections which differ not only in situation, but in character. The want of fixedness or uniformity in the seat of local affections proceeding from constitutional disease, and their diversity in character in some instances, are the grounds for placing certain diseases in this class. A large proportion of the affections nosologically considered as local probably proceed from constitutional disease; but as the local affections are fixed and uniform, and our knowledge of the constitutional disease is, for the most part, inferential, it is more convenient to arrange them in the second division.

The second division, or local diseases, will embrace all which are not reckoned as general or constitutional. The number of local diseases is far greater than that of the diseases embraced in the first division. It is necessary to arrange them into subdivisions. The most convenient plan is to distribute them into groups corresponding to the distribution of the different organs of the body into physiological systems. According to this plan the different local diseases are subdivided as follows:

1. Diseases affecting the respiratory system;
2. Diseases affecting the circulatory system;
3. Diseases affecting the hæmatopoietic system;
4. Diseases affecting the digestive or chylopoietic system;
5. Diseases affecting the nervous system;
6. Diseases affecting the genito-urinary system.

The foregoing arrangement of local diseases will be adopted, and they will be considered in sections corresponding to the groups just enumerated. Afterward a section will be devoted to general diseases.

Of the diseases belonging to these several groups, those only will be considered which are medical as distinguished from surgical. The surgical diseases are those which are either external or in accessible situations. Thus, diseases of the mouth, throat, and nasal passages, of the anus and rectum, of the urethra and bladder, of the eye and ear, and of the vagina and uterus, fall within the province of surgery. Diseases of the skin, being external, belong to surgery, notwithstanding that they receive special attention from the physician perhaps oftener than from the surgeon. As an additional reason for not including them among the diseases to be considered in this work, there are many treatises devoted specially to their consideration.

SECTION FIRST.

DISEASES AFFECTING THE RESPIRATORY SYSTEM.

CHAPTER I.

ACUTE PLEURITIS.

Division of the Diseases affecting the Respiratory System into Inflammatory and Non-inflammatory.—Acute Pleuritis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis.

OF the diseases affecting the respiratory system, an obvious division is based on the existence or non-existence of inflammation. Inflammatory diseases are seated respectively in three important structures comprised in the organs of the respiratory apparatus—namely, the mucous membrane lining the air-passages, the serous membranes which invest the lungs, and the pulmonary parenchyma. Inflammation affecting the structures just named gives rise to pleuritis[1] or pleurisy, pneumonitis or pneumonia, and bronchitis. These will be treated of in the order in which they have just been enumerated. The first disease to be considered—namely, pleuritis—may exist as an acute and as a subacute or chronic affection, the inflammation being diffused over the affected membrane. These two varieties of the disease will claim separate consideration. Other varieties, to be considered separately, are empyema, pneumo-hydrothorax, and circumscribed pleuritis. It will be convenient to notice, in connection with these varieties of pleuritis, the dropsical affection known as hydrothorax.

Acute Pleuritis.

The pleura, like other serous membranes, is divided into a visceral and a parietal layer. The free surfaces of these two layers are smooth, polished, and covered with a single layer of flat nucleated cells called epithelial cells or endothelial cells.[2] These cells rest upon fibrillated connective tissue containing a network of elastic fibres, blood-vessels, and lymphatic vessels. The pleural cavities contain normally a small quantity of serum.

ANATOMICAL CHARACTERS.—The first noticeable change in acute pleuritis is redness of the pleura from active hyperæmia of the vessels within and beneath the affected portion of the pleural membrane. Small extravasations of blood, or ecchymoses, are not infrequently present at an early stage of the inflammation. The pleura loses its glistening appearance, and becomes dull and opaque in consequence of desquamation of the epithelium and beginning exudation.

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[1] With a view to uniformity as regards nomenclature, the terms pleuritis and pneumonitis are used instead of pleurisy and pneumonia.

[2] It has until recently been held that the peritoneal, pericardial, and pleural cavities are large lymph-spaces lined by a variety of connective-tissue cells called endothelium. Recent investigations have cast doubt upon this view, and have made it probable that these serous cavities are derived from the endoblast, and that their lining cells are epithelial in origin.

Very soon the surface of the pleura becomes covered either in patches or continuously with a delicate layer of fibrin. The fibrin appears first in the form of little clumps, which increase in thickness and coalesce with each other. The opposing surfaces of the pleura are agglutinated by the fibrin. The fibrinous exudation was called by the older writers coagulable lymph and false membrane. The fibrin varies in its microscopical appearance, being sometimes granular, sometimes homogeneous, and sometimes finely fibrillated. Certain constituents of the fibrin are furnished by the exuded liquor sanguinis, others by the altered tissue-elements and leucocytes. In some cases of pleuritis, particularly of the circumscribed variety, fibrin containing a few pus-cells is the sole inflammatory product. This form of the affection is called *fibrinous* or *dry pleuritis* (pleuritis sicca).

More commonly, acute pleuritis is of the *sero-fibrinous* variety. In this variety, in addition to the fibrinous coating of the pleura, the cavity contains serum, varying in quantity from a few ounces to several pints. The serum may be clear, but it is usually turbid from admixture with pus-cells and flocculi of fibrin. Upon microscopical examination both pus-cells and red blood-corpuscles are found in the fluid. The red blood-corpuscles may be so abundant as to stain the fluid red. The pleuritis is then called *hemorrhagic*, a variety observed especially in certain dyscrasiæ, such as scorbutus, and in tuberculous and cancerous disease of the pleura.

If the inflamed pleura be examined microscopically, a considerable number of emigrated white blood-corpuscles are found both in the pleural tissue and in the fibrinous layers on its surface. Part of the epithelial cells which normally cover the pleura undergo necrosis and desquamate, and a part, less severely affected by the inflammatory irritant, proliferate and produce flat cells of various shapes with vesicular nuclei. Some of the connective-tissue cells in the pleura swell up and also proliferate. The lymphatics of the pleura are dilated, and frequently contain fibrin, pus-cells, or proliferated endothelium. In from four to six days after the onset of the inflammation new-formed capillaries, which are offshoots from the pleural capillaries, make their appearance in the deeper parts of the layer of fibrin and cells covering the pleura. If at the height of the inflammatory process a microscopical section through the false membrane and the subjacent pleura be examined, the following layers can be distinguished: superficially, a layer of fibrin containing pus-cells; beneath this, a layer of embryonic or granulation-tissue composed of blood-vessels, leucocytes, and variously shaped larger cells which are derived from the epithelial cells and the connective-tissue cells of the pleura, and are intended for the formation of new connective tissue; and thirdly, the pleural membrane itself, containing dilated blood-vessels, leucocytes, and proliferating connective-tissue cells. In the layer of granulation-tissue more or less fibrin is also present. Some pathologists regard the proliferative changes in the epithelial and connective-tissue cells as purely regenerative, and separate them from the strictly inflammatory processes in the blood-vessels which give rise to the exudation of serum, fibrin, and pus.

The terminations of acute pleuritis are either in death, in resolution, in chronic pleuritis, or in empyema.

If resolution take place, the serum is absorbed, the fibrin, pus-cells, and red blood-corpuscles disintegrate and are likewise absorbed, and the granulation-tissue is changed into fibrillated connective tissue. In this formation of connective tissue, fibrillated basement-substance appears between the cells, which are gradually changed into flat, fusiform, or branching connective-tissue cells.

Some of the cells and some of the blood-vessels disappear. The remaining blood-vessels, which at first have thin, easily-ruptured walls, acquire more compact coats. The new connective tissue develops both in the form of diffuse fibrous thickening of the pleura, and in the form of thread-like or band-like adhesions between the two pleural surfaces. The long adhesions are produced by villous outgrowths of the granulation-tissue into the fibrin and fluid which during the inflammation separate the pleural surfaces from each other. It is probable that they may also be caused by stretching of short adhesions by the movements of the pleura. It will be noted that after the most favorable termination of pleuritis the pleura does not return to its normal state, but is left with a new growth of connective tissue, which binds the pleural surfaces together over a great or less extent. In this way the pleural cavity may become entirely obliterated by adhesions. The adhesions are at first weak, and may be broken by active exercise or by acts of coughing. Hemorrhage into the pleural cavity may in this way occur.

If after the lapse of three or four weeks the inflammatory products continue to increase, or remain stationary, or diminish very slowly, the disease has passed into the chronic stage.

When pus-cells accumulate in sufficient number, the exudation becomes purulent and the disease is called empyema. Chronic pleuritis and empyema will be considered subsequently.

The effects of large accumulations of fluid in the pleural cavity upon the lungs are of importance. If the lung be not restrained by adhesions, it is pressed upward and inward against the vertebral column. If the fluid accumulation be very extensive, the compressed lung loses all the physical characters dependent upon the presence of air in the air-cells. It resembles a mass of flesh, and is therefore said to be *carnified*. The pulmonary structure, however, remains intact, and it is capable of being inflated after death nearly or quite to the same extent as if it had not undergone this compression. A similar expansion may take place during life after the absorption of the fluid. So large an accumulation of liquid, however, occurs much oftener in chronic than in acute pleuritis.

Pleuritis may be general or circumscribed. Primary or idiopathic pleuritis is general. Circumscribed pleuritis—which, as already remarked, is often dry—is usually secondary to some affection of parts adjacent to the pleura. Circumscribed pleuritis may affect the pulmonary, the costal, the diaphragmatic, or the mediastinal pleura. Even in circumscribed pleuritis the two opposing surfaces of the pleura are usually inflamed—a circumstance which goes to show that the inflammatory irritant is contained in the exudation, for the opposed surfaces of the pleura have different vascular supplies.

CLINICAL HISTORY.—The most convenient division of acute pleuritis into stages is to consider as the *first stage* the period from the attack to the time when an appreciable quantity of liquid effusion has taken place; the *second stage* will extend to the time when the liquid begins sensibly to diminish; and the *third stage* will comprise the period occupied in the absorption of the liquid. The first stage, or the period anterior to an appreciable amount of liquid effusion, is usually of short duration. The physical signs may show the presence of liquid within a few hours from the date of the attack. This stage rarely extends beyond twenty-four hours. The second stage is of variable duration. The accumulation of liquid may go on with more or less rapidity. When the maximum amount is reached, the quantity may remain stationary for some time or absorption may speedily begin. The liquid may diminish under the effect of treatment, and renewed effusion take place. The disease does not, of course, advance beyond the second stage in the cases in which it eventuates in chronic pleuritis. If a considerable quantity of liquid remain at the end of from three to four weeks, the disease is to be considered as having become chronic. In cases which pursue a favorable course considerable progress should be made in the removal of the liquid after this lapse of time. The duration of the third stage, or the stage of absorption, is quite variable. Generally, some liquid remains when the general symptoms denote convalescence, and even after the patient has apparently recovered. The physical signs may show the presence of fibrin for some time after the date of convalescence. From what has been stated it follows that the duration of the career of the disease is from two to four weeks.

The *invasion* is often sudden and without premonition. In a certain proportion of cases, however, pain or soreness is felt for one, two, or three days before the development of acute inflammation. The development of acute inflammation is characterized by symptoms which sufficiently mark the date of its occurrence. It is sometimes ushered in by a well-pronounced chill, accompanied perhaps with rigors. Chill and rigors, however, are by no means so frequent, or, as a rule, so marked, as in cases of pneumonitis. Not infrequently chilly sensations and slight shiverings only are experienced.

Pain in the affected side attends the onset of acute inflammation in a large majority of cases. The pain is usually intense. It is sharp, cutting, or lancinating in character. It is felt especially in the act of inspiration; and it increases during inspiration, often becoming so severe that the act is instinctively arrested before being completed. Coughing and sneezing occasion severe pain. The movements of the body are painful, but in a far less degree than the respiratory movements. The pain is referred to the middle and lower third of the affected side of the chest, and is often diffused over the anterior, the lateral, and sometimes the posterior portion. It is not, as a rule, circumscribed or limited to a small space. The morbid sensitiveness of the inflamed pleura seems to me adequate to account for the production of acute pain as a result of the movements of the pleural surfaces upon each other and the stretching of the membrane, without requiring the hypothesis that either intercostal neuralgia or an affection of the costal muscles is superadded to the inflammation. Pain, with the characters just described, is almost uniformly present in acute pleuritis, but cases occur in which it is not marked, and is even wanting, other symptoms showing the inflammation to be acute.

Pyrexia is coincident with the development of the inflammation. The pulse is increased in frequency, rarely, however, exceeding 100 per minute. The temperature of the surface is raised. The thermometer in the axilla shows more or less intensity of fever, but the rise rarely exceeds 102°. As a rule, there is less pyrexia than in cases of pneumonitis. The usual concomitants of symptomatic fever—namely, thirst, anorexia, pain in the head and loins, restlessness, muscular weakness, etc.—are more or less marked. The intensity of the fever and of the general or constitutional disturbance varies much in different cases of this as of other inflammations. In rare instances the fever is intense, the temperature rising to 105° or higher, with a corresponding intensity of other constitutional symptoms (pleuritis acutissima). In most of these instances the pleuritis is of the suppurative variety, or empyema.

A comparison of the two sides of the chest as regards temperature shows a slightly higher temperature on the affected side, the difference being a fraction more than one degree.

Cough is usually present, but it is sometimes wanting. The pain in coughing leads instinctively to efforts to repress it, and this gives to the mode of coughing a character denoted by the term suppressed. The patient succeeds, to a certain extent, in these efforts. The expectoration is slight or wanting, and when present it consists of simple mucus. The exceptions to this rule are the cases in which the development of pleuritis is preceded by bronchitis. When these affections are associated it is merely a coincidence; there is no tendency in either one to give rise to the other.

The respirations are increased in frequency, but not greatly. Their increased frequency at this period is due to an arrest of the inspiration before it is completed, in consequence of pain. The patient instinctively multiplies the respiratory acts to compensate for the want of a full inspiration. As a rule, the patient in lying chooses a position on the healthy side, the weight of the body upon the affected side increasing the pain and soreness. This rule is by no means without exceptions. Not infrequently the decubitus is either dorsal or diagonal.

These symptoms constitute the clinical history of this disease during the first stage, or prior to effusion. They continue into the *second stage*, but after a certain amount of effusion has taken place they are materially modified. The pain is notably lessened, and may disappear except on forced breathing. The acts of coughing are less painful and the efforts at suppression are less apparent. The pyrexia diminishes, and may even cease during this stage. Other evidences of constitutional disturbance are less marked. The patient seems better, and may be able to sit up and perhaps walk about the room. The respirations are more or less frequent. Their frequency now depends on the compression of the lung by liquid. Their frequency will be great, other things being equal, in proportion to the quantity of liquid and the rapidity of the effusion. If a considerable amount of liquid be rapidly effused, the respirations are rapid; the patient suffers from a painful sense of the want of breath, or dyspnœa, and may be obliged to maintain the sitting posture. If the quantity be not large, and the effusion have not taken place rapidly, the patient will not be likely to suffer from dyspnœa while remaining quiet, but exercise will cause panting and a sense of the want of breath. Even with the pleural sac distended with liquid, the only effect on the breathing may be an increase of the frequency of the acts to twenty-five or thirty per minute, without suffering so long as the patient remains quiet. The diminution or cessation of pain is due to the restrained movements of the pleural surfaces upon each other in consequence of the presence of the liquid and fibrin, the latter perhaps agglutinating the portions of those surfaces which are in contact. The change as regards fever and other constitutional symptoms proceeds, in part, from the removal of the conditions causing pain, and partly because, after exudation has taken place, the disease is mitigated from an intrinsic tendency to decrease. The patient now, as a rule, prefers to lie upon the affected rather than the healthy side, as in this position he is able to expand more freely the opposite lung. To this rule, however, there are exceptions, and the decubitus in this stage, as in the first stage, is frequently either dorsal or diagonal.

During the stage of *absorption* pain is either wanting or is produced only by muscular efforts; fever has disappeared; cough diminishes or ceases; the respirations become less and less frequent, and are less hurried by exercise; the appetite and digestion return, and the normal condition of all the functions is gradually restored. During this stage the patient is convalescent; he is able to be out of doors, and perhaps to resume in part his usual avocation and habits. Convalescence is declared, and the symptoms, both general and local, may appear to denote that recovery has taken place while some liquid effusion still remains; the fibrin is not fully absorbed, and the formation of new tissues leading to permanent adhesions is going on. A sense of weakness in the affected side, together with some soreness or pain on unusual exertion, frequently continues for some time after the termination of the career of the disease.

Strümpell states that in the first stage of the disease, while effusion is taking place, the secretion of urine is notably diminished, and that the beginning of the absorption of the effused liquid is shown by a notable increase in the quantity of urine. This increase may thus serve to indicate that the disease has advanced to the second stage.[1]

In primary or idiopathic pleuritis the inflammation, as already stated, is diffused over the pleural membrane; it is general, not circumscribed. If circumscribed, it is a complication of some other pulmonary affection, as a rule. It will be seen hereafter that it occurs as a complication of pneumonitis and pericarditis, but it is generally, under these circumstances, subacute. Primary pleuritis does not lead to pneumonitis, except that the air-cells in immediate proximity to the pleura are sometimes involved; nor does it involve any tendency to bronchitis. It is a unilateral disease—that is, it affects the pleura of one side only. The exceptions to this rule are so infrequent that the disease, when bilateral or double, may be presumed to be secondary to another pulmonary affection, generally tuberculosis.

PATHOLOGICAL CHARACTER.—This disease is an example of acute inflammation affecting a serous membrane, and may be regarded as a type of all acute inflammations of serous membrane. The general pathology of inflammation has been considered in the first part of this work, together with the local variations when different structures are inflamed. Pleuritis and inflammations of other serous membranes are characterized by fibrinous exudation as a constant event. (Vide p. 37.) This event, the accumulation of liquid in the closed cavity, and the development of connective tissue are characteristics of these inflammations.

CAUSATION.—Acute pleuritis may be either *primary* or *secondary*. The disease is primary when it occurs independently of any other affection. Primary or idiopathic pleuritis is attributed in certain cases to the action of cold. This has so long been considered as a frequent source of a great number of diseases that patients at once are led to refer an attack to some exposure, often, doubtless, when it had no causative agency or at most acted only as an exciting cause. Still, it is probable that the disease sometimes originates in this way. Acute pleuritis may be produced traumatically by contusions, especially if accompanied with fracture of the ribs, and by penetrating wounds. It is remarkable, however, that severe injuries of the chest often occur without giving rise to general pleuritis.

Primary pleuritis is much less frequent than was formerly supposed. In the majority of cases of pleuritis careful examination, both before and after death, shows the pleuritis to be secondary to some local or constitutional disease. Affections of parts adjacent to the pleura play an important rôle in the causation of pleuritis. The most important of these local affections which cause secondary pleuritis are lobar pneumonitis, lobular pneumonitis, pulmonary tuberculosis, infarctions, abscesses and gangrene of the lung, cancer and perforations of the œsophagus, caseous tubercles in the bronchial glands, caries of the vertebræ and ribs, and inflammations in the mediastinum. The various inflammations of the peritoneum and of the pericardium often lead to pleuritis.

Pleuritis is a frequent complication of many infectious diseases, such as scarlatina, variola, pyæmia, septicæmia, and acute articular rheumatism. It occurs also not infrequently in Bright's disease, scorbutus, and gout.

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[1] *Lehrbuch der Speciellen Pathologie und Therapie der inneren Krankheiten*, Leipsic, 1885.

Pleuritis may be secondary to other diseases of the pleura, such as cancer, sarcoma, and tubercle.

In cases which appear to be primary pleuritis, careful search should be made for some hidden cause, such as tuberculosis or Bright's disease. In some cases, however, no cause can be discovered.

DIAGNOSIS.—Although the symptoms which enter into the clinical history of acute pleuritis are sufficiently characteristic to point to the existence of the disease, they are not sufficient for the diagnosis in all cases. The characteristic pain is not peculiar to the disease. The pain in some cases of intercostal neuralgia and pleurodynia is as strongly marked as in acute pleuritis, and these three affections were formerly often confounded in practice. Moreover, the pleuritic pain in some cases of pneumonitis is as intense as when the pleuritis is a primary disease, and the former disease has not infrequently been mistaken for the latter. On the other hand, acute pleuritis is liable to be overlooked in the cases in which pain is slight or wanting. More or less fever is present in acute pleuritis, but this symptom may be associated accidentally with intercostal neuralgia; it is likely to be present in pleurodynia, and it exists, of course, in pneumonitis. The diagnosis is to be based on the physical signs taken in connection with the symptoms. The latter are of importance, and, indeed, are essential in determining whether the affection be acute or not. The differential diagnosis consists in discriminating between the disease under consideration and intercostal neuralgia, pleurodynia, and pneumonitis.

The diagnosis in the first stage, with the aid of physical signs, cannot always be made with positiveness. Examination of the chest by inspection will show restrained movements on the side to which the pain is referred, the movements on the opposite side being increased, while on percussion distinct disparity between the two sides may not be found; and on auscultation the murmur of respiration is more or less weakened on the affected side. These signs are due to the fact that the pain leads instinctively to a diminished use of the lung on the side affected, while the use of the other lung is increased. They are equally present when the pain is incident to the affections from which this disease is to be discriminated. A slight friction murmur may be discovered in this stage; and if present its diagnostic significance is important. It shows that pleuritis exists, and the discrimination then lies between pleuritis as a primary disease or as a complication of pneumonitis. To decide this point at once is not always easy. The diagnostic sign of pneumonitis—namely, the crepitant râle, if present—settles the question; but as this sign is by no means present uniformly in pneumonitis, its absence does not authorize the exclusion of that disease. The same remark is applicable to the characteristic expectoration of pneumonitis. It must be confessed that in a certain number of cases this differential diagnosis requires some delay. A friction murmur is heard in only a certain proportion of cases during the first stage, and if absent the signs belonging to the second stage are essential in order to render the diagnosis positive.

The signs belonging to the second stage are usually present without much delay. If the disease be idiopathic pleuritis, after the lapse of twelve or twenty-four hours effusion will have taken place in sufficient quantity to be apparent. The signs now denoting the existence of the disease are those due to the effusion. The diagnosis is based upon these signs, in conjunction with the symptoms and history, and on the absence of the signs of pneumonitis.

The signs of effusion are obtained by percussion, auscultation, palpation, and inspection. On percussion, dulness or flatness is found at the base of the chest, extending upward in proportion to the quantity of liquid, the patient being in a sitting posture. By the finger, which serves as a pleximeter, there is also perceived an increased sense of resistance on percussion. The upper limit of the dulness or flatness, the position of the body being vertical, is not on a continuous horizontal line extending over the posterior, lateral, anterior aspects of the chest. In most instances it is higher behind than in front. Prof. G. M. Garland has found that on the posterior aspect the effusion is represented by a curved line resembling the letter ∽.[1] Having ascertained on the anterior aspect of the affected side a point which divides the dulness or flatness below from the resonance above while the patient is sitting, changing the position of the patient to recumbency on the back frequently induces a marked change in the dividing-line between dulness or flatness and the resonance; the liquid gravitating to the posterior part of the chest, the lung descends in front and furnishes resonance for a greater or less distance below the point which was coincident with the level of the liquid while the patient was sitting. This latter test of the presence of liquid is not available in all cases. If the pleural surfaces above the level of the liquid be united by old adhesions or agglutinated by fibrin, the liquid and lung will maintain the same relation in different positions of the body. The failure of this test, therefore, is not proof against the presence of liquid. The test, however, is available in a large majority of cases. The resonance over the lung above the level of the liquid, if the latter rise to a third, a half, or perhaps two-thirds the height of the chest, is increased, and acquires in part a tympanitic quality, constituting vesiculo-tympanitic resonance. That this vesiculo-tympanitic resonance may be due to emphysema of the upper lobe of the lung I have had an opportunity of demonstrating. A hospital case presented the signs of pleuritis with considerable effusion, the vesiculo-tympanitic resonance above the liquid being marked. The patient unexpectedly and suddenly died, and the cause of death was found to be a large thrombus in the left ventricle, the cardiac walls having undergone fatty degeneration. The lower lobe of the lung on the affected side was condensed, and the upper lobe was considerably emphysematous.

On auscultation the respiratory murmur is either suppressed or is feebly appreciable below the level of the liquid. Above the liquid it is usually weak as compared with the murmur on the opposite side. If the lung be considerably reduced in volume by the amount of liquid, the respiratory sound becomes broncho-vesicular and the vocal resonance is increased. If the liquid be sufficient to compress the lung into a solid mass, the latter furnishes the bronchial respiration. The bronchial respiration is heard over the site of the compressed lung. It is usually limited to that site, but in a certain proportion of cases it is diffused over the greater part or the whole of the affected side. The normal vocal resonance, as a rule, is either suppressed or diminished over that portion of the affected side which corresponds to the space occupied by the liquid; but if the accumulation of liquid be large, bronchophony may be produced either over the compressed lung or the portion in contact with the liquid. The bronchophony has sometimes a tremulous or bleating character, and is then ægophony. If the chest be filled with liquid, bronchophony is sometimes diffused over the whole of the affected side.

Vocal fremitus is either arrested or diminished by effusion, as a rule. From this fact is derived valuable evidence of the presence of liquid when the effusion is in the right pleural cavity, in consequence of the greater amount of fremitus over the right side in health. If the question be as to the presence of liquid in the right pleural cavity, and the fremitus be greater on the left than on the right side, the evidence of liquid is strong. There are some rare exceptions to the rule that the vocal resonance and fremitus are either diminished or suppressed over the space occupied by the effusion.[1]

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[1] Vide *Pneumono-dynamics*, 1878.

If the quantity of liquid be large enough to distend the pleural sac, the intercostal depressions may be diminished or destroyed at the inferior, anterior, and lateral portions of the chest. This constitutes valuable evidence of the presence of liquid.

Other and marked signs of effusion are present if the quantity of liquid be sufficient to dilate the chest. This occurs exceptionally in acute pleuritis. It occurs much oftener in the chronic variety of the disease, and the signs referred to will be noticed in connection with the diagnosis of the latter.

The physical signs not only lead to a positive diagnosis after the first stage, but they show the amount of liquid in the pleural sac—a point of importance with reference to treatment. By means of daily explorations the physician is able to ascertain whether the effusion be increasing or diminishing, and the rate of either the increase or diminution. The information thus obtained is highly important as a guide in the employment of therapeutical measures. This information cannot be obtained from the symptoms. The signs, therefore, are essential not only to the diagnosis, but to a proper knowledge of the progress of the disease.

After absorption, to a greater or less extent, of the liquid, the pleural surfaces coming into contact over a larger area, and roughened by fibrin which has now become dense and closely adherent, a friction murmur is frequently produced. This is sometimes so loud as to be heard by the patient. It may continue into convalescence, ceasing when the pleural surfaces have become united by means of newly-formed tissue. A friction murmur, however, by no means occurs invariably, even at this stage of the disease.

The occasional occurrence of a pleural, cardiac friction sound in cases of pleuritis affecting the left side is to be borne in mind. The movements of the heart sometimes occasion a rubbing of pleural surfaces sufficiently to cause a sound which, having the cardiac rhythm and being heard when breathing is suspended, may seem to denote pericarditis. The sound is limited to the left border of the heart, and pericardial effusion is of course wanting. Pericarditis, when this sign is present, it must be confessed, is not at once easily excluded. An error of diagnosis in this regard is more likely to occur from the fact that this disease is not a very infrequent complication of pleuritis.

PROGNOSIS.—The prognosis in cases of acute primary or idiopathic simple pleuritis is always favorable, provided the disease be uncomplicated and the constitution of the patient be not enfeebled. Without any therapeutical interference it would very rarely prove fatal. I have collected several cases in which the disease passed through its course favorably without any treatment. It may possibly prove fatal in consequence of a large amount of effusion occurring very rapidly, death taking place by apnœa, produced, probably, by congestion of the lung on the unaffected side (collateral fluxion), in addition to the loss of function of the lung on the affected side from compression by the liquid. I have known an instance in which the death of a hospital patient suddenly and unexpectedly seemed fairly attributable to the fact that the pleural sac was greatly distended with rapidly-effused liquid. Developed as a sequel to other diseases, or in the course of a grave affection, or in persons with broken constitutions, it may destroy life by asthenia. Acute pericarditis is sometimes developed simultaneously with, or during the progress of, pleuritis, and these associated diseases are liable to prove fatal. Their occasional coincidence is to be borne in mind, or the pericarditis in this connection, if not carefully sought for, will be likely to be overlooked. As already stated, the development of pneumonitis consecutive to pleuritis is not to be expected, nor is there much danger that an acute simple pleuritis, after its development, will eventuate in suppuration, constituting the variety of the disease called empyema. In short, the intrinsic tendency of the disease is to recovery, a fatal termination being due very rarely to the disease *per se*, but to affections with which it may be associated.

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[1] Vide article by the author, entitled "Large Pleuritic Effusion in the Right Side, without Notable Diminution of Vocal Resonance and Fremitus," *Am. Journ. of Med. Sciences*, April, 1882.

CHAPTER II.

ACUTE PLEURITIS (CONTINUED).

Treatment of Acute Pleuritis—Measures indicated in the First Stage—General Considerations relating to Bloodletting in Acute Inflammations—The Use of Opium in Acute Pleuritis and other Acute Inflammations—Measures indicated in the Second and Third Stages of Acute Pleuritis.

THE objects of treatment in acute pleuritis differ in its different stages, and the latter therefore claim, severally, distinct consideration with reference to therapeutic indications. The objects in the first stage are to arrest, if possible, the progress of the disease, to diminish its intensity, if it be not arrested, to limit the amount of morbid products, and to relieve suffering; in other words, the treatment relates to the employment of abortive, curative, and palliative measures. The indications in the first stage of this disease are essentially the same as in the early period of most acute inflammations. The general principles which should govern the treatment in this stage, therefore, will be here considered, and simply referred to hereafter in connection with other diseases to which they are measurably applicable.

Owing to its brief duration, patients are frequently not seen until the first stage has passed. Can the disease be arrested at this stage if the opportunity offer and a positive diagnosis be made? Admitting that physicians formerly did sometimes succeed, as they believed, in cutting short the disease, it must be said in answer to this question that there are no known abortive means which can be relied upon. Bloodletting and other so-called antiphlogistic measures formerly employed for this object have certainly not been successful in a sufficient number of cases to warrant their employment to an extent which will render them likely to do harm if they be not successful. Assuming that the disease goes on to the second stage, the question then is, What curative and palliative measures are to be employed? This question leads at once to the consideration of bloodletting.

A great change has taken place with respect to bloodletting in the treatment of acute inflammations. This measure was formerly thought to be highly important, and was rarely omitted. It is now considered by many as seldom, if ever, called for. The infrequent use of the lancet now, contrasted with its frequent use forty years ago, constitutes one of the most striking of the changes in the practice of medicine which have occurred during this period. It can hardly be doubted that this measure was formerly adopted too indiscriminately, and often employed too largely; but the natural tendency being to pass from one extreme to another, the utility of bloodletting in certain cases at the present time is perhaps not sufficiently appreciated.

Experience and pathological reasoning combine to show that bloodletting rarely has a direct controlling effect upon an inflammatory disease. It may exert a powerful immediate effect as a palliative measure, but whatever curative power it may possess is exerted indirectly., Its therapeutic action in general consists in lessening the frequency and force of the heart's action, and diminishing arterial tension and the intensity of pyrexia. In the early period of an acute inflammation accompanied by notable pyrexia and arterial tension the abstraction of blood affords relief, and may contribute to a favorable progress of the disease. It should enter into the treatment of a certain proportion of cases, provided other and more conservative means for the same ends were not available.

The evils of bloodletting arise from its spoliative effect upon the blood. It diminishes the red globules, and these, during the progress of an acute disease, are not readily reproduced. It induces thus the anæmic condition, and in this way impairs the vital powers. It will be likely to do harm, therefore, whenever it is important to economize the powers of life, and it may contribute to a fatal result in diseases or cases of disease which involve danger of death by asthenia.

The useful effects of bloodletting may frequently, if not generally, be obtained by other means which require less circumspection in their employment, because if injudiciously resorted to they are in a less degree hurtful. The mass of blood may be temporarily lessened by saline purgatives and diaphoretic remedies, conjoined with a restricted ingestion of food and liquids. Depletion is obtained in this way without impoverishment of the blood. The frequency and force of the heart's action may be affected by nauseant sedatives, such as tartar emetic, ipecacuanha, etc., and by direct sedatives—namely, digitalis, aconite, and the veratrum viride. By antipyretic remedies and measures for the abstraction of heat the pyrexia may be lessened without the expenditure of blood, and thus the evils of bloodletting be avoided. The advantage of bloodletting consists mainly in the promptness of its operation. Several hours are required to secure results from the means employed in lieu of bloodletting, whereas the effects of the latter are produced in a few moments.

In accordance with these views, bloodletting is never indicated by the fact simply that acute inflammation exists; it is a measure directed not to the disease *per se*, but to circumstances associated with the disease. The state of the circulation and other circumstances furnish the indications for the employment of this measure. It may be admissible if, with the development of inflammation, there exist notable pyrexia, the patient being robust and in good health when attacked, and the disease not involving danger of death by asthenia. The measure is admissible, under the conditions just stated, whenever the promptness with which its effects are obtained renders it desirable to adopt it in preference to other measures producing the same effects with more or less delay. *Per contra*, bloodletting is not admissible when the development of inflammation is not accompanied by notable pyrexia, when the patient was not in good health when attacked, when the constitution is feeble, and when the disease involves danger of death by asthenia. These rules of practice, while they accord to bloodletting therapeutic value, undoubtedly restrict its use within narrow limits.

Applying these rules to the disease under consideration, a patient in the first stage of acute pleuritis, robust, suffering from severe pain and dyspnœa, with high fever, the pulse moderately accelerated and not weak, will derive immediate relief from the abstraction of from ten to sixteen ounces of blood. The loss of this quantity of blood under such circumstances in a disease like this, which does not tend to destroy life by asthenia, will give rise to no evil results, but will be likely to affect favorably the progress of the disease. On the other hand, a patient feeble or anæmic, with a pulse more or less frequent, but denoting diminished power of the heart's action, should not be bled, notwithstanding the local symptoms would undoubtedly be thereby relieved. By impairing the vital powers the loss of blood will do harm, and is not admissible under these circumstances merely as a palliative remedy. And in the first case, if the local symptoms do not urgently call for immediate relief, other measures may be substituted for the bloodletting. It may be added that, aside from the immediate relief afforded in acute pleuritis by bloodletting, it has been theoretically considered as preventing, in a measure, fibrinous exudation by lessening arterial tension.

Before leaving the consideration of bloodletting several incidental points may be briefly noticed.

This measure is perhaps more applicable to the treatment of inflammation affecting the pulmonary organs than to the treatment of other inflammatory affections, in consequence of the relations of the former to the circulation. The free passage of the blood through the pulmonary circuit seems to be promoted, and the functional labor which the lungs have to perform is diminished by the abstraction of blood. At all events, relief of the pain and dyspnœa attendant on the early stage of acute inflammation of any of the pulmonary structures is more quickly and effectually procured by bloodletting than by other measures. Were it not for its ulterior effects, it would be invaluable as a palliative measure in pleuritis and other inflammatory affections within the chest.

In another mode the abstraction of blood may sometimes be peculiarly useful in affections of the lungs involving considerable or great obstruction to the passage of blood through the pulmonary circuit. This obstruction occasions an accumulation of blood within the cavities of the right side of the heart; hence there is congestion of the systemic veins, giving rise, if sufficient in degree, to lividity or cyanosis. The accumulation of blood in the cavities of the right side of the heart under these circumstances weakens the force with which the ventricle and auricle contract, and paralysis of this portion of the heart from over-distension may follow. The formation of a heart-clot may be a consequence of this accumulation. In certain cases bloodletting may be advisable with a view to diminish the accumulation of blood in the cavities of the right side of the heart; but it is to be employed for that end only when the contraindications which have been mentioned are not present.

After bloodletting, the pulse sometimes appears to indicate increased power of the heart's action. The artery seems to strike against the finger with more force than before the abstraction of blood. Formerly, practitioners were misled by this effect upon the pulse, and bloodletting was employed as a means of increasing the power of the heart's action. The sensation which the finger receives in these cases is delusive, and is caused by the quickness of the movements of the artery. This has been shown by the sphymograph to depend on the diminished tension of the arteries following the abstraction of blood. It is to be borne in mind, in estimating the power of the heart's action by the sensible characters of the pulse, that the sense of resistance which is felt and the amount of pressure required to compress the artery are the evidences of strength.

To a certain extent the advantages of bloodletting, inclusive of the promptness of its operation, may be obtained by arresting temporarily or retarding the circulation in a part more or less remote from the seat of the inflammation. A ligature applied to one or more of the extremities will effect this object; so also will the removal of atmospheric pressure over a considerable space. There is reason to think that these measures as substitutes for bloodletting have not been sufficiently considered. It is probably owing to their effect in withdrawing temporarily a certain quantity of blood from the circulation that dry cups and various so-called revulsive applications to the surface of the body are of utility in the first stage of acute inflammation. A considerable number of dry cups applied to the chest constitute an efficient substitute for bloodletting, often affording promptly notable relief of pain and dyspnœa.

The evils of indiscriminate and excessive bloodletting are manifested by a larger rate of mortality in those diseases which tend to destroy life by asthenia, and it can hardly be doubted that the death-rate has been diminished by a much more sparing use of the lancet within late years. But the results of injudicious bloodletting are manifested in cases which end in recovery as well as in those which end fatally. These results consist in protracted convalescence and subsequent feebleness. The cases of different inflammations treated formerly by bloodletting, together with other measures entering into the so-called antiphlogistic method, and the cases now treated otherwise, present a striking contrast as regards the condition of patients during convalescence and after recovery.

The opinion held by some that diseases have undergone a notable change within the memory of those now living has been already referred to.[1] They who hold this opinion suppose that bloodletting and other antiphlogistic measures are less appropriate now than formerly, in consequence of such a change. With a professional experience of over fifty years, I do not hesitate to express a conviction that acute inflammations at the present day are essentially the same as they were as far back as the time just named, and that antiphlogistic measures were neither more nor less appropriate then than now.

In addition to general bloodletting or the employment of venesection, much importance was formerly attached to the abstraction of blood by cups or leeches applied in the neighborhood of the inflamed part. Local bloodletting in some cases is more convenient than venesection; but, so far as the abstraction of blood is concerned, it is difficult to conceive that it is a matter of much importance from what part of the body or vascular system it is taken. Whether it be abstracted by means of cups, leeches, or the lancet, the benefit or injury will depend on the quantity withdrawn in a given period. Whatever advantage may accrue from the removal of a certain amount of blood by cups or leeches over the extraction of the same amount by venesection must be derived from the operation of the former as a revulsive measure.

With these remarks, leaving the consideration of bloodletting, and reverting to the question concerning the curative and palliative measures to be employed in the first stage of acute pleuritis, we are led to the consideration of opium. And with respect to this remedy certain considerations may here be presented which will apply to the treatment of other inflammations.

A great change has taken place with respect to the use of opium in acute inflammations. It was formerly used with much reserve, under the apprehension that, acting as a stimulant, its influence upon the local disease must be unfavorable. It was regarded as antagonistic to the antiphlogistic plan of treatment. Clinical experience and sounder pathological views, however, have led to the knowledge of its great value in the treatment of inflammatory affections wherever situated. It is valuable, not alone as a palliative but as a curative remedy. Its palliative efficacy is of course intelligible, and we can also to some extent understand its curative influence. By relieving pain it diminishes the determination of blood to the inflamed part; for pain is alone sufficient to occasion an increased afflux of blood to a part. This fact is illustrated by cases of neuralgia affecting a superficial nerve. Neuralgia of the supraorbital nerve, for example, occasions a determination of blood to the conjunctiva, so that this membrane appears to be inflamed, and the congestion disappears as soon as the pain is relieved by a full opiate. Opium exerts also a curative influence by preventing or diminishing the constitutional disturbance which inflammations are apt to occasion; it places the system in a condition to tolerate better the local affection. The severity of an inflammatory disease depends much on the amount of constitutional disturbance which it occasions; and with respect to the latter different cases differ widely, although in each the degree and extent of the local affection be the same. Life is often destroyed, not in consequence of the injury done to the inflamed part, but because the vital powers are inadequate to bear the continuance of the disease. These considerations afford some insight into the utility of opium, especially when patients are able to take this remedy without inconvenience, there being, as is well known, a great difference among different persons in this regard. The evidence of the curative value of opium, of course, is derived from experience, and there is ample ground for the belief that by its judicious use in acute inflammations not only is their course divested of much of the suffering which would otherwise be experienced, but their intensity is lessened, and the danger of death diminished in those inflammations which tend to destroy life by asthenia. The immediate effect of this remedy is often very strikingly manifest in improvement as regards the local and general symptoms.

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[1] Vide p. 110.

Considering, now, the use of opium in the first stage of acute pleuritis after the abstraction of blood, if this be deemed advisable, or after a saline purgative, if this be employed in lieu of bloodletting, it will generally be judicious to prescribe opium,[1] in some form, in doses sufficient to relieve pain and place the system fairly under an anodyne influence. If the pain be severe and the constitutional disturbance considerable, the use of opium need not be delayed for the operation of a purgative, but may be at once entered upon. This is perhaps the most judicious course in the majority of cases, a purgative being deferred until a distinct anodyne impression has been produced and maintained for some hours by opium. Conjoined with the use of opium, sedative remedies, addressed to the circulation, may be employed. These should not be pushed to the extent of producing disorder. If, for example, tartar emetic be given, it should not be carried beyond the point of slight nausea, and the veratrum viride, if given, should occasion no appreciable effects beyond a reduction of the frequency of the heart's action. These remedies, it is to be borne in mind, are addressed, not to the disease *per se*, but to the symptomatic phenomena of the disease.

Aconite is a valuable sedative remedy in the early stage of this as of other acute inflammations. The most effective mode of administration in order to secure promptly and fully its remedial effect is that recommended by Ringer—namely, half a drop or a drop of the tincture to be given every ten minutes or quarter of an hour for two hours, and afterward the dose repeated hourly. Smaller doses are to be given if the pulse become feeble or there be much prostration.

A summary of the treatment of the first stage of acute pleuritis, then, will embrace bloodletting in some cases, the use of opium given sufficiently to relieve pain and tranquillize the system, a saline purgative, and remedies designed to act as sedatives upon the circulation. Blisters should not be applied in this stage. They occasion general disturbance more than enough to counterbalance any effect by way of revulsion. They add an inflammation of the skin to the existing inflammation of the pleura. A sinapism, turpentine stupes, or fomentations to the chest will secure all the benefit of vesication without the annoyance and other evils of the latter. Moreover, a blister is an obstacle in the way of those examinations of the chest which are of importance as affording the only reliable information respecting the progress of the disease. The employment of cold in the form of the bath, the wet sheet, or sponging the body, and other antipyretic measures, are rarely called for in this disease, inasmuch as a high temperature (hyperpyrexia) is of exceptional occurrence. These measures, however, are indicated if the body-heat rise to 104° or 105°, as occurs in the so-called pleuritis acutissima.

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[1] The word "opium" is here used as a general term, including the alkaloids, morphia and codeia.

The application of cold to the affected side by means of the ice-bladder or by napkins dipped in ice-water and renewed every five minutes is a safe and efficient measure for the relief of pain. Theoretically, the application of cold for a local effect may be considered to be useful by causing contraction of the vessels and thereby lessening inflammatory exudation.

Preventing the respiratory movements of the chest on the affected side, by means of long strips of adhesive plaster encircling this side and extending partially over the side not affected, affords notable relief of the lancinating pain felt in the acts of inspiration.

In the second stage, so long as the quantity of effusion is small and a further accumulation may be expected to take place, the indications belonging to the first stage may continue. If the pain be still acute and the fever have not abated, measures to relieve the former and lessen the latter are indicated. Bloodletting is rarely called for, but depletion by purgatives may be useful, not only by lessening fever, but by restraining the amount of effusion. Diaphoretic remedies are useful by contributing to the two ends just stated. Opium is indicated in proportion to the pain and constitutional disturbance, and sedative remedies addressed to the circulation may be employed.

These measures cease to be appropriate when considerable effusion of liquid has taken place, when pain is no longer a prominent symptom, and when the pyrexia has either ceased or become much diminished. The object of treatment now is to promote absorption of the effused liquid. The means which may be employed for this object are hydragogue purgatives, diuretics, blisters, and the use of certain remedies which have been supposed to act directly as sorbefacients.

Hydragogue purgatives act most promptly and efficiently. The more active hydragogues, which are drastic in their operation, should be given circumspectly, and generally need not be employed. Reference is had to such remedies as elaterium and gamboge. The saline purgatives, such as the bitartrate of potassa, either alone or in combination with jalap (pulvis purgans), the citrate or the sulphate of magnesia, are to be preferred as sufficiently effective and less severe. These will sometimes effect a rapid decrease of the liquid.

Diuretics are sometimes equally but less rapidly effective. They effect the object with less perturbation and are less debilitating. The union of two or more diuretic remedies is more likely to prove effective than a single remedy of this class. Half an ounce of the infusion of digitalis, freshly made, given three or four times daily, and either the bitartrate or the acetate of potassa given in addition, form a combination which is often efficient.

Restriction in the amount of fluids ingested is an important point with reference to absorption. The elimination of water by the bowels or kidneys is of little avail if the patient be allowed to take as much into the system as is eliminated. The quantity should be as small as is compatible with comfort.

Blisters in this stage of the disease doubtless contribute to the object under consideration. If other means be effective, however, they may be dispensed with. Their interference with examinations of the chest is a matter of considerable consequence, and the annoyance which they frequently occasion is not an inconsiderable objection. If employed, small blisters successively applied in different situations, removed as soon as vesication commences, the vesicated surfaces being allowed to dry up rapidly, accomplish all the good to be effected in this way, with the smallest amount of inconvenience both to the physician and patient.

Remedies which have been supposed to act as sorbefacients are mercury and iodine. Mercury I have long ceased to employ in this disease. Of doubtful efficacy, the annoyance and other evils incident to mercurialization render it objectionable. The efficacy of iodine is perhaps equally doubtful, but it is not open to similar objections. It may be given internally or applied externally in the form of either the ointment or tincture.

It is an important injunction not to continue perturbatory measures with a view to promote absorption too long nor too push their use too far. The decrease of effusion after it has reached a certain point is likely to go on slowly, for reasons already stated. Whenever this point is reached. cathartics and diuretics should be discontinued or employed with great moderation, else they will be likely to do harm rather than good, by weakening the patient; and if at any time these measures appear to impair the general condition, the propriety of continuing them is doubtful. A leading object in the second stage of the disease is to maintain the constitutional strength. For this object the diet should be nutritious; tonic remedies are useful, and a little wine or spirit may be allowed. These measures should constitute the treatment after those designed to promote absorption have been sufficiently carried out, and should take the place of the latter whenever there is evidence of the strength giving way.

It is very rarely the case that this disease tends to a fatal ending by asthenia. Should such a tendency be manifested by the symptoms—namely, frequency and feebleness of the pulse, muscular prostration, etc.—the measures which constitute the supporting treatment are of course indicated. These measures, which form the most important part of the treatment of the diseases involving danger by asthenia, consist of alcoholics, nutritious alimentation, and tonic remedies. They will be noticed more fully in connection with diseases in the treatment of which they are oftener called for. (Vide Treatment of Pneumonitis, Chapter IV.)

A fatal result in acute pleuritis may be due to rapid and excessive effusion of liquid, as already stated. If the liquid accumulate sufficiently to endanger life, it should be at once withdrawn from the chest by aspiration. This measure is much more frequently indicated in chronic pleuritis, and will be considered in connection with the latter variety of the disease.

The measures which have reference to the promotion of absorption may continue to be indicated in the third stage; that is, for a certain period during the progress of absorption. When these measures have been carried to a sufficient extent, and the amount of liquid is considerably reduced, the patient is to be considered as convalescent. During convalescence the chief object is the re-establishment of the health of the patient. This is to be done by tonic remedies, a nutritious diet, and other hygienic means. Solid food, embracing a fair proportion of meat, may be allowed whenever taken with relish and digested. Alimentation is important; and if the appetite be small and the digestion weak, remedies to improve both are indicated. No apprehension need be felt that a diet as nutritious as can be taken without inconvenience will do any harm; on the contrary, the more nutritious the food which can be taken and digested, the more rapid the convalescence. Wine, spirit, or beer in small quantities, taken with meals, will expedite recovery. Going out of doors and gentle exercise are to be encouraged. The patient will gain more rapidly as regards appetite, digestion, and strength if not confined too long within doors, and with proper precautions no risk of harm is incurred. The removal of the liquid which remains, the absortion of fibrin, and the development of new tissue leading to adhesion will go on rapidly in proportion to the rapidity with which improvement in the general condition takes place.

In the foregoing account of the treatment of simple acute pleuritis it is assumed that, exclusive of a small number of cases in which this disease destroys life either by asthenia or apnœa, its course is toward convalescence without becoming chronic. This is the rule, to which there are exceptions. If the disease become chronic, the measures of treatment indicated are those which will be considered in connection with chronic pleuritis.

The prevention of acute pleuritis hardly claims consideration. The abrupt and rapid development of the disease precludes the employment of prophylactic measures, and there are no premonitions which warrant our anticipation of its occurrence.

Marked dulness on percussion over the affected side, especially at the inferior portion, continues for some time after the effusion has disappeared, the symptoms, general and local, denoting recovery, and the vesicular murmur of respiration being heard over the whole of the affected side. This shows that fibrin remains to be absorbed, but it need occasion no apprehensions. After the effusion has disappeared, as shown by vesicular breathing and vocal resonance extending to the base of the chest, considerable dulness remains, and generally continues for several weeks.

CHAPTER III.

VARIETIES OF PLEURITIS.

Chronic Pleuritis: Anatomical Characters; Clinical History; Pathological Character; Causation; Prognosis; Diagnosis; Treatment; Thoracentesis.—Empyema, Pyothorax, or Suppurative Pleuritis: Diagnosis; Prognosis; Treatment.—Pleuritis with Pneumothorax, Pneumo-hydrothorax, Pneumo-pyothorax, or Pneumo-hæmothorax: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Pneumothorax.—Circumscribed Pleuritis.—Hydrothorax.

OF the varieties of pleuritis, the first to be considered is the simple or ordinary chronic form of the disease. Other varieties are suppurative pleuritis or empyema, and pleuritis with pneumothorax. Circumscribed pleuritis will claim some consideration. This chapter will be devoted to these affections, including a brief account of pneumothorax without pleuritis, and of hydrothorax.

Chronic Pleuritis.

Anatomical Characters.—The anatomical characters in simple chronic or sero-fibrinous pleuritis are essentially the same as in the acute form. The pleural cavity contains serum and fibrin in variable relative proportions, but as a rule the quantity of fibrin is less and the accumulation of liquid greater than in acute pleuritis. Effusion frequently takes place to such an extent that the affected side is more or less dilated. The lung is then compressed into a small solid mass, usually situated at the upper and posterior part; the thoracic walls are expanded in every direction; the intercostal spaces are pushed out to a level with the ribs or even beyond this level, and show no depresssion with the act of inspiration; the diaphragm may be depressed, pushing downward the abdominal organs situated in proximity to it—namely, the stomach, spleen, and liver; the lateral pressure may be sufficient to move the heart from its normal situation, and, if the effusion be in the left side, the heart may be carried quite into the space belonging to the right side of the chest.

The dilatation decreases as absorption of the liquid takes place, and contraction of the affected side succeeds. The latter arises from the fact that compressed lung does not readily resume its former volume when the pressure of the liquid is removed, in consequence of the layers of fibrin with which it is more or less invested, and sometimes because pleuritic adhesions have already taken place. Other things being equal, the extent of contraction is in proportion to the amount of the previous dilatation and its duration. Usually, the dimensions of the affected side in every direction are lessened, the shoulder is lowered, and lateral spinal curvature is likely to ensue. In some cases in which the lung expands to a certain point and becomes fixed by permanent adhesions at that point, the removal of the liquid below leads to a deep depression; the chest presents an appearance as if the lower ribs had been crushed inward. The contraction succeeding a large effusion is generally permanent. It becomes less marked after several months or years in some cases, and if the patient be young it may in the progress of time nearly disappear. In a patient who had chronic pleuritis with very large effusion at the age of seventeen years, followed by great contraction, I found, ten years afterward, the two sides presenting only a slight disparity. The formation of new connective tissue in the shape of fibrous thickening of the pleural membrane and of bands of adhesion between the pleural surfaces is more extensive after chronic pleuritis than after the acute form.

Clinical History.—Chronic pleuritis, in the great majority of cases, is a subacute affection from the first. Occasionally it follows the acute form of the disease. The chronic is a more frequent affection than the acute—a fact which shows that the latter does not usually precede the former, as has been stated by some authors. Of 35 recorded cases which I analyzed with reference to this point many years ago, the inflammation was subacute from the first in 29. The left side is affected oftener than the right, as in acute pleuritis. Of 42 cases, the left side was affected in 23. Of 78 cases analyzed by Blakiston, the left side was affected in 58.

This variety of pleuritis is often developed imperceptibly, and, as far as the symptoms are concerned, it may be one of the most insidious and latent of diseases. It is often overlooked by those who do not employ the physical methods of examination. Its development is not, as in the vast majority of cases of acute pleuritis, characterized by pain. Pain is often either wanting or so slight as scarcely to attract attention. In some cases, however, the pain is moderate or considerable, but falling much short of that intensity which belongs to the acute affection. Tenderness over the chest is slight or moderate, and may be wanting.

Cough and expectoration are frequently wanting, and are rarely prominent symptoms. The cough, if present, is dry, short, hacking, and the matter of expectoration consists of mucus, transparent or slightly opaque. If there be considerable expectoration and the matter expectorated be muco-purulent, either bronchitis coexists or the pleuritis is associated with phthisis.

The frequency of the respirations is increased, assuming the pleuritis to be accompanied by a large effusion of liquid. The increase, however, may be moderate while the patient is at rest, but it becomes marked on exercise. Deficiency of breath is also shown by inability to speak with a sustained expiration, the speech being interrupted at short intervals for a fresh inspiration. Dyspnœa is rarely present except on exercise. Here, as in acute pleuritis, the embarrassment of breathing depends much on the rapidity with which the effusion takes place. The suppression of the respiratory function of an entire lung in this affection, if it be slowly induced, does not usually occasion lividity of the face or prolabia.

The pulse is generally more or less accelerated, ranging from 80 to 120 per minute. It is small and compressible. There is, however, considerable variation in different cases as regards these characters of the pulse. The surface of the body frequently presents more or less capillary congestion. Sweating is apt to occur, especially during the night, and may be profuse without having been preceded by a chill or febrile movement. Irregular, slight chills, however, are not unusual, leading sometimes to the error of supposing the disease to be malarial.

The appetite is sometimes notably impaired, but sometimes it is preserved and the digestion continues good. Diarrhœa occurs in some cases. Generally, the countenance is pallid, denoting anæmia, but this is not true of all cases, and a healthy aspect is sometimes maintained. Emaciation is not usually marked, except when the disease is associated with phthisis. The strength is often retained in a remarkable degree. Patients usually do not take to the bed, and they may be able to be up and out of doors even with one side of the chest filled with liquid. I have known repeatedly persons in this condition to continue pursuits which required active muscular exertion.

This sketch of the symptomatology applies, of course, to cases of chronic pleuritis not associated with other affections furnishing symptoms which are combined with those belonging to the disease under consideration. It may be associated with phthisis. The coexistence of the latter affection renders cough more prominent, and gives rise to a more or less abundant expectoration. Emaciation and muscular feebleness are also more marked. Pericarditis coexists in a small proportion of cases. With this complication the circulation is more disturbed, there is more suffering from dyspnœa, and the symptoms in general denote greater gravity of disease. The affection may be developed in the course of either acute or chronic disease of the kidneys; under these circumstances general dropsy may be present, emaciation and feebleness are greater, and the tendency to asthenia is more apparent. In like manner, other affections which may be associated modify and add to the symptoms pertaining to the pleuritic inflammation.

The duration is variable, but the career rarely ends until after several weeks, and generally it extends over several months. As regards stages, when acute pleuritis has not preceded, cases do not come under observation until more or less effusion has taken place, and, it may be, not until the pleural sac is filled with liquid. It suffices to consider the career of the disease as consisting of two stages—one embracing the period during which the liquid either is accumulating or remains stationary, and the other embracing the period during which absorption of the liquid is going on. After absorption has made a certain amount of progress the patient may be regarded as convalescent.

Pathological Character.—Simple chronic sero-fibrinous pleuritis is a subacute inflammation, differing from the acute form of the disease only in a lesser degree of the inflammatory condition. The inflammation has no special character, but is of a low grade of intensity, with a tendency to continuance for an indefinite period.

Causation.—Chronic pleuritis may be produced traumatically. It may sometimes be due to cold. It occurs occasionally during the course of affections of the kidneys. In many cases it proceeds from internal causes which are not understood, and it is then said to be spontaneous. In a small proportion of cases the chronic follows the acute disease. In much the larger proportion the inflammation is subacute from the first.

An important point of inquiry relates to its pathological relations to phthisis. According to some authors, it is generally consecutive to and dependent upon a phthisical affection. There has been no evidence of this in a large majority of the cases which have come under my observation. Of 42 cases analyzed several years ago, the evidence of coexisting phthisis was present in only 3, and of a large number of cases which I have since observed I am sure that the two affections have been associated in but a small proportion. As, however, the occurrence of pleuritic effusion retards and contributes to the arrest of the progress of phthisis, the association of the two affections may sometimes exist without being apparent.

Chronic as well as acute pleuritis occurs rarely in infancy. It is not of infrequent occurrence in childhood and early life, but it is most frequent between puberty and middle life. It is of rare occurrence in the aged.

Prognosis.—Simple chronic pleuritis, not associated with phthisis or complicated with any serious affection, and occurring in a healthy subject, tends to recovery. A fatal result may take place in consequence of a rapid and large accumulation of liquid, but instances of this are exceedingly rare. If death occur, it is generally from associated affections or complications.

Recovery takes place, leaving the affected side more or less contracted, and sometimes the contraction is so marked as, in connection with the lateral curvature of the spine which it occasions, to produce a considerable and permanent deformity. In general, however, the contraction which follows is attended with little or no inconvenience, and is not observed unless the chest be denuded and the two sides compared.

A permanent dislocation of the heart to the right side of the sternum is sometimes an effect of the removal of a large collection of liquid from the right pleural sac; and in some cases of pleuritis with large effusion in the left side the heart, which was dislocated by the lateral pressure of the liquid, is found on the right side of the sternum after recovery from the pleuritis, adhesions having taken place which prevent it from returning to the left side after the liquid is removed.

Phthisis becomes manifest sooner or later after recovery in a certain proportion of cases. In some of the cases in which this disease appears to be a sequel it may have existed prior to the pleuritis and have undergone arrest. The proportion of cases in which phthisis follows is not sufficiently large to show a predisposition to this disease derived from the pleuritis. The opinion, frequently and perhaps commonly entertained, that chronic pleuritis, when it does not supervene on a phthisical affection, predisposes strongly to the occurrence of the latter, I believe to be incorrect. This belief is based on my own experience. It is sustained by statistics communicated many years since by Blakiston.[1] Of 53 cases observed by that author, the patients remaining under observation for several years after recovery, not one became affected with pulmonary tuberculosis. This result is striking, for it might be expected that out of so large a number of cases of any disease, occurring, as does pleuritis, for the most part at an early age, a certain proportion would be likely in the course of several years to become phthisical. In estimating the influence of this or any disease in determining the subsequent development of phthisis, the liability to the latter, irrespective of antecedent affections, is of course to be taken into account.

The idea that chronic pleuritis affecting the right side of the chest denotes phthisis has been disproved by statistics reported by Bowditch.[2]

The mortality from chronic pleuritis, even without regard to associated affections and complications, is not great. The rate of mortality probably does not exceed that which several years ago I deduced from my recorded cases—namely, 17 per cent.

Recovery is understood to imply the removal, by absorption, of the effusion. The disease, however, may terminate in another way, leaving no local symptoms, the patient regaining good health. This consists in the affected side of the chest remaining permanently filled with liquid. The following case, illustrative of this termination, came under my observation in the Charity Hospital of New Orleans in 1861:

The patient, a man aged about forty-five, stated that he had been ill and confined to the bed for four days, having previously been in good health. In answer to inquiries suggested by the marks of cupping on the left side, he stated that he had had pleurisy several years before. The left side was dilated, everywhere flat on percussion, and the respiratory murmur suppressed. Death occurred on the third day after his admission, and my notes do not contain data showing the probable cause of death. The man presented a robust appearance, and declared that he was quite well when seized with illness four days before his admission. His habits were intemperate. On examination of the chest after death, the left pleural sac was found to be so resisting that it was removed entire and brought to the college. The sac was then opened with some difficulty, and a large amount of turbid liquid, estimated to be two gallons, removed. After the liquid had escaped, the pleural sac was sufficiently rigid to retain the shape which it had before being opened. A calcified fibrous tissue, from one-eighth to one-quarter of an inch in thickness, lined the entire pleura. The parietal portion of the membrane could in some places be stripped from this calcified tissue. Calcareous matter was abundantly deposited in the latter. The lung was compressed into a solid, small mass which resisted efforts at insufflation.

Another case, illustrative of a similar permanency of effusion, came under my observation in private practice. A lawyer, aged about thirty, had an attack of pleuritis, which confined him for some time to the bed, nine years before my first examination, in 1861. Five years afterward he was in excellent health, being entirely free from any pulmonary symptoms except deficiency of breath on exercise. Flatness on percussion existed over the whole of the left side, with suppression of respiratory sound and vocal resonance, except at the summit, where bronchial respiration and bronchophony existed. These signs were considered as denoting liquid in sufficient quantity to fill the pleural sac and compress the lung into a solid mass. The patient stated that he had been repeatedly examined since the attack of pleuritis nine years before, and there had been no change in the condition of the chest since his apparent recovery from that disease.

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[1] *Practical Observations on Certain Diseases of the Chest, etc.*, Amer. ed., 1848.

[2] *American Journal of the Medical Sciences*, January, 1863.

DIAGNOSIS.—Owing to the latency of chronic pleuritis as regards the subjective symptoms in many cases, the existence of intrathoracic disease is often overlooked by those who do not employ physical exploration. Of the cases which have come under my observation, in not a few the patients have been supposed to have either dyspepsia, irregular malarial fever, hepatic trouble, or some ailment not connected with the chest. If the symptoms point to the existence of intrathoracic disease, patients are often supposed to be phthisical, and the cough, deficiency of breath, night perspirations, etc. rationally favor such a conclusion. A positive diagnosis can hardly be made without the aid of physical signs.

The signs on which, taken in connection with the history and symptoms, the diagnosis may be based are those denoting pleuritic effusion. The signs of effusion are the same in chronic as in acute pleuritis, and need not be again stated; but in chronic more frequently than in acute pleuritis the accumulation of liquid is so large as to occasion more or less dilatation of the affected side, and then are superadded signs obtained by inspection and palpation, notice of which was deferred in treating of the acute disease.

Dilatation of the affected side by liquid is apparent to the eye, and may be ascertained by mensuration either with graduated tape or calipers. The intercostal spaces are pushed out to a level with the ribs, and may be bulging. The side is motionless or nearly so in the acts of respiration, the movements of the opposite side being exaggerated. By applying the fingers within the intercostal spaces, and making peripheral and diametrical percussion, a sense of fluctuation is sometimes obtained. The heart is moved from its normal situation, as ascertained either by palpation or auscultation. If the effusion be in the left side, the apex-beat and the maximum intensity of the heart-sounds are on the right side of the sternum, and they are carried toward the left lateral portion of the chest if the effusion be in the right side. The diaphragm is depressed, pushing downward the abdominal organs in contact with it. The addition of these signs renders the diagnosis of pleuritic effusion still more complete and positive than the signs which denote the presence of liquid without dilatation.

The presence of liquid is demonstrated by the introduction of a small exploring trocar, previously disinfected. A hypodermic syringe, the needle being sufficiently strong to obviate danger of its being broken off and remaining in the chest, answers for the exploratory puncture, or a small aspirating needle may be used. This should be resorted to in all cases, both of acute and of the different varieties of chronic pleuritis, in order to ascertain the character as well as the presence of liquid. With as well as without this exploration there is one liability to error in diagnosis—namely, carcinomatous or sarcomatous infiltration of an entire lung, which may cause an enlargement of its volume, and, a certain amount of pleuritic effusion coexisting, the signs may seem to denote simply chronic pleuritis. An exploratory puncture demonstrates the presence of liquid. The diagnosis can then be settled only by aspiration. Removing the liquid, the affected side remains enlarged; the needle or trocar even passes into a solid mass, as shown by its remaining fixed; that is, the point not movable while in the chest.

A sanguinolent effusion, if scorbutus be excluded, renders the coexistence of phthisis probable.

During the stage of absorption the signs show the progressive diminution of liquid until the quantity is no longer sufficient to occasion dilatation, and from this point the continued decrease in quantity is ascertained as in acute pleuritis. The contraction of the affected side which follows chronic pleuritis is more marked than that succeeding the acute affection.

TREATMENT.—The main objects of treatment in simple chronic pleuritis are twofold—namely, *first*, to effect the removal of the effused liquid; and *second*, to develop and sustain the powers of the system. For the first object the measures which may be employed are the same as in the second and the third stage of acute pleuritis, consisting of the milder hydragogues, diuretics, small blisters applied in succession over different portions of the affected side, and the use of iodine internally and externally. Bloodletting, general or local, is never indicated in chronic pleuritis.

In pursuing measures with reference to the first object, the second is not to be overlooked or neglected. Cases are often injudiciously treated in consequence of the attention being too much engrossed with the measures to promote absorption; and these measures will do harm if continued too long or pushed too far. After a certain amount of effect has been produced, if these measures cease to be further effective, they should be discontinued, at least for a time; nor should they be persisted in if they fail to have any effect. Harm is produced by their injudicious employment in proportion as they debilitate.

The second object calls for tonic remedies, nutritious alimentation, the moderate use of alcoholic stimulants, and gentle exercise out of doors. These measures are always important, and they are pre-eminently so if from associated affections, complications, or the general condition of the patient the powers of the system be notably lowered. I have repeatedly seen a marked change for the better occur at once, followed by progressive improvement in all respects, on the discontinuance of measures having reference to the absorption of liquid, and the substitution of measures for building up the system. Hygienic measures—namely, nutritious diet and exercise in the open air under proper restrictions—are especially important.

A mode of effecting the first object—namely, the removal of the liquid—requires distinct consideration. Reference is now had to the operation of *paracentesis* or *thoracentesis*. The propriety of puncturing the chest when the accumulation of liquid is sufficient to place the patient in imminent danger is unquestionable. But it is a question whether it be not advisable to puncture although the patient be not in great distress or danger. It is clearly an object to get rid of the liquid, and the point is whether it be better to resort to the direct mode—that is, to puncture and withdraw it—or to endeavor to effect the object indirectly by purgatives, diuretics, etc.

Formerly, this operation was performed only as a *dernier ressort*, when little was to be expected from any measure. It was deferred as long as possible, sometimes on account of doubt as to the diagnosis, and because the perforation and introduction of air involves danger of an increase of the inflammation. A considerable opening was necessary in order to give free exit to the liquid, and it was difficult to prevent air from entering the pleural cavity. Objection to the operation on the score of diagnosis is now removed by our present knowledge of physical signs. Moreover, the operation was divested of all severity, and the liability to the introduction of air provided against, by the application of suction in 1850. The introduction of air, aside from the entrance of organisms giving rise to putrefactive changes, is objectionable, because its presence is an obstacle to the full expansion of the lung after the liquid is removed, and with the air enter germs which occasion putrefactive and suppurative processes. Its introduction is prevented by the employment of a suction force in withdrawing the liquid. The operation is rendered trivial, because with suction a small exploring trocar suffices to make the puncture, which causes very little pain and closes as soon as the canula is removed.

The credit of the introduction of the practice of withdrawing the liquid through a small canula by means of suction—or, as it is now called, aspiration—is due to Bowditch. He operated in this way 250 times on 154 persons in the course of twenty years. In no instance was any permanent injury referable to the operation. It was in some cases repeated several times during the progress of the disease. In a considerable proportion of cases recovery appeared to be attributable to the operation. Immediate and great relief was obtained in cases which ultimately proved fatal. Bowditch has been led by his experience to operate in all cases in which the quantity of liquid induces either permanent or occasional dyspnœa of a severe character, and in all cases in which the pleural cavity is filled, if, after a reasonable amount of general treatment, the liquid do not diminish.

My own experience in the practice of the operation has been considerable, and I can testify in behalf of its innocuousness, the relief which it affords, and its value as a means of rescuing in some instances patients from a condition of imminent danger to life. The conclusions of Bowditch, as just stated, are not only sustained by his large experience, and at the present time by the experience of many others, but they are consonant with common sense. If, by an operation trivial with respect to pain or any evil effects, and easily performed, the effusion may be withdrawn at pleasure, this mode is certainly to be preferred to measures which are indirect, not very reliable, requiring considerable time, and producing more or less disturbance and debility of the system. An important advantage of the operation is, the liquid being removed before the lung has been subjected to long pressure and has become adherent by dense layers of fibrin or perhaps organized tissue, the expansion takes place more fully and is followed by less deformity of the chest from contraction of the affected side. It is a noteworthy fact that directly after the operation the temperature declines and may fall to the normal standard.

Thoracentesis by means of a small trocar with the aid of suction, the puncture made at a convenient point either behind, laterally, or in front, is admissible whenever the pleural cavity remains filled with liquid after a brief trial of the measures designed to promote absorption; and the operation should not be delayed whenever the accumulation of liquid is sufficient to involve danger or distressing dyspnœa.

For many years, in place of the suction-pump employed by Bowditch, I have used a simpler apparatus—namely, a small trocar and a canula fitted to screw upon the flexible suction-tube of Davidson's syringe. The canula should be provided with a stopcock. The trocar and canula being introduced into the chest, the trocar is withdrawn and the canula attached to the syringe; the liquid is then removed by means of the expansion of the India-rubber bulb after its compression with the hand. I have used this apparatus in many cases with satisfactory results. It has also been used with success by many others. After a certain quantity of liquid has been withdrawn, the patient often is seized with cough and experiences a distressing sense of constriction in the chest. These symptoms probably arise from the atmospheric pressure upon the thoracic wall and from the disruption of the pleural surfaces, united by fibrin, by the expansion of the compressed lung. They may also be in part due to the suction force exerted laterally upon the lung of the healthy side. They may be delayed, and sometimes avoided, by withdrawing the liquid very slowly, thus allowing time for the expansion of the compressed lung and the gradual accommodation of the parts to fill the vacuum occasioned by the removal of the liquid. When these symptoms occur the further removal of liquid should be postponed for another operation. As regards the quantity removed at one operation, much will be gained by following the injunction to withdraw the liquid slowly, especially when the patient begins to cough and complain of constriction. The advantage of my apparatus over the suction-pump is its greater simplicity, the ease with which it is managed, and a less liability to get out of order.

More recently an instrument for aspiration—or aspirator, as it is called—constructed on a different plan, was introduced by Dieulafoy. The peculiarity of Dieulafoy's aspirator consists in first producing a vacuum in a glass receiver, and then opening a communication between the latter and a canula or hollow needle introduced into the chest. The objections to the instrument are—first, being complicated, it is likely to get out of order; and second, it is expensive as compared with the suction-pump, and especially with that devised by me. A little appendage has been added to the canula to be attached to Davidson's syringe, so that it may be in an instant connected with the latter. It is easy to secure more expansive force than is obtained by the ordinary syringe used for enemata, by having an instrument made especially for aspiration, with a larger and thicker India-rubber bulb.

A much simpler instrument than the aspirator of Dieulafoy, on the same principle, is a glass bottle from which, by an arrangement for that purpose, the air is withdrawn by means of a small syringe, and the communication with the canula within the chest then opened through a tube connecting the latter with the bottle. This kind of aspirator is as efficient as the more costly one of Dieulafoy.

Dieulafoy's aspirators are provided with small trocars and hollow needles. The latter should never be used in aspirating the chest. The sharp needle-points are liable to tear the lung with which it may come in contact after a certain quantity of liquid has been withdrawn, giving rise to pneumothorax.

Failure in aspiration may be caused by the trocar carrying before it a portion of false membrane which prevents the liquid from being reached. With a view to obviate this difficulty, the trocar should be pushed with a certain degree of quickness and force into the chest. Another difficulty is the plugging of the end of the canula with floating pieces of fibrin. This obstruction may generally be removed by forcing some of the fluid back again through the canula into the chest.

Instances have been reported of grave symptoms, and even death, resulting from aspiration. They have been most frequently reported by French writers, who affixed to the condition the name *expectoration séro-albumineuse*, derived from one of the most prominent symptoms. The condition is characterized by dyspnœa, cough, with expectoration of frothy, watery, and sometimes blood-stained liquid, and the symptoms of collapse. A probable explanation is the hyperæmia which follows removal of pressure from the capillaries of the compressed lung, these capillaries being more or less impaired in their nutrition by long-continued pressure. Recovery is common, following the administration of heart-stimulants. I believe that the risk is almost *nil*, if the fluid be withdrawn slowly and aspiration be suspended as soon as cough or dyspnœa is produced. But even if this statement be not admitted, the risk is too small to constitute a serious objection to the measure if it be indicated. I may add that during the past twenty years aspiration has been practised almost weekly in the wards of Bellevue Hospital, and I have not heard of a single instance in which any serious consequences have resulted from it. Only one such instance has come within my cognizance in private practice. In this instance an alarming dyspnœa followed the removal of a large quantity of pus. This effect was temporary, and the case ended in recovery. It is probable that the effect in that instance was attributable to a too rapid withdrawal of the liquid. I had previously withdrawn a large quantity of pus in this case without any unpleasant symptoms.

Empyema—Pyothorax—Suppurative Pleuritis.

These names denote a form of pleuritis characterized by the accumulation of a purulent liquid in the pleural cavity. The term empyema expresses only the existence of pus without indicating its situation. Pyothorax is a better name, but this does not express the existence of pleuritic inflammation. Suppurative pleuritis is a simple title suggested as more fully expressive of the affection.

Suppurative pleuritis may be either acute or subacute at the beginning, but it becomes chronic. It is a more severe form of inflammation than sero-fibrinous pleuritis. At the beginning the inflammatory products may be the same in both diseases, but in suppurative pleuritis the emigration of white blood-corpuscles is greater, causing the fluid in the pleural cavity to acquire a purulent character. Sometimes the fluid is thick, creamy pus, but usually it is thin and sero-purulent or fibrino-purulent. There is less fibrin in the exudation than in sero-fibrinous pleuritis, suppurative inflammation checking the production of fibrin. The pleural membrane itself is more severely affected than in acute pleuritis. It is thickened and converted into a kind of granulation-tissue, containing leucocytes, new connective-tissue cells, and new blood-vessels. The inflammation may even extend to the subpleural tissues.

Suppurative pleuritis, at least in the great majority of cases, is not to be regarded as an intense variety of ordinary sero-fibrinous pleuritis, but as an inflammation depending upon some special cause. The micro-organisms which have been found in suppurative inflammations elsewhere have been observed in this affection. There are various micro-organisms capable of inducing suppuration, so that the specific infective agent is not the same in all cases. It is not probable that sero-fibrinous pleuritis can eventuate in suppurative pleuritis without the addition of some new special agent of infection. The inflammation has generally a suppurative character from the first, but in some cases the emigration of white blood-corpuscles is for some time insufficient to render the exudation distinctly purulent.

In rare instances the walls of the pleural cavity become gangrenous and the cavity contains a brownish, fetid fluid. The pleuritis is then designated as *gangrenous* or *putrid*. Putrid pleuritis is usually secondary to gangrene of the lungs, but it may occur as an independent affection. The putrefaction is the result of the invasion of the pleura with the bacteria of decomposition. In putrid pleuritis are found bacilli as well as micrococci.

Sometimes, as a result of empyema, but more frequently in connection with putrid pleuritis, the ribs become carious and the parietal pleura is easily detached from them.

There are no distinctive points pertaining to the symptomatology of suppurative pleuritis, and it is not necessary, therefore, to consider under a distinct head its clinical history.

As regards causation, suppurative pleuritis may be produced traumatically. It is sometimes secondary to lobar pneumonia. It may be developed in pyæmia or as a complication of other acute infectious diseases, and it may be secondary to abscesses in adjoining parts, as in the lungs, the chest-walls, the liver, or the peritoneum. In many cases it occurs as an idiopathic or primary disease. As already stated, the special cause is probably, in most if not in all cases, infection with a special form of micro-organism, usually the micrococci of suppuration.

DIAGNOSIS.—The signs as well as symptoms in suppurative pleuritis are not distinctive of this disease. They represent the presence of more or less liquid in the pleural cavity, and they are essentially the same when the liquid is purulent as when it is sero-fibrinous. The point of inquiry in regard to diagnosis is whether there be any means of determining that the liquid is purulent. This is an important point with reference to prognosis and treatment. The occurrence of chills, more fever, hectic paroxysms, etc. is not reliable in this differential diagnosis. As a rule, however, the axillary temperature is higher than in simple pleuritis. The symptoms and signs, in fact, exclusive of spontaneous perforation of the thoracic walls, will hardly warrant a positive diagnosis. But the purulent character of the liquid may be inferred with considerable confidence if the quantity progressively continue to increase notwithstanding the employment of measures to promote absorption. A serous effusion is generally either diminished or retarded by the use of hydragogues, diuretics, etc., but these measures are not likely to exert any influence upon a purulent liquid.

A positive diagnosis, however, may be easily made by an exploratory puncture. A small trocar or the needle attached to a hypodermic syringe may be introduced into the chest and a little of the liquid withdrawn, when the gross and microscopical characters of the specimen thus obtained will at once settle the question. This procedure is warrantable as a means of diagnosis in view of the bearing on the prognosis and treatment. Indeed, the operation is so slight as to be in no wise objectionable. The needle should be sufficiently strong not to incur the risk of fracture, leaving a portion within the chest. I have known of several instances in which this accident has occurred.

Spontaneous perforation of the walls of the chest occurs, after a time, in cases of suppurative pleuritis. When this occurs, before ulceration of the integument ensues, a soft fluctuating tumor makes its appearance over or near the point of perforation. The appearance of such a tumor, developed rapidly without having been preceded by inflammation exterior to the thoracic walls, is at once significant of this variety of pleuritis. The size of the tumor may be observed to vary with forcible acts of inspiration and expiration. The action of the heart upon the liquid within the chest may communicate a pulsation to the tumor, and this may at first lead to a suspicion of aneurism; but an examination of the chest, by furnishing the evidence of the pleural sac being filled with liquid, will show the character of the affection. If the tumor be not opened, inflammation of the integument from distension and ulceration supervene, and the pus makes its escape from the tumor and the pleural cavity. "Paracentesis from necessity," as it is called, has then taken place. This result is almost exclusively due to suppurative pleuritis, but I have known an instance in which spontaneous perforation took place when the liquid did not have the gross characters of pus.

In a smaller proportion of cases a spontaneous evacuation occurs in another direction. Perforation of the lung takes place, and the pus finds its way into the air-passages. The occurrence of this event is marked by a sudden and copious purulent expectoration. This expectoration continues for an indefinite period. With rare exceptions, air enters the pleural cavity, and the disease is converted into pneumo-pyothorax, which will presently be considered under a separate heading. Perforation successively of both the walls of the chest and of the lung sometimes occurs.

PROGNOSIS.—Suppurative inflammation of the pleura is a more serious disease than simple pleuritis, whether acute or chronic. Inasmuch as the pus cannot be removed by absorption, but must be evacuated either spontaneously or by puncture, the condition is analogous to that of a large abscess which, after opening, continues to discharge through a fistulous orifice. After the purulent contents of the pleural cavity are evacuated, the membrane continues to furnish pus, and thus the discharge continues indefinitely. Emaciation, debility, hectic paroxysms, etc. result from long-continued suppuration, and death takes place by slow asthenia. An unfavorable termination may be due, as in certain cases of simple pleuritis, to the coexistence of phthisis, pericarditis, or some other serious affection. In the case of a patient ten years of age which came under my observation sudden death followed perforation of the lung, the discharge of pus taking place rapidly and causing suffocation by filling the bronchial tubes.

Recovery from suppurative pleuritis, however, is by no means infrequent, and may reasonably be hoped for if the patient have a good constitution and the affection be uncomplicated. The prognosis is more favorable in children than in adults. The purulent discharge continues for weeks, months, and perhaps years, but progressively diminishes; pleuritic adhesions take place, the affection becoming more and more circumscribed, until at length they become universal, and the recovery is completed, leaving the affected side considerably or greatly contracted.

TREATMENT.—In the treatment of suppurative pleuritis no reliance is to be placed on measures to cause absorption of the purulent liquid. If the pleural cavity contain pus in considerable quantity, it will not be absorbed; but if life be sufficiently prolonged and thoracentesis be not resorted to, it will sooner or later make its way either into the air-passages or through the thoracic walls. Hydragogues, diuretics, blisters, etc. will not only prove unavailing, but they do harm by impairing the powers of the system. Thoracentesis should be performed as soon as the purulent character of the liquid is ascertained. Aspiration may be employed and repeated, but in many cases, owing to the reaccumulation of pus, this mode of performing thoracentesis will prove unsatisfactory. An opening should be made at the bottom of the pleural sac, allowing the pus to escape freely. Air will of course pass into the chest, and, since this is unavoidable, its ingress and egress should be unobstructed. A small orifice, not sufficient for the free escape of the pus and allowing air to be pent up within the pleural cavity, is injudicious. Having made a free opening, allowing all the pus to escape, the orifice should be kept open by the introduction of an oakum tent. This is preferable to the insertion of a drainage-tube, which is a foreign body within the pleural cavity, and therefore objectionable. The pleural cavity should be daily cleansed by the injection of tepid water to which a very small quantity of carbolic acid (not more than 1 per cent.) is added. For the injection a double-tubed catheter and Davidson's syringe suffice. If this be not done, the pus within the chest is liable to become fetid from the introduction of putrefactive germs, and septic fever is thereby induced. I have repeatedly demonstrated these effects by omitting to wash out the pleural cavity for a day or two. Billroth has enjoined caution against danger from injecting a cold liquid into the pleural cavity. It may cause death by shock.[1] In cases of putrid or gangrenous pleuritis antiseptic injections are especially important. If carbolic acid be used, the quantity should be extremely small, in order to avoid toxical effects. Other antiseptic solutions which may be used are those of thymol or salicylic acid. I should add that I have known a complete withdrawal of the pus by a single aspiration to prove sufficient, but the probability of this result is small. Aspiration repeatedly performed has succeeded in several cases under my observation, but these cases are exceptional. There is no objection to giving aspiration a fair trial before resorting to a free open-

[1] Vide *Am. Journ. of Med. Sciences*, April, 1882.

ing. Indeed, this is generally advisable. In children, simple aspiration not infrequently effects a cure.

Of late years there has been considerable discussion as to the best method of operative procedure in case of suppurative pleuritis. There is agreement as to the necessity of risking a free opening into the chest, but many are opposed to subsequently washing out the pleural cavity. The resection of one, two, and even six or eight ribs in order to favor sinking in of the chest-wall and closure of the pleural cavity has been recommended. This procedure seems to me highly objectionable. I have known of two instances in which death was fairly attributable to this operation. Trephining the ribs is less objectionable, and may be advisable in some cases.[1]

If spontaneous perforation of the chest-wall take place, as denoted by the development of a fluctuating tumor, the latter may be at once opened; but if the perforation be at a point so far above the bottom of the sac that a portion only of the pus is evacuated, more or less remaining constantly below the point of perforation, a counter-opening may be requisite. Aspiration, however, may be fairly tried before resorting to this measure and before making an incision into the subcutaneous tumor.

If perforation of the lung take place aspiration should be employed, and if this do not succeed a free incision into the chest-wall should be made, provided the pus be not fully discharged by expectoration. The fact that only a portion of the pus within the pleural cavity is expelled through the pulmonary orifice is readily ascertained by means of the physical signs. In most instances perforation of the lung converts the disease into pneumo-pyothorax, but sometimes pus passes through the opening into the bronchial tubes without the entrance of air into the pleural space.

To support the powers of the system is an object of still greater importance in the treatment of suppurative than of simple chronic pleuritis. The importance is greater because there is more danger of death by asthenia, and in favorable cases the duration of the affection is longer. The measures to be employed for this object are the same in both forms of the disease.

Pleuritis with Pneumothorax—Pneumo-hydrothorax—Pneumo-pyothorax—Pneumo-hæmothorax.

The term pneumo-hydrothorax denotes the presence of air or gas and liquid in the pleural cavity. As the liquid, however, is either sero-fibrinous or purulent, hydrothorax, which signifies a purely serous liquid, is not strictly appropriate; the liquid is due to inflammation and the affection is a variety of pleuritis. It would be more correct to say pleuritis with pneumothorax. Pneumothorax denotes the presence of air without liquid. The latter affection is rare, but it occurs and claims separate notice.

ANATOMICAL CHARACTERS.—Gas escapes with more or less force after the knife penetrates the chest in making the post-mortem examination. If the patient have survived sufficiently long after the perforation, the pleural cavity usually contains an inflammatory exudation. This exudation is in most cases purulent (pyo-pneumothorax); and in rare instances it is sero-fibrinous (sero-pneumothorax. Sometimes the perforation is attended by hemorrhage into the pleural cavity (hæmato-pneumothorax). If the disease be associated with pulmonary gangrene, and exceptionally in other cases, the air and liquid are fetid in consequence of putrefaction.

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[1] Vide article by Dabney, in *American Journ. of Med. Sciences*, October, 1882; abstract of König's treatment in same journal, April, 1881; article by T. G. Richardson in *Journal of the American Medical Association*, vol. i. No. 2. Reference to recent literature of the subject may be found in *Virchow and Hirsch's Jahresbericht*, under heading "Pleuritis," 1884 and 1885.

The size of the perforation varies. It is usually small when the affection supervenes on phthisis, but in cases of gangrene it may be quite large. Usually, the site of the perforation can be demonstrated by inflating the lungs through the trachea. Sometimes, however, it is impossible to find the perforation.

The perforation may be found open or closed, or in the form of a valve-like structure which allows air to enter, but not to escape from, the pleural cavity. The tension of the enclosed air is usually greatest when the perforation is valvular. The perforation may be closed by a fibrinous layer. The lung on the affected side is compressed into a solid mass if this be not prevented by old adhesions.

CLINICAL HISTORY.—The occurrence of perforation of the lung is usually marked by acute pain denoting the development of pleuritis. The introduction of air and the speedy effusion of liquid give rise generally to notable dyspnœa, with accelerated breathing, accompanied frequently with lividity. The pulse is in most cases rapid and feeble. These symptoms show the supervention of a serious affection of some kind, and point to the chest as its seat.

In some cases the accelerated breathing and dyspnœa continue to be prominent symptoms, the patient suffering greatly, and perhaps being unable to lie down (orthopnœa), the affection ending fatally, usually under these circumstances after the lapse of a few days, and it may end fatally in the course of a few hours. But in other cases the violence of the symptoms subsides after a short time, and the dyspnœa may be moderate or wanting, save on exercise. In some instances even considerable exercise is practicable. As regards strength and the general condition there is much variation in different cases. In the majority of cases patients are confined to the room and bed, but sometimes they are able to be out of doors, preserving appetite and not presenting a notably morbid aspect. Much will of course depend on the condition as regards the affections of which this is a complication.

As an exception to the rule, pleuritis with pneumothorax from perforation is developed gradually and imperceptibly. It may remain for a greater or less period quite latent. In some instances the accumulation of liquid becomes large, the air disappears, and the affection is converted into simple pleuritis with effusion. In several instances of this kind which have fallen under my observation thoracentesis was resorted to, and the communication between the air-passages and pleural cavity through the perforation was resumed after the liquid was withdrawn.

CAUSATION.—It is estimated that from 80 to 90 per cent. of all cases of pneumothorax are due to the rupture of a phthisical cavity into the pleural cavity. Although this rupture usually occurs in an advanced stage of phthisis, it may take place before any suspicion of this disease exists. The perforation is more frequently at the middle or inferior portion of the lung than at the summit. Gangrene, abscesses, and hemorrhages in the lungs are rare causes of pneumothorax. It is sometimes due to the bursting of an emphysematous bleb situated just beneath the pleura. As in such cases the air may not contain any inflammatory irritant, the pneumothorax may exist without any inflammatory exudation. Bronchiectasis may lead to rupture. Next in frequency to phthisis as a cause of pneumothorax is the ulceration of an empyema into the lung. When an empyema opens spontaneously through the thoracic walls, it may be that no pneumothorax follows, because the opening is sinuous and in some cases acts like a valve. Injuries of the chest which cause laceration of the lung are attended by pneumothorax. The most frequent of these is fracture of the ribs. Simple stab-wounds of the lung may occur without causing pneumothorax. A very small number of cases of pneumothorax are the result of communication between the pleural cavity on the one hand and the œsophagus, the stomach, or the intestine on the other hand. Ulcerative and cancerous diseases of these organs may lead to this abnormal communication.

It is held by some authorities that gas may be spontaneously generated in the pleural cavity by the decomposition of inflammatory exudations or of blood. This view, however, is not proven. The failure to find a perforation cannot in itself be considered evidence of the spontaneous generation of gas.

DIAGNOSIS.—When this affection is developed with marked symptoms in the course of phthisis, its existence should certainly be at once suspected; but the symptoms are not sufficiently distinctive of this affection, as contrasted with pleuritis and effusion without the presence of air, to warrant a positive differential diagnosis. Moreover, as just stated, the affection is sometimes latent, the symptoms not warranting even the diagnosis of pleuritis. A positive diagnosis, in short, must be based on physical signs, and these are easily obtained and very explicit.

The presence of air in the pleural cavity renders the resonance on percussion purely tympanitic. If the quantity of liquid be small or moderate, tympanitic resonance may extend over the whole of the affected side, being propagated from the level to the bottom of the liquid. If the liquid be large or considerable in quantity, dulness or flatness will be found at the base and extending upward (the patient sitting) to a certain height, tympanitic resonance existing above. Under these circumstances the relation of resonance and dulness or flatness will always be found to vary with the changed position of the body of the patient. The tympanitic resonance sometimes has an amphoric character.

Respiratory sound is sometimes wanting over the whole of the affected side. Over the compressed lung bronchial respiration may often be found. The ordinary cavernous respiration is sometimes heard, but in most cases amphoric respiration and amphoric voice or echo are present, either limited or diffused over the whole of the affected side, and metallic tinkling is generally conjoined. The presence of these striking and distinctive signs renders the diagnosis easy and positive, taken in connection with other signs.

Succussion almost invariably develops a splashing sound, frequently having the same kind of musical intonation as the respiration, voice, and tinkling sounds. This proof of the presence of air and liquid is positive, and it enables the diagnosis to be made in an instant.

The affected side is frequently dilated, and presents the same appearances as in cases of dilatation from liquid either in simple or suppurative pleuritis.

Leyden has called attention to the rare occurrence of abscesses containing air, situated just below the diaphragm, either on the right or the left side, oftener on the right, between the diaphragm and the upper surface of the liver.[1] The diagnostic features of these abscesses were first recognized by Barlow and Wilks in 1845. Leyden calls the affection pneumothorax subphrenicus. Cossy calls it false pneumothorax. The abscesses result from perforating ulcers of the stomach or duodenum from which gas is derived, circumscribed peritonitis having been produced. The signs of pyo-pneumothorax are present. Leyden diagnosticated the affection in two cases, each connected with perforating ulcer of the stomach. The differential points are—the presence of the respiratory murmur from the clavicle down to the third rib; the extension of the respiratory murmur downward by deep inspiration; and, at first, absence of cough and expectoration. The abscesses may burst eventually into the lungs. A capital point in the diagnosis is the history of the development of the abscess with the signs of peritonitis following symptoms referable to the stomach or intestine.

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[1] *Zeitschrift für klinische Medicin*, Bd. 1, p. 320.

PROGNOSIS.—The prognosis in cases of pleuritis with pneumothorax is always unfavorable. In this statement traumatic cases are excluded; the prognosis in these cases is generally favorable. Of the cases of empyema in which pneumo-pyothorax is produced by perforation of lung there is a fair prospect of recovery if other circumstances be favorable. If the affection be connected with pulmonary gangrene, death takes place generally within a short period. Other things being equal, when it occurs in the course of phthisis a fatal termination is speedy in proportion to the progress which the latter affection has made. There is a wide variation in different cases as regards its duration before a fatal ending; in many cases it ends fatally within a few days or weeks, but sometimes life is prolonged for months or even years. It may continue for an indefinite period, the patient experiencing so little inconvenience as to consider himself in fair health. The following unique case will serve to verify this statement, and also to illustrate the latency of the affection occurring in connection with phthisis:

I received from Dr. J. H. H. Burge of Brooklyn, N. Y., the left lung which he had removed at a post-mortem examination made two or three days before. The patient, aged eighteen, an accountant, was seized four days before his death with an acute affection which proved to be pneumonitis affecting the lower lobe of the right lung. At the time of the attack he considered himself in fair health. He was attended by Dr. Burge, who found the physical signs denoting pneumonitis, and the affected lobe was found after death in the second stage of this disease. On an examination of the chest, in addition to the pneumonitis Dr. Burge discovered the signs of pneumo-hydrothorax affecting the left side. Metallic tinkling and amphoric respiration and voice were well marked. The upper portion of the chest on this side yielded a tympanitic, and the lower portion a dull, resonance on percussion.

On inquiry as to the previous health the patient declared that he was well when attacked with the acute disease; that is, four days before his death. On close questioning, however, it was ascertained that he had had a slight cough for a year, but so slight that he thought nothing of it. He was not conscious of any deficiency of breath, was not subject to pain in the side, and, in short, there were no symptoms pointing to so grave an affection as pleuritis with pneumothorax.

The left lung was completely condensed from compression. The lower lobe was readily inflated; the upper lobe but slightly, owing to the free escape of air through a perforation as large as a crow's quill situated on the anterior aspect of this lobe about midway from the upper to the lower extremity. On closing the aperture this lobe was readily inflated. The perforation was gaping, the orifice being surrounded by a cartilaginous rim. On passing a probe into the aperture it entered a small cavity of about the size of an American walnut. The cavity was smooth and lined by a membrane. This was the only cavity in the left lung, and there were no phthisical deposits. The whole surface of the lung was covered with fibrin, which was dense and closely adherent. The pleural cavity contained more than a quart of turbid liquid which was not fetid. When this side of the chest was opened air escaped with force, emitting no fetor. The upper portion of the right lung contained several phthisical cavities, the largest of the size of an English walnut, with small phthisical deposits.

TREATMENT.—The treatment of pleuritis with pneumothorax in most cases embraces only palliative and sustaining measures. The severity of the pain and the distressing dyspnœa frequently call urgently for measures to procure relief. Opium, soothing applications to the chest, and the ethereal preparations are indicated for this object. The powers of the system are to be sustained by tonic remedies, alcoholic stimulants, and a nutritious diet. These measures will generally serve only to mitigate the sufferings of the patient and prolong life; and in the rare cases in which the affection continues for months, years, or indefinitely, and is not incompatible with comfortable health, there is no special treatment to be pursued, the main object being to invigorate and support the powers of the system.

If the patient suffer greatly from dyspnœa arising from dilatation of the affected side, the chest may be punctured as a palliative measure. I have resorted to this operation to the great relief of the patient. The air will be likely to reaccumulate and the side become again dilated as before; but I have known this not to occur for some weeks, and the operation may be repeated as often as called for by the dyspnœa arising from the dilatation.

These remarks on the treatment have reference to pneumo-pyothorax occurring in connection with phthisis, and not to the perforation of lung from without inward in cases of suppurative pleuritis. The treatment of pneumo-pyothorax when thus secondary to the latter disease has been already considered. (Vide p. 42.) It is a question whether, when occurring in cases of phthisis, a free incision into the chest and a permanent opening may not be advisable. I have resorted to this measure in a few cases with notable relief, and, apparently, life has been thereby prolonged. It is within the range of possibility that this procedure may effect a cure. If the phthisical affection be small and cease to progress, the perforation possibly may become closed by adhesions, and recovery from the pleuritic disease takes place. Considering that there is no prospect of effecting a cure by any other method of treatment, and that the condition of the patient is not injured but improved by making a free opening into the chest, there can be no objection to resorting to it. In making a free opening into the chest in a case of pneumo-hydrothorax an anæsthetic should not be administered. I am cognizant of a case in which death was due to the liquid making its way from the pleural cavity through the open perforation into the bronchi and trachea and being inspired into the opposite lung. This occurred while the patient was under the influence of chloroform and the operation for opening the chest was in progress.

When the chest, after perforation of the lung in cases of phthisis, becomes filled with liquid, this should not be withdrawn for a considerable period. Permanent closure of the perforation may sometimes ensue. An instance of recovery under these circumstances has not fallen under my observation, but I have known several weeks to elapse after aspiration before pneumothorax again occurred.

Pneumothorax.

The term pneumothorax denotes the presence of air, without liquid, in the pleural cavity. Under these circumstances pleuritis either does not exist or it is not accompanied by effusion. This affection is certainly extremely rare. Its occurrence has been doubted, but its existence is sufficiently established. In cases of pneumothorax without pleuritis the perforation of lung is not incident to either gangrene or phthisis, but is probably connected generally, if not invariably, with emphysema, which may not be general but circumscribed. Emphysematous dilatation of the air-cells, perhaps confined to a few lobules at the margin of a lobe or an emphysematous bleb, may lead to a minute rupture through which air is forced by the acts of breathing into the cavity of the pleura. Under these circumstances rupture may be caused by lifting a heavy burden, severe coughing, or violent exertions of any kind. The perforation, as remarked by Gairdner, may be no larger than a pinhole. The presence of the air does not excite pleuritis, but the accumulation may be sufficient to compress the lung into a solid mass, as in pleuritis with large effusion, dilating the affected side and removing the heart from its normal situation.

Pneumothorax without effusion from pleuritis is characterized by the absence of the symptoms and signs denoting inflammation and the presence of liquid. The patient suffers chiefly from the mechanical effects of the presence of air. These effects relate exclusively to the respiration and circulation. There is little or no febrile movement; the appetite, digestion, and nutrition are not affected, and the vital powers are maintained. If the affection were to end fatally, it would be by apnœa, assuming that pleuritis is not developed sooner or later. It is unquestionable that this affection may end in recovery. Under the influence of compression the perforation may become sealed up by a circumscribed pleuritis with the exudation of fibrin, or by the adhesion of the pleural surfaces at the point of perforation, the lung again expanding and resuming its function. Gairdner has quoted the report of a remarkable case illustrative of the occurrence of pneumothorax without pleuritis, and of its favorable course and termination.[1] Gairdner regards this case as unique in respect of the distinctness of the clinical facts on which the diagnosis was based. A case equally striking, and not less distinct as regards the diagnosis, has fallen under my observation. The important points taken from the history of this case, which was recorded very fully, are as follows:

A Polish peddler, aged twenty-nine, was admitted into hospital January 29, 1856. He stated that twelve days before his admission, being apparently in perfect health, he was attacked suddenly with a sharp pain in the region of the left nipple, accompanied with shortness of breath. This occurred while he was carrying his pack, which weighed about seventy pounds. He had no cough or expectoration. The pain soon disappeared, but the want of breath obliged him to give up his occupation and led him to the hospital. He was a vigorous man, weighing about one hundred and seventy-five pounds. He presented a healthy aspect, complaining only of deficiency of breath on exercise. This difficulty did not prevent him from being up and walking freely about the ward.

The left side was dilated and the intercostal depressions obliterated; the resonance over this side was purely tympanitic, and auscultation furnished a well-marked amphoric echo without any respiratory sound. Subsequently amphoric respiration became extremely well marked, together with metallic tinkling. The heart-sounds showed the heart to be to the right of the sternum.

After a month there was considerable improvement with regard to the deficiency of breath on exercise. The dilatation of the left side was no longer apparent, and the intercostal depressions became visible; vesicular respiration extended from the summit to the nipple, and the amphoric sounds, with the metallic tinkling, had disappeared.

Three weeks afterward the patient felt sufficiently improved to leave the hospital and return to his occupation. The respiratory murmur on the left side was feeble, but everywhere appreciable, with no abnormal modification save in intensity, and no amphoric sounds. The impulse of the heart was felt on the left side of the sternum.

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[1] *Clinical Medicine*, Edinburgh, 1862, p. 381.

The patient consulted me a month after his discharge from the hospital. He had returned to his occupation of peddling, carrying his pack on his back as before. He had experienced no difficulty from want of breath until three days before his visit to me, but for three days the deficiency of breath on exercise had been considerable. On examination of the chest the left side was everywhere tympanitic; there was no respiratory murmur on that side, and the heart was on the right side of the sternum. No amphoric sounds. He complained of nothing but the want of breath; excepting this he was well.

I did not see the patient afterward for eleven months. At the end of that time he called to see me with another patient. After his former call upon me he gave up peddling and became a farm-laborer. He now appeared and reported himself to be perfectly well. The respiratory murmur was everywhere heard over the left side, but was slightly weaker than on the right side. The apex-beat of the heart was in its normal situation. The left side laterally and posteriorly presented to the eye slight contraction.

This was a case of simple pneumothorax without pleuritis. A pinhole perforation at a weak point or the rupture of an emphysematous bleb probably took place in consequence of the muscular exertion of carrying his pack. He had nearly recovered when, on resuming his occupation, the perforation was reopened and the pneumothorax reproduced. Recovery again took place, and after the lapse of several months appeared to be permanent.

A sufficient number of cases of pneumothorax without pleuritis to serve as a basis for the clinical history and prognosis are not yet available. The infrequency of the affection is such that the collection of a considerable number of cases for analysis cannot soon be expected. The case just given, and the case reported by Gairdner, go to show that a favorable prognosis may be entertained. The physical signs are those of pneumo-pyothorax or pneumo-hydrothorax, excluding those denoting the presence of liquid. The affection is to be differentiated from diaphragmatic hernia.

As regards treatment, it is obvious that no special course of medication is called for. The compression of lung probably favors the healing up of the perforation, so that it would not be advisable to puncture the chest in order to allow the air to escape. Active exercise or muscular efforts, as in straining, are to be avoided, not only during the continuance of the affection, but for some time after recovery, lest the orifice may be reopened, as in the case just detailed. Soothing embrocations to the chest will be useful as palliatives, but nothing can be gained by active counter-irritation. A nutritious diet may be allowed. Cathartics, diuretics, and rubefacients are not indicated.

Circumscribed Pleuritis.

The frequency with which pleuritic adhesions over a limited area are found after death is proof that circumscribed pleuritis often occurs. But the inflammation which results in these adhesions is often subacute, and generally occurs without giving rise to much pain or other disturbance. As already stated, circumscribed pleuritis is secondary to some pulmonary affection, especially phthisis and pneumonitis. It differs from a primary general pleuritis in not giving rise to pleuritic effusion. Fibrinous exudation is sometimes small, and perhaps may be altogether absent, the adhesions taking place purely by a hyperplasia or proliferation of the connective tissue. Limited to the pleura investing the diaphragm (diaphragmatic or phrenic pleuritis), inflammation is supposed to give rise to symptoms which are distinctive—namely, hiccough attended with pain, and pain referable to the diaphragm in the acts of coughing. It may be doubted, however, if these symptoms be sufficient to mark the situation of inflammation or to distinguish inflammation from pleurodynia. If from the symptoms circumscribed pleuritis be suspected, the treatment indicated embraces moderate counter-irritation, soothing applications to the chest, and anodyne remedies. The successive attacks of circumscribed pleuritis which occur during the progress of phthisis, and which lead to firm adhesions, are seldom accompanied by much pain and only call for palliative measures.

Circumscribed collections of sero-fibrinous or purulent liquid are usually, if not always, preceded by general pleuritis, which has led to the adhesions surrounding the space in which the liquid is contained. These collections may be situated at the lower part of the chest, between the lung and diaphragm or between the lobes. The previous history and the appearances of the chest will generally show the prior existence of general pleuritis. The diagnosis of circumscribed collections of liquid is not always easy. For the physical signs the reader is referred to works which treat more fully of thoracic diseases. The treatment embraces the measures for promoting absorption, and if the liquid be purulent, this fact having been demonstrated by means of the exploring trocar, it is advisable to effect its removal by a free opening after a trial of repeated aspirations. The latter have been successful in some cases under my observation.

Hydrothorax.

Hydrothorax, using the term correctly, is not a variety of pleuritis, but it is noticed in this connection because, as far as certain symptoms and physical signs are concerned, it has points in common with pleuritis accompanied by liquid effusion. In the proper sense of the term it denotes a dropsical affection. The effused liquid is serum devoid of fibrin. The effusion is not a result of inflammation or any other affection of the pleural membrane, but proceeds from those conditions relating to the blood and circulation on which dropsical effusion in other situations depends. It very rarely, if ever, occurs alone, but is an element of general dropsy; that is, dropsical effusion exists at the same time in the peritoneal cavity and in the subcutaneous areolar tissue, the latter constituting either œdema or anasarca. As a rule, in cases of general dropsy the hydrothorax is subsequent to the anasarca and to hydroperitoneum.

The signs which denote the presence of liquid in the pleural cavity are applicable alike to inflammatory and dropsical effusion. The proof afforded by the change of level of the liquid, corresponding with changes of the position of the body, is more constantly available in hydrothorax, because fibrin is not present to agglutinate the pleural surfaces and lead to permanent adhesions. But the distinctive feature of this affection is the existence of effusion in both pleural cavities. Hydrothorax is bilateral, provided the pleural cavity on one side be not abolished by universal adhesions due to a previous attack of pleuritis, whereas pleuritis when it is primary is unilateral. The amount of dropsical effusion, however, in both sides is generally not uniform. It is not uncommon to find in the pleural cavity on one side a quantity of liquid considerably larger than is contained in the cavity on the other side. It may then be laid down as a rule that if the physical signs show the presence of liquid in both sides and general dropsy coexist, the affection is hydrothorax. A friction murmur is of course not developed in this affection.

Symptoms denoting inflammation—namely, pain, cough, and febrile movement—do not belong to the clinical history of hydrothorax. The respirations are increased in frequency, and the patient suffers from deficiency of breath or dyspnœa in proportion to the amount of dropsical effusion. Existing in both sides, it is obvious that the embarrassment of respiration will be the same as if all the liquid were in one pleural cavity, and twice as much as if the effusion in one side were unaccompanied by an equal effusion in the other side. If there be considerable effusion dyspnœa will be marked, amounting perhaps to orthopnœa, and death by apnœa may be due to the amount of effusion. Cardiac lesions producing obstruction of the mitral orifice especially favor the occurrence of hydrothorax after these lesions have led to dilatation of the right cavities of the heart.

The measures of treatment in hydrothorax are those indicated in general dropsy, and these need not be considered in this connection. Aspiration should be resorted to as a palliative measure if the amount of effusion be sufficiently large to occasion much suffering, although not sufficient to endanger life. If there be any danger from apnœa, aspiration is to be employed without delay. It is an unobjectionable measure. I have resorted to it frequently. As there is no obstacle to the expansion of the compressed lung in cases of hydrothorax, all the effused liquid may be at once removed without the distress which is liable to accompany aspiration in cases of pleurisy.

It should be added that, in some cases in which it is dependent on mitral obstruction, hydrothorax is greatly out of proportion to the amount of dropsical effusion in other situations. I have repeatedly observed prompt, complete, and permanent relief procured by the hydragogue operation of elaterium in these cases, but, as a more prompt and less debilitating measure, aspiration is to be preferred.

CHAPTER IV.

PNEUMONITIS.

Seat of the Inflammation.—Varieties: Acute Lobar Pneumonitis: Anatomical Characters; Laws of the Disease; Clinical History; Causation; Diagnosis; Pathological Character; Prognosis.

HAVING considered in the preceding chapters inflammation affecting the serous membrane investing the lungs, inflammation seated in the pulmonary substance or parenchyma is next to be considered. The latter is called *pneumonitis* or *pneumonia*. Pneumonia is the name commonly used, but pneumonitis is the more appropriate term, being in conformity with the plan of distinguishing inflammatory affections by the suffix *itis*. The latter term will be adopted in the headings, but elsewhere the two names will be used indifferently. The question at once arises, What constitutes the parenchyma or substance of the lungs? These terms relate to the pulmonary lobules, which are composed of air-cells, infundibula, alveolar passages, and bronchioles. A bronchus after it enters a lobule is called a lobular bronchus or bronchiole; it divides dichotomously once or twice, and terminates in the alveolar passages, into which open the air-cells, air-vesicles, or alveoli. The alveolar passages also divide a few times, and end in funnel-shaped spaces called infundibula, the walls of which are composed of thickly-placed air-vesicles. The lobular bronchi are devoid of cartilage-plates and of mucous glands, and are lined with a layer of low cylindrical ciliated epithelial cells. The walls of the air-cells and alveolar passages are composed of a homogeneous basement-substance containing a few oval nuclei of connective-tissue cells and a rich network of elastic fibres and of pulmonary capillaries. They are lined with a single layer of flat polygonal cells, some of which are small, nucleated, and granular, others larger, non-nucleated, and homogeneous in appearance. It is only the nucleated, granular, epithelial cells which take any active part in inflammatory diseases of the lungs. In acute pneumonitis the inflammation is seated in the air-cells and bronchioles. The differences in structure between the membrane composing the wall of these parts and the bronchial mucous membrane, together with a difference of function and of blood-supply, will serve to account for the fact that the inflammation in pneumonitis may be limited to the pulmonary substance, and also the fact that in bronchitis the inflammation rarely extends to the pulmonary parenchyma. This is in accordance with a pathological law — namely, that inflammation of a structure does not, as a rule, extend to another structure, however proximate, which has important anatomical differences or which differs as regards its functions. Some striking examples of conservatism in disease will be found to hinge upon this pathological law. The fact also that the lining membrane of the air-cells is not, strictly speaking, a mucous membrane will account for important differences pertaining to the inflammatory products of pneumonitis when compared with bronchitis and other mucous inflammations. The relation of the air-cells to the terminal branches of the pulmonary artery is also involved in the pathological phenomena of inflammation of the parenchyma of the lungs.

The different forms of pneumonitis may be classified as fibrinous or lobar, catarrhal or lobular, purulent, fibrous, and caseous or tubercular. Caseous pneumonitis and fibrous pneumonitis will be considered in connection with phthisis.

Acute Lobar Pneumonitis—Pneumonic Fever.

Acute lobar pneumonitis is the disease called by German writers *croupous pneumonia*. By others, especially French writers, it is sometimes called *fibrinous pneumonitis*. According to the view which will be presented in treating of the pathological character of the disease, each of these names is incorrect, and they all imply that the disease is a purely local inflammation, whereas there are sufficient grounds for regarding it as an essential fever. A more appropriate name, therefore, is pneumonic fever. The name acute lobar pneumonitis is here retained in conformity with usage and for the sake of convenience for reference.

ANATOMICAL CHARACTERS.—The first appreciable change resulting from acute inflammation is the same here as in other situations—namely, active congestion accompanied by transudation of serum. This is known as the first stage or stage of congestion or engorgement (*engouement*). The inflamed portion of the lung is heaver than in its healthy state; on section the cut surfaces present a dark-red appearance, and a bloody, frothy liquid flows in abundance. The affected portion of the lung presents the same appearance as in œdema with marked congestion. The microscopical examination shows the pulmonary capillaries widely distended with blood, and the air-cells filled with a serous fluid containing red blood-corpuscles in abundance, a few white blood-corpuscles, and larger granular cells which are the epithelial cells washed off by the watery transudation from the blood. In this stage the affected portion of the lung does not sink in water.

In the second stage, usually called the stage of red hepatization, the air-cells and bronchioles become filled with a solid exudation and cease to contain air. The solidified lung is of a brownish-red color, non-crepitant, and presents an appearance not unlike that of the liver, whence the name hepatization. Upon its cut or torn surface it presents a very characteristic granular appearance, due to the projection of the little solid masses which distend the air-cells and bronchioles. In a lung previously affected with emphysema these granules are very large and distinct. When examined soon after death the cut surface is dry; later, a viscid, turbid, reddish fluid may be made to escape. The substance of the lung breaks down under the pressure of the finger more readily than in its healthy state; that is, it is more friable. Its volume is about equal to that of the lung in a state of full inspiration, and it does not collapse when the chest is opened. Its weight is much increased; a single lobe solidified by inflammatory exudation increases in weight from one to two pounds, and an entire lung solidified may acquire an additional weight of four pounds. Owing to the absence of air, portions of the solidified lung sink when thrown into water. Examined with the microscope, the substance filling the air-cells and small bronchi is found to be composed of a network of fibrillated fibrin containing in its meshes a large number of pus-cells or white blood-corpuscles and of red blood-corpuscles. A few desquamated and proliferating epithelial cells of the alveoli and a variable amount of amorphous granular matter are also present. The reddish color of the lung in this stage is due to congestion and to the large number of red blood-corpuscles present in the exudation. Some of the air-cells often appear to be completely filled with fibrin and red blood-corpuscles, conveying the impression of an extravasation of blood. Probably most of the red blood-corpuscles escape by diapedesis. There can be little doubt that the pus-cells are emigrated white blood-corpuscles. The lung-texture is not essentially altered by the inflammatory process. It contains, especially in the interlobular connective tissue, some emigrant cells.

When the brownish-red color of the solidified lung gives place to a gray color, the lung is said to be in the stage of gray hepatization. The granular appearance is then less distinct and the surface is moist. The red blood-corpuscles are fewer in number, but the pus-cells are more abundant and contain molecules of fat. The lung may present the appearance of gray hepatization after death within a few days from the onset of the disease, or the aspect may be that of red hepatization after the disease has lasted two weeks or more. Most frequently the lung is found in the condition of mottled hepatization; that is, of mixed gray and red. It is usually considered that gray hepatization is a later stage of red hepatization, the inflammatory products being in process of absorption. Absorption may take place with great rapidity. The statement by Rindfleisch and others that the greater part of the exudation is expectorated is certainly incorrect. The matter expectorated, in general, bears no proportion to the quantity of exudation, and there are cases in which the exudation disappears without any expectoration.

In the third stage, or stage of absorption,[1] the red blood-corpuscles lose their coloring matter, the pus-cells undergo fatty degeneration, the fibrin becomes granular, and an effusion of serum takes place, so that a kind of granulo-fatty emulsion is formed suitable for absorption by the lymphatics and blood-vessels. In favorable cases after the removal of the exudation the air-cells are found to have sustained no damage. The pulmonary structure remains intact while the exudation remains, and its functional capacity is fully restored after the exudation disappears.

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[1] Anatomically, it is customary to reckon four stages of pneumonitis—viz. engorgement, red hepatization, gray hepatization, and resolution. As red hepatization and gray hepatization cannot be distinguished from each other clinically, they are here combined into one stage, that of consolidation.

When after death the color of the inflamed lung is yellow or grayish-yellow, the granular appearance absent, the tissue soft and exuding a creamy fluid, the lung is said to be in the condition of purulent infiltration. The exudation which fills the air-cells consists almost wholly of pus, and pus-cells are present abundantly in the interlobular tissue, but the lung-tissue is not broken down. This condition of purulent infiltration is thought to favor the formation of pulmonary abscesses in which collections of pus take place in cavities resulting from breaking down of the lung-tissue. Gangrene of the affected portion of lungs sometimes occurs, but this, as well as the occurrence of abscess, is extremely rare. Lobar pneumonitis never terminates directly in caseous degeneration, as has been claimed by some writers. When phthisis intervenes upon lobar pneumonitis an infection with the tuberculous virus must have occurred.

It is probable that in rare instances lobar pneumonitis terminates in interstitial pneumonitis characterized by a new growth of connective tissue in the walls of the air-cells. Little nodules of connective tissue may grow from the alveolar walls into the interior of the air-cells, which are thereby more or less completely occluded. There is, however, a difference of opinion as to whether these cases of interstitial pneumonitis apparently following lobar pneumonitis are not from the beginning a peculiar form of pneumonia not to be identified with the ordinary lobar variety. The symptoms, however, at the onset of the disease may not be distinguishable from those of lobar pneumonia.

Pleuritis with fibrinous exudation is developed in the portion of the pleura covering the affected lobe or lobes of the lung. This concurrent pleuritis is never absent except in those rare cases in which the pneumonitis has not extended to the surface of the lung. The pleuritis is circumscribed and dry; much liquid effusion occurs only as an exception to the rule. To these exceptional cases with liquid effusion may be applied the term *pleuro-pneumonia*, although in a strict sense nearly every case of pneumonia is pleuro-pneumonia.

More or less bronchitis, affecting the bronchial tubes within the affected lobe or lobes, usually exists with pneumonitis. In some cases, however, the pneumonitis passes through its whole course without affording any evidence of this circumscribed bronchitis. Bronchitis affecting the bronchial tubes of both lungs is sometimes, but rarely, present with pneumonitis. When these two affections are combined it is accidental; bronchitis, as the primary affection, does not tend to the development of pneumonitis, and the latter has no tendency to give rise to bronchitis save within the affected lobe or lobes.

Of the various forms of bacteria which may be found in the affected bronchi and air-cells in lobar pnenmonitis, at present the most important are the elliptical organisms which are described by Friedländer as micrococci, but which are classified by some authorities preferably as bacilli. These oval bacteria occur frequently in pairs and sometimes in chains. Their special morphological characteristic is the presence of a gelatinous capsule which can be stained by certain aniline dyes. This capsule, however, may exist around other forms of bacteria. Friedländer's capsule cocci are found in the exudation especially during the stage of red and of mottled hepatization, and less abundantly in gray hepatization. The pure cultures of the cocci in nutrient gelatin are compared in their form to a nail, the head of the nail being represented by the knob-like growth of the organisms on the surface of the gelatin, and the rest of the nail by the growth in the line produced when the gelatin is stabbed by the wire used in planting the organisms. These bacteria have been found not only in the lungs after death, but also in the sputum during life, and in sanguinolent fluid withdrawn by a hypodermic syringe from the hepatized lung during life. Injections of the pure cultures into the lungs of mice causes lobular pneumonia, pleurisy, and invasion of the blood by the organisms. Pneumonia has been produced by the injection of the organisms into the lungs of dogs and of Guinea pigs, but the effects are inconstant. Rabbits seem to be refractory to the inoculation of the organisms.

The evidence that the capsule cocci of Friedländer are the infectious agents in lobar pneumonia cannot be considered conclusive. Among the objections to accepting them as the cause of pneumonia may be mentioned their inconstancy, the presence of other bacteria in pneumonia, and the possibility of producing similar results in animals by the inoculation of different kinds of bacteria. Bacteria morphologically identical with the so-called cocci of pneumonia have been found in the sputum in the absence of pneumonia. Further observations and experiments are necessary in order to determine the real significance of the pneumonic cocci.[1] It may be added, however, that if acute lobar pneumonia be correctly regarded as an infectious disease, its pathological connection with a specific micro-organism is a rational inference. The question, then, is whether the bacteria described by Friedländer have a special pathological connection with this disease, or whether a special parasitic organism is yet to be discovered.

To the foregoing sketch of the anatomical characters may be added certain laws of the disease, which are best presented in this connection. One of these relates to the situation of the pulmonary affection. Pneumonitis attacks, in the great majority of cases, the lower lobe, and the lower lobe of the right oftener than of the left lung. Exceptionally, it sometimes attacks primarily an upper lobe, and in these cases, as a rule, the disease is more severe; but to this rule there are exceptions. The disease very rarely, if ever, attacks two lobes simultaneously, but it invades not infrequently in succession a second and even a third lobe. The inflammation does not extend from one lobe to another, but whenever a new lobe is affected it is the seat of a new invasion. The lobes of one lung may be successively invaded or a single lobe on both sides; or, an entire lung being first affected, a lobe of the other lung may be attacked. In the two latter cases the pneumonitis is said to be double. The name "crossed pneumonia" has been applied to the disease when the lower lobe of one lung and the upper lobe of the opposite lung are affected.

The inflammation extends at least over an entire lobe, as the name lobar pneumonitis implies, in the great majority of cases. There are exceptions to this rule. I have known some instances in which the physical signs clearly showed the occurrence of inflammation extending over a limited area, without any evidence of the circumscribed pneumonitis being a complication of any other pulmonary disease. In a lobe invaded secondarily—that is, one lobe being already affected—the inflammation is sometimes found after death to have extended over a portion only of the lobe. This fact I have repeatedly noted, but perhaps had the life of the patients been prolonged the whole of the lobe would have been affected.

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[1] It is claimed by Sternberg and by Fränkel that the micrococci which are sometimes found in the sputum of healthy persons, and which when inoculated into rabbits cause a severe and fatal form of septicæmia (Sternberg's sputum septicæmia), are the probable specific cause of acute lobar pneumonia. These sputum micrococci have been found in the lung in lobar pneumonia, and are more common in pneumonic sputum than Friedländer's bacilli. No positive proof of the correctness of these views has been brought forward.

The whole of the lobe first invaded is not at once affected. The inflammation begins at a certain point or at several distinct points, and extends from lobule to lobule until the entire lobe is involved. The point or points of departure may be at either the upper or lower extremity of the lobe, and at either the superficies or centre of the lobe. The diffusion of the inflammation over the lobe takes place with more or less rapidity; sometimes a few hours suffice, but in some cases it occupies several days. The progress of the solidification from the exudation may be determined very accurately from day to day or from hour to hour by means of physical signs.

The inflammation very rarely ends with the occurrence of engorgement only, without exudation. Solidification almost always occurs, and, when the inflammation is not secondary to another pulmonary disease, usually extends over the lobe or lobes invaded.

With respect to the situation and extent of the affection, the following are the results of an analysis of 121 cases:[1] In 29 cases it was limited to the lower lobe of the *right*, and in 25 cases to the lower lobe of the *left* lung. It extended over the whole of the *right* lung in 27, and over the whole of the *left* lung in 9 cases. It was limited to the upper lobe of the *right* lung in 8, and to the upper lobe of the *left* lung in 3 cases. It was seated in the lower lobe of both lungs in 8 cases.

CLINICAL HISTORY.—The division of acute pneumonitis into stages is based upon differences as regards the anatomical characters at different periods of the disease. The *first* stage embraces the period during which the affected lobe is in the state of active congestion or engorgement. This stage is called the stage of engorgement. The disease is considered as passing into the *second* stage when the affected lobe or a greater part of it has become solidified by the inflammatory exudation. This stage is called the stage of solidification or hepatization. In the *third* stage the affected lobe is in one of two conditions. If the disease pursue a favorable course, the third stage begins when it is evident that absorption of the exuded matter is going on, and convalescence takes place during this period. This may be called the stage of resolution. If the disease pursue an unfavorable course the third stage is one of purulent infiltration. If this stage occur the disease generally ends fatally.

The duration of each of these stages varies much in different cases. The stage of engorgement may last for only a few hours. I have known an entire lobe to be solidified by two pounds of exudation matter, as determined after death, in less than twelve hours. Not infrequently this stage does not extend beyond twenty-four hours. But sometimes the solidification occupies two, three, or four days, and in some cases even a longer period. In the majority of cases this stage is from twenty-four to forty-eight hours in duration. The stage of solidification may also be of short duration. I have known resolution to begin and to make considerable progress in twenty hours. But this may not be evident for two, three, or four days, or even for a considerably longer period. In the majority of cases the duration of this stage is from two to four days. The stage of resolution is still more variable. There is a notable difference in different cases as regards the rapidity or slowness with which the solidifying deposit is removed. It is very rarely the case that the resolution is completed in less than three or four days, and generally eight or ten days are required. In some cases two or three weeks elapse before the air-cells are restored to their normal condition, and occasionally the resolving process is even much more protracted. If the disease pass into the purulent stage, death usually takes place within a few days; but if the disease end in recovery, many days and perhaps weeks elapse before the normal condition is restored.

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[1] "Clinical Report on Pneumonia, based on an Analysis of 133 Cases," by the author. Vide *American Journal of the Medical Sciences*, January, 1861.

In the great majority of cases acute pneumonitis begins with a well-pronounced chill, frequently accompanied by rigors. The thermometer in the axilla shows an increase of temperature, although coldness exists as a subjective symptom. The invasion is usually abrupt, with few or no premonitions. The attack is liable to occur during the night. Coincident with or speedily following the chill is the occurrence of pain. Sometimes, however, pain is not felt until several hours after the chill. The pain is frequently severe. It is acute, lancinating, and in all respects identical with the pain in acute pleuritis. It proceeds from the pleuritis developed in conjunction with the pneumonitis. As a rule, the intensity of the pain is in proportion to the amount of coexisting pleuritis; but it is true alike of pleuritis thus developed as a complication and when it occurs primarily that it is sometimes attended with little or no pain. Cases of pneumonitis differ considerably as regards this symptom, and it may be quite prominent or present in a moderate degree or altogether wanting. The pain is usually referred to a circumscribed space at or near the nipple of the affected side. This limitation of the pain is a point distinctive of pneumonitis as compared with pleuritis.

Cough is usually present at or soon after the invasion. It is more or less prominent. It is sometimes wanting. It is painful in proportion as pain is a prominent symptom irrespective of the cough. The cough is frequently accompanied by expectoration. The matter at first expectorated is scanty, transparent, and viscid, and in a certain proportion of cases it soon assumes characters which are highly distinctive of the disease. It becomes semi-transparent, adhesive, and has a reddish tint like that of iron rust; hence it is commonly known as the rusty expectoration. This appearance is due to a small quantity of blood which has become intimately mixed with the matter in its passage from the smaller to the larger bronchial tubes. The adhesiveness is such that when a considerable quantity has accumulated it adheres so closely to the bottom of the vessel as to remain when the vessel is inverted. The rusty expectoration is by no means uniformly present. The matter is in some cases semi-transparent and adhesive without the reddish tint. It may have a yellowish tint, as if mixed with a little bile, which, however, is not the fact. It sometimes contains blood in abundance, and when the blood is of a dark color it is what is called the prune-juice expectoration. The characteristic oval bacteria surrounded by a capsule may be found in the sputum, as has already been described, but they are not invariably present. It is to be borne in mind that expectoration in some cases of pneumonitis is entirely wanting.

Pyrexia exists at the outset, together with pain in the head, loss of appetite, thirst, prostration, heat of skin, etc. The pulse varies much in frequency in different cases, ranging from 80 to 120, and is more or less full and hard. The thermometer in the axilla indicates more or less increase of heat, the increase in mild cases not exceeding 104° F. An increase above this point always denotes severity of the disease. A sudden increase of temperature points to the invasion of a new lobe or to some intercurrent affection. Cases differ greatly as regards general or constitutional disturbance, the system being much more tolerant of the disease in some persons than in others.

The respirations are increased in frequency. This may depend on the pleuritic pain if it be marked, and also on the interruption of the function of hæmatosis in the portion of lung affected.

These are the important symptoms belonging to the clinical history of the beginning and during the first stage of the disease. During the stage of solidification the symptoms change. Pain, if it have existed, diminishes or ceases. Cough and expectoration continue, but the cough is less hard and painful and the expectoration is effected with more facility. The matter expectorated loses its rusty appearance, and becomes opaque, less adhesive, and more abundant. It is now the expectoration of resolving bronchitis. It is furnished chiefly by the bronchial tubes within the affected portion of lung, and is abundant in proportion as the bronchial mucous membrane is involved in the inflammation. On microscopical examination of the sputa fibrinous casts of the minute bronchial tubes may be found. The pyrexia continues, but with diminished intensity. The pulse in this stage may be small and deficient in strength, especially if more than one lobe be involved, owing to the obstruction to the pulmonary circulation, which diminishes the quantity of blood received from the lungs by the left auricle, and owing also to the amount of material withdrawn from the blood and deposited in the air-cells. The respirations are increased in frequency out of proportion to the increased frequency of the heart's action, in consequence of the solidified portion of lung taking no part in the respiratory function.

These are the modifications of the symptoms, assuming a single lobe only to be affected and that the course of the disease is favorable. The invasion of another lobe is rarely accompanied by a chill. The pyrexia persists or is increased, and the respirations become more frequent, accompanied perhaps by labored breathing, marked dilatation of the alæ nasi, and dyspnœa. The affection, however, of an entire lung does not always give rise to symptoms denoting great gravity of disease. The pyrexia is not always intense, and may be slight; the respirations may be but little increased in frequency; cough and expectoration may be not only not prominent, but wanting, and the system may be but little disturbed.

The symptoms during the stage of resolution denote progressive improvement. The pyrexia diminishes and ceases. The cough and expectoration become less and less, the respirations resume their normal frequency, the appetite returns, the strength is increased, and, in short, convalescence is declared. In some cases the fever ceases abruptly, a copious perspiration preceding the defervescence. It is customary to say that the disease in these cases ends with a crisis. In most cases the defervescence is gradual (lysis), and without the occurrence of any event which may properly be called critical. If, however, the disease pass into the stage of suppuration, the symptoms denote an unfavorable course. The pulse becomes more and more frequent and feeble. The expectoration is abundant and purulent. The respirations are frequent. The strength of the patient fails, and death takes place by asthenia, the accumulation of morbid products in the air-tubes frequently contributing to the fatal termination.

There are certain symptoms belonging to the clinical history of pneumonitis which are not included in the foregoing sketch of its symptomatology. One of these relates to the urine. While the process of exudation is going on, and after solidification has taken place, until resolution begins the chlorides frequently are greatly diminished or disappear from the urine. The disappearance of the chlorides from the urine is not peculiar to pneumonitis. It is observed in other diseases. It is not, therefore, a diagnostic character of the disease, but it constitutes evidence either that the exudation is going on or that resolution has not begun. It is stated that the chlorides are found in abundance in the matter expectorated during the time of their disappearance from the urine. The quantity of urine is in general more or less diminished. The urea eliminated exceeds the amount in health, and, owing to the diminished quantity of water, the urates are often deposited in abundance when the urine becomes cold. The increase of urea denotes an abnormal destructive metabolism of nitrogenous substances. It is not uncommon for the urine to contain a trace or a moderate quantity of albumen. The presence of albumen is probably due to systemic venous congestion or to an altered condition of the blood, as in other acute general diseases. Albumen in abundance in the urine denotes coexisting renal disease.

Delirium not infrequently occurs in acute pneumonitis. It may be mild and merely incidental to the pyrexia; but it is sometimes a prominent symptom. It may be active, the patient attempting to get out of bed, resisting efforts of restraint, and perhaps using violence; or it may be of the low, incoherent, muttering kind, such as occurs in typhoid fever. Delirium is more likely to occur if the upper lobe be affected than if the affection be limited to the lower lobe. It is evidence of gravity of the disease in proportion as it is prominent, active, and persisting. It is likely to occur in spirit-drinkers.

A dusky redness of one or both cheeks, the margins of the redness being abruptly defined, is frequently observed, and, if the hectic flush of pulmonary tuberculosis be excluded, is quite distinctive of acute pneumonitis. When confined to one cheek it does not follow, as was formerly supposed, that the pulmonary inflammation is seated on the same side. It is not uncommon for an herpetic eruption to occur upon the face, more especially around the mouth.

Slight jaundice is an occasional concomitant, due to duodenitis. It occurs especially in spirit-drinkers. Its occurrence is probably accidental. At all events, it is not associated often enough to show any pathological connection between pneumonitis and hepatic disease. Owing to the accumulation of blood in the right cavities of the heart, and the consequent congestion of the systemic veins, the liver sometimes becomes more or less enlarged.

The occurrence of abscess and gangrene is accompanied by symptomatic phenomena denoting these events. The collection of pus in an abscess leads to a sudden and copious purulent expectoration if the life of the patient be sufficiently prolonged, and the existence of a cavity may be determinable by physical signs. This is a grave event, but it by no means necessarily renders the disease fatal. It occurred in 4 out of 133 cases which I recorded and analyzed, and 2 of these 4 cases ended in recovery. I have notes of a case, in addition to the 4 cases just referred to, in which a series of purulent collections were expectorated and the recovery was complete. The infrequency of gangrene is shown by its having occurred in but 1 of 133 cases. The disease proved fatal in that case. Since the analysis of these cases I have known gangrene to occur to a limited extent in a case which ended in recovery. Its occurrence is shown by the characteristic fetor attending expectoration, together with the appearance of decomposed pulmonary tissue in the matter expectorated.

The combination of other affections with acute pneumonitis will of course involve the addition of symptoms derived therefrom. In malarial districts it may be conjoined with periodical fever. This is an important complication as regards danger and therapeutical indications. The existence of the pneumonitis may serve to interrupt to a greater or less extent the regular succession of periodical paroxysms, and the latter may tend to obscure the phenomena of the pulmonary affection. Pneumonitis occurring in the intemperate is liable to lead to the development of delirium tremens. The complication is always serious, and is important with reference to treatment. It occurred in 8 of 133 cases. Pericarditis is another grave complication. This occurred in 8 of 133 cases.

The supervention of certain symptoms is expressed by the term *typhoid pneumonitis*. This term denotes the existence of symptoms analogous to those which belong to typhoid fever, of which the most prominent is low, muttering delirium. A distinction is here to be made between typhoid pneumonitis and typhoid fever with pneumonitis as a complication. Pneumonitis is liable to be developed in the course of typhoid fever, the latter being the primary affection; but in the so-called typhoid pneumonitis the latter is the disease, certain of the typhoid phenomena being superadded. In other words, the typhoid state may be developed in connection with pneumonitis as with other affections, but this typhoid state does not involve the essential fever designated as typhoid. There is not sufficient ground for considering "alcoholic pneumonia" as a distinct variety, but habits of spirit-drinking occasion certain distinctive features—namely, jaundice and the phenomena of delirium tremens; the ability to resist and recover from the disease is also thereby impaired.

The duration of acute pneumonitis, when uncomplicated, varies within pretty wide limits. Dating from the attack to the time when the patient could be pronounced convalescent in 30 cases, the shortest was five and the longest twenty-three days. The mean duration was a fraction over twelve days. In 14 fatal cases the shortest duration from the attack to the time of death was three, and the longest twenty days, the mean duration being a fraction over ten days.

A notable decrease of temperature sometimes precedes the fall of the pulse and improvement in other symptoms; and the defervescence, if the disease be uncomplicated, is usually rapid. It is then said to end by crisis instead of lysis. The pulse during convalescence is not infrequently below the normal frequency, falling in some cases to 40 per minute.

CAUSATION.—Statistics show that no period of life involves either exemption from, or a notable proclivity to, this disease. It occurs under five years of age, but not often. Cases are less infrequent between the ages of five and ten. Of 118 cases, the patients all were over ten years of age; 13 were under twenty; 44 between twenty and thirty years; 37 were between thirty and forty years; 17 were between forty and fifty years; 7 were between fifty and sixty years; and in no case was the age over sixty. Cases, however, are not infrequent after the age last named. The disease occurs much oftener among males than females. The analysis of my cases with reference to occupation does not show that any particular calling predisposes to this disease, but the proportion of laborers engaged in outdoor work over those employed within doors shows that they are most liable to the disease who are exposed to the vicissitudes of the weather. Not infrequently the attack is excited by some unusual exposure, such as working in the cold and wet or sleeping out of doors at night. The large proportion of hospital cases in which patients are addicted to excessive drinking shows that causative influences proceed from intemperance. In 10 of 37 cases in which the habits were noted the attack followed a debauch. It is questionable whether the abuse of alcohol acts directly as a cause; probably the disease is caused by circumstances incidental to intemperance, such as exposure to cold, etc., these acting as exciting causes.

The association of periodical fever and pneumonitis has already been adverted to. My observations do not show the existence of any pathological connection between these affections. There is no ground for the conclusion that persons subject to intermittent fever are thereby more prone to pneumonitis, or that the latter affection tends to reproduce an attack of the former. Their coexistence is due to the coincidence of the causes proper to each.

With respect to causative influences pertaining to other pulmonary affections, facts show that bronchitis does not eventuate in pneumonitis. The same is true of pleuritis. Tuberculous patients are not particularly prone to the disease, and when it does supervene it may or may not affect the upper lobe, in which the deposit of tubercle is most abundant. Certain pulmonary affections—namely, emphysema, asthma, and chronic pleuritis—are so infrequently associated with pneumonitis as to show that, instead of involving a predisposition to this disease, they afford more or less protection against it. Pneumonitis is not often developed in persons affected with organic disease of the heart.

Pneumonitis occurs as a complication of other than pulmonary diseases. It is not infrequently developed in the course of typhus and typhoid fever, rubeola, dysentery, etc. The different affections of the kidneys have been supposed to stand in a causative relation to it. My own experience does not furnish evidence of such a relation. In not one of 133 cases analyzed was the disease preceded by albuminuria.

It occurs in certain seasons of the year in preference to other seasons. It occurs everywhere much oftener during the winter than during the summer months, and in our Northern States cases are more frequent in the spring months than at any other season. In this country the disease occurs in the Middle and Southern much oftener than in the Northern States. It prevails much more in some years than in others. In sections of the Southern States it prevails at times sufficiently to be considered as endemic. It is the prevalent disease during the winter months in these States, and, affecting especially the negro population, often proves the scourge of the sugar and cotton plantations of the South. It is a severer disease in the Southern sections of the country than at the North, being more liable to extend beyond a single lobe and proving fatal in a larger proportion of cases. This liability is greater in the same district in some years than in others. In some years also there is an unusual tendency in the disease to attack primarily an upper lobe. Such a tendency is manifest at the time of writing these remarks. Within two months (October and November) in hospital practice I have met with five cases in which the disease has been limited to an upper lobe, being about one-half the cases which occurred under my observation during this period.

In a large proportion of the cases of acute pneumonitis the disease is apparently developed spontaneously; that is, it is not referable to any obvious causative agency. When it appears to follow exposure to cold, it is probable that this acts only as an exciting cause co-operating with the action of a special cause. Litten has described cases of acute lobar pneumonitis following two or three days after contusion of the chest without any external wound. It is possible that in these cases physical changes in the lung produced by the contusion favored the development of a special infectious agent of pneumonia. Direct injury of the lung, as in stab-wounds and gunshot wounds, has no tendency to cause lobar pneumonia.

Clinical evidence seems inadequate to prove contagiousness. Instances in which persons in the same neighborhood and members of the same household are attacked successively may be explained by supposing an exposure in common to an extrinsic cause. Yet if it be demonstrated that the disease can be produced in an inferior animal by inoculation, this fact is proof of communicability, and its apparent incongruity with clinical experience is to be accounted for, as in the instance of pulmonary phthisis, by supposing a peculiar predisposition, or, in other words, a favorable soil, to be an essential factor in the causation. The supposed dependence of this disease upon a specific parasitic organism has been referred to in connection with the anatomical characters. An attack affords no protection against subsequent attacks.

DIAGNOSIS.—Acute pneumonitis in some cases is accompanied by symptoms which are highly distinctive. If a patient be seized with a chill followed by febrile movement, together with a pleuritic stitch referred to a circumscribed space near the nipple, and the characteristic rusty expectoration occur, the diagnosis is readily made without the aid of physical signs. But these symptoms are by no means constantly present. The disease is not infrequently entirely latent as regards diagnostic symptoms, and it is often overlooked by those who do not employ physical exploration. Even if its existence be revealed by symptoms irrespective of signs, the situation and extent of the affection can only be ascertained by means of the latter. With the aid of signs the diagnosis in the great majority of cases is made without difficulty. The diagnostic symptoms are especially liable to be absent and the disease to be developed insidiously when it is associated with other diseases. Not infrequently an examination of the chest reveals its existence unexpectedly. I have been accustomed to characterize it under these circumstances as *sneaking pneumonia*, using the language of personification which is so often used in speaking or writing of diseases. The term "asthenic pneumonia" has been applied to it when the symptomatic characters which belong to typical cases are wanting. On the other hand, the term "frank pneumonia" is often used to distinguish cases in which these characters are well marked.

Patients with this disease do not always take at once to the bed. It is not very uncommon to meet with cases in dispensary practice, the disease having gone on to the second stage. These may be distinguished as "walking cases." A young woman once consulted me at my house, complaining chiefly of weakness and loss of appetite, with a slight cough. The temperature in the axilla was 103°, and physical exploration showed solidification of the entire lower lobe of the right lung. She had taken a long drive the previous day, thinking it might be of benefit to her, and she had come two miles to see me. The history rendered it certain that the disease was acute lobar pneumonitis.

In the first stage the signs on which the diagnosis is to be based are slight or moderate dulness on percussion and the crepitant râle. The latter, if persistent and well marked, is almost pathognomonic. Care must be taken, however, not to confound this râle with the subcrepitant, its distinctive characters being its fineness, dryness, and limitation to inspiration. With a distinct appreciation of these characters it should never be confounded with the so-called subcrepitant râle, which, however fine, is a moist, bubbling sound, and liable to be heard in expiration as well as in inspiration. Care must be taken not to mistake for the crepitant râle the sound produced by movements of the stethoscope on the chest if covered with hair, and the crepitus of a fractured rib. It is also to be borne in mind that after rising to the sitting posture a feeble patient with any disease may furnish for a few deep inspirations a true crepitant râle on the posterior aspect of the chest. But the crepitant râle is not uniformly present in lobar pneumonia. In its absence a positive diagnosis may require some delay until a sufficient number of lobules are solidified to give rise to an appreciable modification of the respiratory murmur. The modification is that which I have described under the name broncho-vesicular respiration[1]—a modification approximating, more or less, toward the bronchial or tubular respiration. With the occurrence of this modification the dulness on percussion becomes more marked. In most cases, if the disease be observed from the beginning, the signs of the second stage are soon declared. In hospital practice and in many cases in private practice the disease has already advanced to this stage when the patient is first examined.

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[1] Vide *Physical Exploration of the Chest and the Diagnosis of Diseases affecting the Respiratory Organs*, 2d ed., 1866; also, *Manual of Auscultation and Percussion*, 3d ed., 1885.

When the solidification is sufficient in degree and has extended sufficiently over the affected lobe to furnish the signs of that condition, the diagnosis, if it have not been already fully made, is rendered clear by the presence of these signs. Bronchial respiration, bronchophony, and whispering bronchophony are the signs denoting the condition of solidification. They are first manifested over a limited portion of the affected lobe, and are thence diffused over its whole extent, either quickly or gradually according to the rapidity or slowness with which the whole lobe becomes solidified. The progress of the second stage may be determined by the extension of these signs. They are generally all present, but if one be wanting the others will be likely to be present. It is exceedingly rare for all to be absent. Dulness on percussion is now marked, amounting perhaps nearly or quite to flatness. This dulness or flatness extends over a space corresponding to that occupied by the solidified lobe; or, percussing anteriorly, the boundary-line separating the dulness or flatness from the pulmonary resonance is found to pursue a course coincident with the situation of the interlobular fissure—namely, obliquely upward and outward from the fourth or fifth costal cartilage toward the axilla. And this boundary-line is the same whether the patient be sitting or lying upon the back. This is assuming that the pleural cavity does not contain liquid, which is ascertained by the signs indicated in connection with the diagnosis of simple pleuritis. The crepitant râle, if it have existed in the first stage, may continue, more or less diminished, during the second stage, or it may disappear after the affected side has become solidified. The moist and dry bronchial râles are liable to be heard in this stage.

The invasion of a second or third lobe is denoted by dulness on percussion and the auscultatory signs of solidification, the crepitant râle rarely occurring in the lobes which become secondarily attacked. By means of the signs of solidification—namely, broncho-vesicular and bronchial respiration, and bronchophony with the loud and whispered voice—the extension of the affection to other lobes in addition to the one first invaded is speedily ascertained. If the upper lobe become solidified, it is sometimes flat on percussion, and sometimes it yields a tympanitic resonance which occasionally has an amphoric intonation.

The beginning of resolution and its progress from day to day are shown by modifications of the signs denoting solidification. The bronchial respiration gives place to the broncho-vesicular, and the latter progressively approaches more and more to the normal vesicular murmur, into which it becomes finally merged when the resolution is completed. The characters of bronchophony are gradually lost. Dulness on percussion becomes less and less marked, but some degree of dulness over the affected lobe or lobes continues for some time after the auscultatory signs show the exudation to have been removed. During the progress of resolution the subcrepitant râle is frequently heard, and sometimes the crepitant râle reappears, constituting the *crepitant râle redux*.

If the disease pass into the stage of suppuration, the dulness or flatness on percussion continues, and the moist bronchial râles, due to pus in the air-tubes, are prominent. The auscultatory signs of solidification continue, but are less marked. If abscess of the lung take place, and the patient's life be prolonged until a discharge of the pus into the bronchial tubes occurs, cavernous respiration may become well marked.

The occurrence of suppurative pleuritis is shown by the signs of that affection. With this complication, the affected side may be dilated, the intercostal depressions are obliterated, and obvious contraction of the chest may follow recovery.

Pathological Character.—Acute lobar pneumonitis, in the nosological systems of the present as of past time, is placed among the local diseases, and in regard to certain questions it has been regarded as the type of a purely inflammatory affection. This view of its pathological character is now held to be erroneous. The pulmonary affection is doubtless inflammatory; but it is the local manifestation or the anatomical characteristic of an infectious febrile disease, sustaining to the latter a relation analogous to that which the affection of the solitary and agminated intestinal follicles sustains to typhoid fever. If this doctrine be true, the proper place for the disease in the nosology is among the essential fevers. It is here retained in the class of diseases of the respiratory system for convenience of reference so long as its claims to be transferred to the division of the general diseases are not generally recognized. The name acute lobar pneumonitis denotes only a local inflammatory disease. A more correct name is pneumonic fever (febris pneumonica)—a name used by the older writers, and corresponding to the popular term "lung fever" formerly in vogue in some parts of this country.[1]

Assuming that there are grounds sufficient for adding to the list of essential fevers *febris pneumonica*, or pneumonic fever, we may define the disease as follows:

It is a fever characterized anatomically by an abundant exudative deposit in the air-vesicles of a single lobe, or of two and sometimes three lobes of the lungs, with, in general, circumscribed bronchitis and dry pleurisy. It is a fever which rapidly reaches its maximum intensity, and has a short career, the duration averaging about eleven days. It proves fatal chiefly in consequence of associated diseases, complications, or accidents, and in the mode of dying asthenia usually predominates. It depends on a cause or on causes specific in character, the nature of which is not at present established. It sometimes aborts spontaneously, and it is in some instances arrested by remedies. If not arrested, it may be favorably modified, its duration abridged, and the danger to life diminished by treatment addressed, not to the pulmonary affection, but to the fever.

Prognosis.—The prognosis in cases of acute pneumonitis will depend on the extent of lung involved, the intensity of the pyrexia, the diseases with which it may be connected as an intercurrent affection, its complications, the previous constitution of the patient, etc. These circumstances will affect the gravity and danger to such a degree that, in respect to the probable termination, cases differ as much as if they were cases of different diseases. Occurring as a primary disease, limited to a lower lobe, remaining uncomplicated, and the person affected having a fair constitution, the intrinsic tendency is to recovery; indeed, recovery is not only the rule, but the exceptions are exceedingly infrequent. Of the 133 cases which I have analyzed, in only 2 of the fatal cases was the disease limited to one lobe and not complicated or associated with other important affections. In one of these two cases the inflammation was seated in an upper lobe and eventuated in abscess. In the other case the absence of complications was predicated on the examinations during life, a post-mortem examination not having been made.

Even if more than one lobe be involved, provided the disease be primary and uncomplicated, a favorable termination may reasonably be expected in a subject not enfeebled by age or other causes. I have known recovery to take place in a case in which an entire lung was involved and the patient situated under as unfavorable hygienic circumstances as could well be imagined. In the case referred to the patient was attacked when working alone in a shanty during the winter season in the swamp near New Orleans, there being at the time two inches of water on the ground. After the attack he was unable to leave the bed for any purpose for a week, and during this time he was entirely alone. He had a quart of brandy, which he drank during the week. His habits, as he stated, were temperate. At the end of a week he was visited by some one (not a physician) who gave him thirty grains of calomel. After this he remained alone for ten days. A friend at length came to him, gave him some doses of quinia, and removed him first to his own house and afterward to the Charity Hospital of New Orleans. The physical signs on his admission into the hospital showed pneumonitis affecting the whole of the right lung, and resolution progressing. He remained in hospital six days, convalescence going on rapidly, and at the end of that time he was well enough to be discharged.

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[1] The grounds for holding this doctrine were embodied by me in a paper published in the *Transactions of the New York Medical Society* for 1877.

I have reported another case in which the patient for four days after the attack remained without food or drink in a lumber-yard, with no shelter but a pile of boards. During this time there was a snowstorm and the temperature was as low as 10° F. On the fourth day he was admitted into Bellevue Hospital. The physical signs showed solidification of the lower lobe of the left lung. On the fourth day after his admission defervescence occurred, and four days afterward he was up and dressed.

The gravity and danger, then, in cases of this disease proceed not so much from the disease *per se* as from coexisting affections and other incidental circumstances. Developed in the course of continued fever, measles, or other diseases, it may lead to a fatal termination. In aged and feeble persons it may end fatally without any coexisting disease, especially if more than a single lobe be involved. Occurring in persons affected with organic disease of the heart, it is likely to prove a serious affection. I have known it to destroy life in the first stage when developed in this connection. The pulmonary symptoms are rendered more severe and the danger is increased by antecedent emphysema.

Complications which are likely to render it fatal are pericarditis, intermittent fever, and delirium tremens. These complications invest cases with much gravity and danger, but recovery takes place in a certain proportion of cases notwithstanding their existence. An occasional complication to be included in this category is acute diffuse nephritis, as shown by the presence in the urine of albumen, tube-casts, and blood. In drunkards an occasional fatal complication is cerebral meningitis. The gravity and danger from all these complications are of course greater if the disease invade more than one lobe.

There is a liability in the course of this disease to an occurrence which claims especial notice—namely, coagulation of fibrin in the right auricle or ventricle; that is, to heart thrombus. This is of not infrequent occurrence in fatal cases of pneumonitis. It occurs especially when an entire lung becomes involved, and in cases of double pneumonitis. In such cases the obstruction to the passage of blood through the lungs, caused by the presence of the exudation, involves an over-accumulation of blood within the right cavities of the heart. The right ventricle and auricle are enfeebled by distension, and this condition, in conjunction with hyperinosis, leads to coagulation. On examination after death, in the right ventricle is a dense thrombus devoid of red corpuscles, closely intertwined with the tendinous cords and adherent to the ventricular walls. Thrombi formed *ante-mortem*, and not infrequently the immediate cause of death, are to be distinguished from those produced in the last moments of life and from *post-mortem* clots. (Vide Part I. p. 29.) Their formation sometimes may be determined with much confidence during life. In a a case presenting no symptoms which denote imminent danger a sudden change takes place for the worse; the circulation is notably disturbed, as shown by the frequency, feebleness, and irregularity of the pulse; there is a sense of the want of air; the expression is haggard and anxious; cyanosis is more or less marked; the patient falls speedily into a moribund state; and this unexpected change is not connected with an extension of the disease to a new lobe or any newly-developed inflammatory complication. Under these circumstances the formation of a heart-clot is highly probable. The probability of this accident is rendered stronger if a newly-developed cardiac murmur be discovered and referred to the right side of the heart; and it is sometimes practicable to appreciate the absence of the tricuspid valvular element of the first sound of the heart.[1]

The invasion of a second lobe in a feeble subject may be accompanied by notable prostration, amounting sometimes to a state of collapse. In the previous editions of this work the synopsis of a case was given in which the patient was apparently moribund, restoration taking place under the free administration of alcohol. I have met with cases in which a primary invasion occurring in aged persons produced such a degree of prostration and disturbance of the heart's action that death by rapid asthenia seemed imminent, life being apparently saved by free alcoholic stimulation.

As regards anatomical changes pertaining to the lungs, gangrene, abscess, and the stage of purulent infiltration render the prognosis extremely unfavorable. Yet I have known recovery to take place in cases in which the symptoms denoted the occurrence of these events.

Symptoms which are unfavorable as prognostics are the following: Frequency and feebleness of the pulse; great frequency and labor of respiration; lividity of the prolabia and face; an abundant purulent or muco-purulent expectoration; bloody, dark-colored sputa, commonly known as the prune-juice expectoration; active, violent delirium; low, muttering delirium, with prostration and subsultus tendinum, constituting the typhoid state. Cases presenting the symptoms last stated are often distinguished as cases of typhoid pneumonitis.

In the majority of fatal cases of acute pneumonitis death takes place by asthenia in combination with apnœa, the former predominating. Death purely by apnœa may occur if two or three lobes become rapidly involved, but its occurrence is rare. The occurrence of an abundant pleuritic effusion increases the danger from apnœa. So also does the occurrence of the disease in persons affected with emphysema and in cases of pertussis. Œdema of the portions of lung not affected by the pneumonia (collateral œdema) is an immediate cause of death by apnœa in some cases. This may be an inflammatory œdema, or, when general, it occurs, according to the experimental observations of Prof. Wm. H. Welch, when the left ventricle of the heart becomes disproportionately weak as contrasted with the right ventricle. In general, life is not lost in consequence of the extent of interference with respiration, but, owing to concomitant affections or other circumstances, the vital powers give way and the patient dies from exhaustion or heart-failure. This is an fact important in its bearing on therapeutical indications.

When convalescence takes place, it generally progresses until the recovery is complete. The disease never becomes chronic, unless it may be so considered when resolution is extremely slow. Exceptionally, instead of resolution being completed within a few days, it persists for several weeks, recovery at length taking place. A relapse never takes place. It is rare for phthisis to become developed as a sequel of lobar pneumonitis. When phthisis appears to be a sequel, it probably existed prior to the pneumonitis.

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[1] For cases of thrombosis of the right side of the heart, with remarks, vide *Clinical and Pathological Observations in India*, by Sir Joseph Fayrer, p. 95 *et seq.*

If the upper lobe be primarily attacked, it has been supposed that the prior existence of phthisis may be inferred. There may be some ground for this inference, but I have often known the inflammation to be limited to the upper lobe in cases in which the anterior and subsequent history furnished no evidence of phthisis.

CHAPTER V.

PNEUMONITIS (CONTINUED).

Treatment of Acute Lobar Pneumonitis.—Lobar Pneumonitis in Children.—Lobular Pneumonitis.—Atelectasis.—Suppurative Pneumonitis.—Embolic Pneumonitis.—Abscess of the Lung.—Pneumonokoniosis, Anthracosis, etc.—Brown or Pigment Induration of the Lungs.—Hyperæmia of the Lungs.—Hypostatic Congestion.—Hypostatic Pneumonitis.—Pleurodynia and Intercostal Neuralgia.

THE different stages of acute pneumonitis furnishing different therapeutical indications, the treatment of each stage is to be considered separately.

The question whether the disease may be arrested relates to the first stage. Measures which have heretofore been considered as abortive are bloodletting, cathartics, and other remedies entering into the so-called antiphlogistic method of treatment. Experience has abundantly shown that these measures cannot be relied upon for the arrest of this more than of other inflammations. Admitting that they sometimes succeed, the probability of success is not sufficient to warrant their employment under circumstances which will be likely to render their operation hurtful should they not prove successful. Full doses of quinia—that is, from 20 to 40 grains—given either at once or within eight or ten hours, may arrest the disease. This statement is based on my own experience as well as on the testimony of others. If a patient be seen in the first stage, an effort should be made to render the disease abortive by means of this remedy, inasmuch as it does no harm, aside from the annoyance of cinchonism, if it do not succeed; on the contrary, a favorable influence upon the course of the disease is produced either by its antipyretic effect or in other ways when it fails as an abortive remedy. The disease sometimes aborts spontaneously; that is, without treatment. A few instances have fallen under my observation. The probability, however, of the abortion occurring spontaneously is so slight that if in several cases it follow treatment employed for that end, it may fairly be considered as a successful result of such treatment. Rational objects of treatment, exclusive of that employed for an arrest of the disease in the first stage, are to lessen the intensity of the inflammation, to lower the temperature of the body, to relieve pain, and to promote toleration of the disease.

As regards bloodletting, irrespective of its employment as an abortive measure, the reader is referred to the general considerations presented in Chapter II. Part II. (vide p. 124 *et seq.*). It is admissible in certain cases as a palliative, and perhaps to some extent a curative measure, in view of the promptness of its operation. The circumstances which warrant its employment are—high fever, the pulse more or less resisting compression, or, in other words, arterial tension and a robust constitution. It is contraindicated whenever the fever is not high, when the pulse is frequent and weak, and when the patient is anæmic or has a feeble constitution. The relief of pain and embarrassment of breathing by bloodletting is often immediate and marked. The efficiency of this measure as a palliative is due in part to its effect on the general circulation and to the diminution of the functional labor of the lungs; but the relief may be in part explained by the effect in diminishing congestion of the portions of the pulmonary organs not inflamed (the collateral fluxion of Virchow), and also by the effect in lessening the accumulation of blood in the cavities of the right side of the heart. In most of the cases, however, in which the same ends can be secured by other means than bloodletting, the former are to be preferred. These consist in depletion by saline purgatives and in sedative remedies. After the operation of a saline purgative, if the skin be hot and the pulse frequent, tartar emetic or some antimonial preparation may be given as a nauseant sedative; but the doses should not be carried to the extent of producing marked or distressing nausea. The veratrum viride may be given under the same restriction. In this country not a few practitioners regard this remedy as possessing notable curative as well as palliative efficacy. These remedies are contraindicated by feebleness or a tendency to depression. Aconite is an effective sedative remedy, and is to be preferred to the sedatives just named whenever it be desirable to avoid the depressing effects of the latter. Opium may be given with propriety and advantage in the first stage in doses sufficient to relieve the pain and tranquillize the system. Blisters are injudicious, but dry cups, sinapisms, or stimulating liniments may be employed. Stupes or warm fomentations applied to the chest are useful. In many cases a saline purgative followed by some form of opium, the latter continued at intervals, together with soothing applications to the chest, will meet the indications pertaining to the first stage. German authors extol cold applications over the part of the chest corresponding to the affected portion of the lung by means either of napkins which have been dipped in cold water, and which are to be renewed every five minutes, or the ice-bag. It is stated that after a few hours patients are sensible of relief, and often both the respiration and the pulse become less frequent. Not infrequently the temperature of the body is notably reduced. In some cases, however, patients complain of the application, and if so it should not be continued. It by no means follows that because warm applications are useful the application of cold is not also useful, or *vice versâ;* either may have a favorable effect, the *modus operandi* of each differing from that of the other. Warm applications soothe and act as revulsives; the application of cold diminishes the determination of blood to the part and relieves pain by obtunding the sensibility.

If the fever be high, as denoted by a temperature of 103° or more, antipyretic measures are indicated, as in the diseases which are generally recognized as essential fevers. These measures are the employment of cold for the direct abstraction of heat from the body and the administration of quinine in full doses, the latter often antagonizing an excessive production of heat. Antipyrine may be substituted for quinia if the latter be not well tolerated or if it prove inefficacious. Cold may be employed by means of the bath, the wet sheet, or sponging the body. Of these different modes, the two latter are less inconvenient both for the patient and others than the first, and the last mode will in most instances suffice. The object is to reduce the temperature to as near the normal maximum as practicable. Whenever, after this object is effected, the temperature rises, cold is to be again applied. It is desirable that the employment of these measures be under the personal direction of the physician or an experienced assistant. An alcoholic stimulant should be given whenever the bath, the wet sheet, or sponging is employed. My own practical knowledge of these measures in this disease is not large, but as far as it goes it is confirmatory of their safety and usefulness.[1] They are opposed by existing popular notions; hence in private practice patients submit with reluctance at first, and the physician incurs a risk of being blamed if the cases do not do well. These difficulties are not in the way of the use of quinia and other antipyretic drugs. Quinia is the most efficient of these. It should be given in the same way as with reference to an abortion of the disease. This remedy may suffice for the antipyretic object, rendering the cold-water treatment unnecessary, and it may be conjoined with the latter. Exposing the surface of the body to cool or cold air is not without considerable antipyretic effect, and is unattended by any risk, although from prevalent ideas it seems hazardous to the minds of most persons. The axiom that patients with fever do not "take cold" is one which it is extremely desirable should become popularized. Popular apprehensions on this score often stand in the way of proper ventilation in cases of disease.

The treatment in the second stage has reference to the promotion of resolution, palliation of symptoms, and supporting the powers of the system. Blood-letting in this stage is not admissible, and depletion by means of salines is not called for. It is not an object to divert the blood from the solidified lung, for the exudation has already deprived it of blood, and the amount of exudation involves the withdrawal from the blood of a pound or more of solid matter even when the inflammation is limited to a single lobe. Hyperæmia of the lung on the unaffected side (collateral fluxion), with œdema, occurs less often, as it seems to me, than has been supposed. It is indicated by increased frequency of the respirations and dyspnœa, accompanied by the development of fine, moist râles over the unaffected side. The application of a considerable number of dry cups over the chest is the most effectual measure for relief. Experience has shown the inutility of remedies formerly employed as sorbefacients—namely, tartar emetic in large doses and mercury.

Repeated applications of the tincture of iodine will secure a sufficient amount of counter-irritation if any be desirable. Blisters are not advisable, on account of the general disturbance which they are liable to occasion and their interference with physical examinations of the chest. If pain and soreness continue in this stage, stupes with warm water only or with some stimulating liquid, as the liniment of turpentine, will afford relief. The latter will not be required if the tincture of iodine have been applied.

Opium is a valuable remedy in the second as well as in the first stage. It is indicated not only by the continuance of pain, but by vigilance, restlessness, and symptoms denoting constitutional disturbance. I have repeatedly observed a rapid and notable diminution of the frequency of the pulse and of the respirations, with refreshing sleep and a condition of comfort, to follow full doses of opium. The free use of opium does not delay the beginning or retard the progress of resolution. An accumulation of mucous secretion in the bronchial tubes contraindicates the use of opium in full doses.

Remedies to promote expectoration, as a rule, are not indicated. The use of remedies of this class is based on the erroneous idea that the matter of exudation is expectorated. Clinical observation shows that the removal of this matter may go on with great rapidity without any expectoration. The expectoration in the second stage of the disease is due to bronchitis limited to the affected lobe or lobes. The mucous products rarely accumulate in the bronchial tubes to an extent to occasion inconvenience, except as a consequence of a degree of exhaustion sufficient to render the muscular power inadequate to efficient efforts of expectoration; and under these circumstances expectorant remedies will not afford relief.

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[1] For report of cases treated by the wet sheet by the author, vide Gaillard's *Medical Journal*, March, 1881; also, *Transactions of the New York State Medical Association*, vol. ii.

Sedative remedies, such as aconite and the veratrum viride, are admissible in this stage if there be considerable or high fever without a tendency to asthenia. They should be cautiously given, so as not to incur risk of constitutional disturbance or depression. A high temperature in this as in the first stage calls for antipyretic treatment, and the same measures may be employed. These measures are indicated whenever the thermometer shows a temperature at or above 103°.

To support the powers of life is the leading general indication in the second stage. Resolution will be sure to begin and continue if the life of the patient be sufficiently prolonged. The danger is generally, not from the amount or persistence of the solidification of lung, but from failure of the vital powers before the resolution takes place. This disease belongs among those distinguished as self-limited; if uncomplicated and not attended by accidents, it runs a definite career ending in restoration, provided the powers of life hold out. These considerations, together with the results of clinical experience, enforce the importance of the supporting treatment.

The indication for supporting measures as regards urgency varies much in different cases. In general terms, it is urgent in proportion to the danger from asthenia. It should govern the treatment in cases sometimes characterized as asthenic, and whenever there are grounds for distrust of the adequateness of the vital powers to carry the patient safely through the disease. It is a serious mistake to defer supporting measures until the symptoms denote imminent danger from failure of the powers of life. If deferred so long they will probably be too late. The observing and skilful practitioner will foresee and endeavor to forestall a degree of prostration attended with imminent danger. The constitution of the patient, his previous health, and his habits are to be taken into account in judging early of the ability to sustain the disease. Other things being equal, in a warm climate patients are less able to sustain the disease than in cold or temperate climates; supporting treatment, therefore, is oftener and earlier called for in the former than in the latter. In cases which are distinguished as asthenic and typhoid the reliance for successful management must be on supporting measures. These views are the more to be impressed because it is undoubtedly true that until lately the minds of medical men have been so much occupied with the means of subduing inflammation as to overlook the fact that means employed for this end not only often conflict with those which are more important for recovery, but may be positively injurious, if not, indeed, destructive to life. The attention has been directed too much to the disease and too little to the patient.

The supporting treatment embraces tonic remedies, alcoholics, and nutritious diet. Of tonic remedies, quinia is to be preferred. Alcoholics form an essential part of the supporting treatment, as in all other diseases whenever the object is to keep the patient alive until the disease has reached the end of its career and advanced into the stage of resolution. The principle is the same as in other essential fevers; and here, as in the management of the latter, alcoholics are indicated to an extent commensurate with the danger from failure of the vital powers. In certain cases of pneumonitis, as in typhus or typhoid fever and other diseases, there is often a remarkable tolerance of alcohol, and the only guide as regards quantity is the effect as manifested by the symptoms. No abstract rules can be laid down as applicable to all cases, but careful observation must furnish the rule proper to each individual case. Here, too, as in the continued fevers, because alcoholics are vastly important in some cases it is not to be inferred that they are invariably indicated or that they can never do harm; on the contrary, if pushed to an injudicious extreme they are potent for evil, as they are potent for good when judiciously used.

As regards the circumstances under which the use of alcoholics is to be begun, they are always indicated as soon as evidence appears of any tendency to failure of the powers of life. And of this the action of the heart, as represented by the pulse and by the systolic sound over the apex, is the criterion. Feebleness, great frequency, and a pulse vibratory or thrilling but compressible, denoting increased activity but diminished power of the ventricular contractions; also, feebleness of the first sound on auscultation over the apex, and, in addition to feebleness, the shortness and valvular quality of this sound,—these are the characters which indicate supporting measures, of which alcoholics are an essential part. Given at first in small or moderate doses, the effect is to be watched, and the quantity increased in proportion to the urgency of the indication. The habits of the patient as regards the habitual use of alcoholic drinks are of course to be taken into account. Whenever the question arises in the management of a case whether alcoholics be advisable or not, it should be borne in mind that to begin earlier than they are required is far preferable to subsequent delay; for with proper care they can be suspended without any injury having been done, whereas the time lost by beginning too late cannot be regained.

Alimentation is an essential part of the supporting treatment. It is as important to nourish patients affected with pneumonitis as those affected with any febrile or other disease whenever there is danger from failure of the vital powers. The statement that patients with pneumonitis should be encouraged to take nutritious food during the whole course of the disease is based on considerable experience, and alimentation should enter into the treatment in proportion as the symptoms denote a tendency to asthenia. Milk with farinaceous substances and animal broths should form the diet, thus combining a proper variety of alimentary principles. The desires and taste of the patient may generally be trusted.

These remarks on the supporting treatment are not specially applicable to pneumonitis, but apply alike to cases of any disease the gravity or danger of which is manifested by symptoms denoting failure of the vital powers, and they will be referred to in connection with the treatment of other affections. As applied to pneumonitis, they relate chiefly to cases in which this disease is rendered grave or dangerous either by the extent of lung involved or by coexisting affections. It will be borne in mind that in a large proportion of the cases in which the disease is limited to a single lobe and disconnected from other affections there is little or no intrinsic tendency to a fatal termination. In such cases no active treatment is required, either with a view to lessen the intensity of inflammation or to support the powers of the system; patients pass through the disease satisfactorily under simple palliative measures. I have treated many cases simply with attention to hygiene. It by no means follows that because this disease exists remedies are to be employed; they are to be employed only when there are indications calling for therapeutical interference.

In cases of pneumonitis associated with intermittent fever quinia should be given promptly and in efficient doses. The paroxysms should be arrested as speedily as possible, as the patient may be placed in great danger by their repetition. In a malarial region, or if the patient have been subject to intermittent fever, it is judicious to forestall the possible development of the latter affection by moderate doses of quinia. The malarial cachexia impairs the power of resisting the disease; hence it is more likely to prove fatal in malarial regions, even when uncomplicated with intermittent fever.

Cases of pneumonitis complicated with delirium tremens call for the pretty free use of opium, together with alcoholics and a nutritious diet. Delirium and vigilance, if protracted, are likely to lead to a fatal termination. Pericarditis as a complication adds greatly to the gravity and the danger, but by perseverance in the judicious employment of supporting measures the patient may be carried safely through this combination of diseases. The occurrence of gangrene furnishes an additional indication for support. Pneumonitis occurring in connection with the continued or the eruptive fevers generally gives rise to the indications for supporting treatment.

During the progress of resolution the principles of treatment which have just been presented are applicable until the improvement in the local and general symptoms, in connection with the physical signs, denote convalescence. When convalescence is established, as already stated, there is no danger of a renewed attack. There is, therefore, no need of extreme precautions in order to prevent a liability to relapse. Experience shows that a solid, substantial diet may be entered upon as soon as the patient is fairly on the road to recovery, and that the recovery is more rapid than if the appetite be too much restrained. As a rule, ordinary wholesome, digestible articles of food may be allowed when they are desired by the patient. Permitting or encouraging the patient to sit up will be found not to retard recovery, but, on the other hand, apparently to hasten the progress of resolution. Early going out of doors is not objectionable.

If the disease proceed to the suppurative stage, purulent matter being either infiltrated or forming abscesses, the prognosis is extremely unfavorable, but perseverance in the employment of supporting measures is sometimes successful. It may be added that timely and efficient support probably affords the best security against suppuration, which happily is extremely rare.

I have for several years advised the carbonate or muriate of ammonia during the progress of the disease with a view to prevent cardiac thrombosis. Of course it is difficult to obtain clinical proof of the protective efficacy of this or any other remedy against that accident. There can be no objection to the use of this remedy on therapeutical grounds, since it does not in any manner affect unfavorably the progress of the disease. Dr. A. Patton of Indiana, in a paper advocating the free use of this remedy throughout the disease—that is, from 5 to 10 grains every three hours—states that of 96 cases in which this constituted all the medicinal treatment, in only 2 cases was the disease fatal.[1] It is probable that digitalis, given with a view to increase the power of the heart's action, is useful in preventing heart-clot. This remedy has seemed to me often signally useful when the action of the heart is feeble or irregular, and also to be of use as a nervous sedative when delirium and vigilance are prominent symptoms.

Acute Lobar Pneumonitis in Children.

Acute pneumonitis, the solidification extending over an entire lobe, sometimes involving a second lobe, and presenting after death the anatomical characters which have been described as belonging to this disease in the adult, occurs in young children, although infrequently as compared with its occurrence after this period of life. It is a much graver disease in young children than in adults, proving fatal in a large proportion of cases. From the absence of the subjective symptoms and the difficulty of obtaining physical signs the diagnosis is less easy, and the disease is not infrequently overlooked.

The attack is sometimes ushered in by a convulsion. This may mislead by directing attention to the head. Dulness, drowsiness, or stupor sometimes accompanies the progress of the disease, and these symptoms may mislead in the same way if the symptoms referable to the chest be not marked. Vomiting is not a rare symptom at the onset of the disease. The expectoration, being swallowed, cannot be observed in young children. As regards local symptoms, the disease may be latent in the child, as it not infrequently is in the adult. But in a certain proportion of cases the existence of sharp pleuritic pain is manifested by the expression, and also by the cry in acts of coughing or whenever a deep inspiration is taken. A diagnostic symptom of frequent occurrence is a moaning or grunting sound with the expiratory act. Attention to this symptom is especially important, as it points very strongly to the existence of pneumonitis. Increased frequency of the respirations and dilatation of the nostrils show the existence of some pulmonary affection compromising the respiratory function. The respirations in some cases are very frequent, numbering 40, 50, 60, and sometimes even many more, per minute. One or both cheeks may present a circumscribed flush. If the progress of the disease be unfavorable, lividity of the prolabia and face becomes marked; more or less acceleration of the pulse occurs, and the frequency in some cases is very great, amounting to from 150 to 200 per minute.

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[1] *Am. Journ. Med. Sci.*, Oct., 1870. Of 207 cases treated chiefly with this remedy by Dr. J. P. Thomas, only 3 were fatal. (Vide *Virginia Medical Monthly*, April, 1880.)

When the existence of some acute affection of the chest is declared by the symptoms, the differential diagnosis lies between primary pleuritis, capillary bronchitis, the so-called lobular or broncho-pneumonitis, and lobar pneumonitis. The first of these affections being extremely rare under five years of age, the problem is usually to differentiate the latter from the two other affections. In solving this problem the physical signs are to be relied upon, and the reliance is also upon these for the diagnosis in cases in which the symptoms denoting an acute thoracic affection are not marked. The physical signs are generally available with care and patience, notwithstanding the difficulty of exploring the chest in the young child.

The crepitant râle is oftener wanting in pneumonitis affecting the child than the adult, but it is present in a certain proportion of cases, and is of course to be sought after. This sign belongs exclusively to pneumonitis, whereas fine bubbling (subcrepitant) râles belong to capillary bronchitis. If there be doubt as regards the discrimination between the crepitant and the bubbling râles, it is to be borne in mind that capillary bronchitis is a bilateral disease, and the bubbling râles will be present in both sides; whereas in the great majority of cases pneumonitis in the child, as in the adult, is unilateral, and the crepitant râle will be limited accordingly to one side. Dulness on percussion is readily determinable in the child; and this is an important point in the differential diagnosis. Bronchial respiration and bronchophony may generally be obtained in the child by perseverance in auscultation, the cry answering for the voice. These signs do not belong to capillary bronchitis, and are rarely present in lobular pneumonitis. In short, the diagnosis is to be based on the same signs as in the adult, but patient efforts may be requisite to obtain them. As regards primary pleuritis, the signs denoting effusion are available in the child as well as in the adult, and their absence warrants the exclusion of that affection.

The treatment of acute lobar pneumonitis in children involves the same principles as the treatment of the disease in adults, with those modifications which therapeutical measures require in their application to infantile life. It is questionable whether bloodletting be ever advisable in young children, even when the diagnosis is clear in the first stage of the disease. As a rule, this measure is not to be employed, and the exceptions to the rule, if there be any, are few; depletion by salines may take its place. Antimonial preparations, if given at all, are to be prescribed with great caution, and in general other and less depressing nauseant sedatives are to be preferred. The veratrum viride, if given, should be prescribed in small doses and its effects very carefully watched. Aconite, in children as well as adults, is a useful sedative in this affection. Blisters should not be employed. A sinapism or turpentine stupes may be applied to the chest, followed by a poultice or the water-dressing. Particular attention should be given to the latter, in order to see that either the entire chest or the whole of the affected side is covered with several thicknesses of flannel and a layer of oiled muslin. The spongio-piline is a good substitute for the poultice or the water-dressing. Opium need not be withheld, but it must be given with circumspection. Finally, as much depends in severe cases, in the child as in the adult, upon the early, judicious, and persistent employment of supporting measures. In children, as in adults, under certain circumstances there is a remarkable tolerance of alcoholics. I have notes of the case of a child of a medical friend (age fourteen months), presenting the utmost gravity of symptoms, the pulse 200 and the respiration 120 per minute, in which brandy was increased to at least an ounce hourly, and under this amount the pulse fell rapidly to 124 and the respiration to 50 per minute, the carbonate of ammonia and a little morphia constituting the additional treatment. Recovery took place. This case is cited in illustration of the extent to which in some cases the employment of alcoholics may be carried. It does not follow that they are to be generally employed excessively or largely. The same rules are to be observed in their use in children as in adults, with a view, on the one hand, to secure the benefits of alcoholics, and, on the other hand, to avoid the evils of their over-use.

Lobular Pneumonitis—Broncho-pneumonitis—Atelectasis.

Lobular pneumonitis is also frequently called *catarrhal pneumonia*, from the fact that the exudation is chiefly cellular. This latter name, however, is inappropriate, because the term "catarrhal" is only applicable to inflammations of mucous membranes, and the wall of the air-cells and alveolar passages cannot be considered properly a mucous membrane; and, moreover, fibrin is sometimes present in the exudation of so-called catarrhal pneumonia as well as in that of lobar pneumonitis. The name *cellular pneumonia* employed by Virchow is not sufficiently distinctive. The terms *broncho-pneumonitis*, implying the coincident affection of the bronchi and of the air-cells, and *lobular pneumonitis*, signifying the lobular dissemination of the inflammation, are to be preferred.

Lobular pneumonitis is a secondary affection, and almost always secondary to inflammation of the smaller bronchi. It is frequent in childhood, but rare in adults, except in the debilitated and the aged. In the new-born and in very young children it is usually preceded and accompanied by *atelectasis* or collapse of a greater or less number of pulmonary lobules (apneumatosis). Atelectasis may attend lobular pneumonitis occurring later in life, but it is a less constant accompaniment. Portions of the lung-tissue in the condition of atelectasis contain no air; they are non-crepitant and sink in water. They present a bluish-red appearance through the pleura, and a more brownish-red color on cross-section. They have a smooth surface, a firm consistence, and are depressed below the level of the surrounding lung-tissue. They can be inflated by blowing through the bronchi. In many cases, but not always, occlusion of the bronchi immediately connected with the atelectatic spots can be demonstrated. The most frequent source of obstruction is an accumulation of the products of inflammation in the smaller bronchi. The air-cells connected with the occluded bronchi collapse after the absorption of the contained air. Microscopical examination of the collapsed lobules shows distension of the pulmonary capillaries with blood, and the presence of granular, desquamated epithelial cells in the collapsed alveoli. Atelectasis of portions of the lungs may be a *congenital* condition in the new-born and in infants a few days or weeks old, the affected lobules having never been inflated, in consequence either of weakness of the respiratory efforts or of obstruction of the bronchi by mucus, meconium, etc. *Acquired atelectasis* is far more frequent in children than in adults. The name atelectasis is also sometimes applied to the condition called carnification of the lungs, the air having been expelled by pressure from without as by an extensive fluid accumulation in the pleural cavity. Atelectasis of whatever form is more frequent in the lower lobes than in the upper. Single circumscribed lobules may be affected, or even a whole lobe. When there is extensive atelectasis the remaining parenchyma of the lung is usually in the condition of vicarious vesicular emphysema.

There has been much controversy as to the relations between atelectasis and lobular pneumonitis. Formerly, writers regarded atelectasis as congenital pneumonia, and since the true nature of the condition has been recognized it has usually been held that collapse of the air-cells in itself leads to the development of lobular inflammation. It is probably not correct to regard either acquired pulmonary collapse or a persistence of the fœtal state of the lungs as alone a cause of inflammation. We may consider that lobular pneumonitis occurs only when there is present within the alveoli an irritant capable of inducing inflammation. Most foreign particles, and particularly the stagnating secretion of the inflamed bronchial mucous membrane, are irritants capable of exciting bronchitis and lobular pneumonitis by entrance into the small bronchi and the air-cells, whether or not they completely obstruct the bronchi and lead to atelectasis.

The pathological changes occurring in lobular pneumonitis have been divided into the same three stages as in lobar pneumonitis—namely, first, congestion or engorgement; second, red hepatization; and third, gray hepatization. Such a division is, however, less important and well defined in lobular than in lobar pneumonitis. In a typical case of lobular or broncho-pneumonitis disseminated throughout both lungs are small firm spots varying in size from a pea to a pigeon's egg. The color may be brownish-red, reddish-gray, gray, or yellowish, and the periphery is usually darker than the centre. The surface is lustreless, smooth or slightly granular, and somewhat elevated above the surrounding lung-tissue. The consolidated pulmonary substance is non-crepitant and sinks in water. When the solidified lobules are situated at the surface of the lung the overlying pleura is coated with a delicate layer of fibrin. Usually, drops of muco-pus can be squeezed out of the smaller bronchi. The microscopical examination of the solidified lobules shows the air-cells to be filled, though less distended than in lobar pneumonitis, with pus-cells, epithelial cells, and some granular matter in varying proportion. In some cases the pus-cells predominate; in other cases, large granular cells with vesicular nuclei, thought to be alveolar epithelial cells. Red blood-corpuscles are usually present, but not in such number as in lobar pneumonitis. The presence of large numbers of pus and epithelial cells in the alveoli, the comparatively small number of red blood-corpuscles, and the absence of fibrin may be considered to be the characteristics of the exudation of broncho-pneumonitis as distinguished from the lobar form. Sometimes, however, fibrillated fibrin may be found in the exudation of lobular pneumonitis, and in fact, as far as the inflammatory products are concerned, there is every grade of transition between a typical, purely cellular exudation and the fibrino-cellular and almost hemorrhagic exudation of lobar pneumonitis. Mucus and cells are sometimes inspired from the bronchi into the alveoli. The walls of the inflamed bronchi are often infiltrated with leucocytes. The inflammation may extend by continuity to the air-cells immediately adjacent to these inflamed walls. There may be some dilatation of the bronchi. Various forms of bacteria have been found in the inflammatory exudation in the bronchi and air-cells, and doubtless in many cases bacteria stand in a causative relation to the disease.

There is great variety in the extent of lung involved in the inflammatory process. The pneumonitis may be confined to a few lobules, as, for instance, those surrounding a hemorrhagic infarction or a new growth; there may be countless nodules of consolidation scattered throughout both lungs, or even all the lobules of a lobe may become involved. In the rare cases of lobar consolidation from lobular pneumonitis the fact that the lobules are not simultaneously but successively involved gives to the surface a peculiarly variegated appearance different from that observed in lobar pneumonitis. The surface is also less granular than in the latter disease. As to the origin of the cells which fill the alveoli, it is customary to regard the pus-cells as emigrated white blood-corpuscles. Whether the alveolar epithelium play an active part in the inflammatory process is a matter of dispute. There is no proof of the production of pus-cells from proliferating epithelial cells, and, in those cases in which the number of large epithelium-like cells in the alveoli seems greater than can be accounted for by a mere desquamation of the epithelial lining, it is not necessary to assume active proliferative changes in the epithelium, as it is abundantly proven that wandering leucocytes can become transformed into epithelioid cells.

The most frequent termination of lobular pneumonitis is in resolution. As in lobar pneumonitis, the cell-elements undergo fatty degeneration, and by transudation of serum a kind of emulsion is formed capable of absorption. When the broncho-pneumonia persists for a long time, the alveolar walls usually become thickened by a new growth of connective tissue.

It has been held by most authorities that the inflammatory products of lobular pneumonitis may undergo caseous metamorphosis which leads to the development of pulmonary phthisis. We now know that in such cases infection with the tubercular virus exists. It is probable that in some cases the existence of broncho-pneumonia favors the lodgment and development of the tubercle bacilli. It is, however, an error to confound with ordinary broncho-pneumonia the lobular caseous pneumonia so often found in phthisical lungs. The latter affection is anatomically and etiologically a distinct disease.

CLINICAL HISTORY.—Inasmuch as a bronchitis affecting both sides (bilateral) precedes and accompanies the collapse of lobules in lobular pneumonitis, the symptoms of that disease are always present. As will be seen in treating of Bronchitis in a following chapter (Chapter IX.), the symptoms attributable to the bronchial inflamation vary according to its extension into tubes of small size. In addition to cough and expectoration, increase in the frequency of the respirations, dyspnœa, and cyanosis may be accounted for by the bronchitis alone if tubes of small size become involved (capillary bronchitis). These symptoms are more marked in proportion to the number of lobules collapsed and inflamed. The symptoms are chiefly due to the pneumonitis if the bronchitis be limited to tubes of large or medium size.

The bronchial inflammation may be acute or subacute. The pyrexia is proportionate to the acuteness of the bronchial inflammation and to the extent of lung affected. This will, therefore, vary considerably in different cases. The symptoms relating to the frequency and other characters of the pulse offer variations dependent on the intensity of the fever and the obstruction to the passage of blood through the pulmonary vessels. Corresponding variations in other constitutional symptoms are incident to the fever and disturbance of the circulation. It follows that broncho-pneumonitis may be a disease of great severity and danger, or it may be comparatively devoid of gravity. Some cases are characterized by notable fluctuations during the course of the disease, owing to an increase at different periods of the number of lobules implicated. The symptoms on successive days may fluctuate, collapsed lobules becoming inflated or an additional number of lobules becoming affected.

DIAGNOSIS.—The diagnosis relates to two factors as regards the symptomatic phenomena—namely, the bronchitis and the collapsed or inflamed lobules. A third factor is the occurrence of emphysematous lobules (lobular emphysema). The physical signs denoting the bronchitis are the moist bronchial or bubbling râles, which are coarse, fine, or subcrepitant according to the size of the tubes in which they are produced. Râles varying in coarseness or fineness may be combined. The collapsed and inflamed lobules may furnish no distinctive signs if in the form of small nodules, disseminated, and about equal in number on the two sides. Broncho-vesicular or bronchial respiration, increased vocal resonance, and bronchophony, together with dulness on percussion within a circumscribed space on one side or on both sides, may be found wherever a sufficient number of lobules in close proximity to each other are affected so as to give rise to solidification extending over a considerable area. Lobar pneumonitis is excluded by the fact that the solidification does not extend over an entire lobe. On the other hand, the signs of solidification show something more than bronchitis, this disease alone not giving rise to these signs. If no signs of solidification be discovered, it is difficult or impossible to differentiate broncho-pneumonitis from bronchitis affecting tubes of small size. In severe cases inspection shows retraction of the lower part of the chest on both sides with the acts of inspiration, and sinking in of the soft parts above the clavicles, as in cases of capillary bronchitis. Emphysematous lobules of the upper lobes will give rise to a vesiculo-tympanitic resonance on percussion.

CAUSATION.—Age is to be regarded as the most important element in the causation. The fact that in a very large majority of cases the subjects are either infants or children or persons advanced in years, is probably to be explained by the much greater tendency in bronchial inflammation to extend to the smaller bronchi in the two extremes of life. The affection occurs not infrequently as a sequel of the eruptive fevers, rubeola, scarlatina, or small-pox; also of whooping cough, diphtheria, and, more rarely, of the continued fevers. It may be produced traumatically in persons of any age by the inhalation of chlorine or other gases, of vapors, and of irritating substances of various kinds. Exposure to the latter causes may be incident to certain occupations. (See *Pneumonokoniosis*, p. 180.) The pneumonitis which is liable to accompany exudative laryngitis (croup) in children is a broncho-pneumonitis. Its occurrence in cases of whooping cough is not infrequent. In the insane, the delirious, and the greatly enfeebled, with obtunded sensibility of the air-passages, foreign particles, such as bits of food, the buccal fluids, etc., are likely to be inspired into the small bronchi and air-cells, giving rise to lobular pneumonia, which in these cases may be of a rather diffuse character. This is the aspiration pneumonia (Schluck-pneumonie) of German writers. Not infrequently the inspired foreign particles are so irritating that the inflamed foci terminate in abscess, and occasionally they lead to gangrene.

TREATMENT.—The therapeutic indications have reference, first, to the bronchitis, and second, to the inflammation and the collapse of lobules. The former are the same as in cases of bronchitis irrespective of the lobular conditions, and they will be considered in a following chapter. The prompt and efficient treatment of bronchitis, especially in the young and old, is important by way of preventing the collapse and inflammation of lobules. Energetic inspirations are important in order to prevent collapse of lobules and for the inflation of those already collapsed. For these objects the air which the patient breathes should be cool or even cold, and the vital powers should be sustained by nutritious stimulation and alcoholics. Jürgensen's method of exciting deep inspirations by the cold affusion or a douche directed upon the nucha may be employed. If there be hyperpyrexia, it is to be combated by the cold bath or by sponging the surface of the body, as in other diseases whenever antipyretic treatment is indicated, and by quinia, antipyrine, or other antipyretic remedies. Alcoholics and digitalis are indicated by feebleness of the heart's action, as in cases of acute lobar pneumonitis. Emetics are indicated in children by accumulations in the bronchial tubes, which the patient does not remove by voluntary expectoration. The emetic selected should not produce prolonged nausea or much prostration. The dorsal decubitus should not be constantly maintained, changes of position being important as preventive of collapse of lobules.

Congenital atelectasis in the newly born is denoted by rapid, short acts of breathing, feebleness of the cry, difficulty in nursing from want of breath, and cyanosis. The absence of cough and bronchial râles suffices to exclude bronchitis. Congenital malformations of the heart are excluded by the absence of their physical signs. The respiratory murmur is feeble, especially at the lower and posterior portions of the chest. The lower part of the chest in front is retracted in the act of inspiration.

Inasmuch as the deficient expansion of the lungs is due to weakness of the respiratory acts, the strength of these should be increased by the momentary application of cold water to the chest, exposing the body for a few moments to cold air, and exciting in various ways crying efforts. Care should be taken to free the nostrils and throat from accumulations of mucus.

Suppurative Pneumonitis—Embolic Pneumonitis—Abscess of the Lung.

Suppurative pneumonitis is characterized by the formation of pus and breaking down of lung-tissue. Sometimes the pus infiltrates the walls of the bronchi or of the blood-vessels, or follows bands of interstitial tissue (dissecting pneumonia). The most important form of suppurative pneumonitis is that which results in the formation of abscess. The abscess-cavity usually varies in size from that of a pea to that of a hen's egg, but in some cases the abscess takes in the greater part or the whole of a lobe. The shape of the cavity may be round or irregular. The walls of the abscess are usually ragged. In cases of long standing a fibrous capsule forms around the abscess.

As has already been mentioned, lobar pneumonia and lobular pneumonia rarely terminate in abscess. Abscesses may be produced by the inspiration of foreign irritating particles, as in many cases of aspiration pneumonia (p. 177). Wounds of the lung, especially when attended by the lodgment of foreign bodies, may give rise to abscess. In new-born children there is a form of septic pneumonia which frequently terminates in abscess. In this form of pneumonia an enormous number of micrococci are found in the air-cells. In chronic phthisis suppurative processes in the walls of cavities, and sometimes in other parts of the lung, may be an important pathological element.

The most frequent form of suppurative pneumonitis is that produced by infectious emboli, which cause metastatic abscesses in the lungs. The source of the emboli is usually to be sought in thrombi formed in veins leading from some centre of infection, such as external wounds, inflammation of the uterine veins in puerperal women, bed-sores, abscess of the middle or external ear and of other situations, and ulcerative endocarditis. The embolus brings with it some poison which acts as an intense inflammatory irritant. It is not always possible to find the primary thrombus, nor can an embolus in all cases be demonstrated. It is probable that the poison may be carried by the blood in a finely molecular form. Metastatic or pyæmic pulmonary abscesses vary in number from one to several hundred, and in size from a pin's head to those involving an entire lobe. They may or may not be accompanied by the mechanical effects of emboli; that is, by hemorrhagic infarctions, which are to be described subsequently.

The pleura covering an abscess is inflamed, and there is often a general pleuritis, which may also be of a purulent character. The abscess may rupture into the pleural cavity.

Micrococci are always present in the pus. The same micro-organisms which cause suppuration elsewhere also occasion suppurative inflammation of the lung. (See Part I. p. 36.)

The most important aid in the DIAGNOSIS of abscess of the lung is the examination of the sputum. The color of the sputum is usually yellow, like that of ordinary pus, but it may be green or brownish. The odor is usually somewhat offensive, but it has not the fetor of gangrene. Of the utmost importance is the presence in the sputum of particles of lung-tissue, which can often be detected even with the naked eye. These particles are found by microscopical examination to contain elastic fibres (which are usually absent or scanty in gangrene of the lung), fatty crystals, amorphous pigment, and especially hæmatoidin crystals. Micrococci, which do not stain blue with iodine like certain ones in gangrene, are present in large number. The physical signs of a cavity may be detected if the abscess be of sufficient size, but these signs are not in themselves distinctive of abscess. Other symptoms, such as pain, dyspnœa, pyrexia, rigors, and sweats, are often present. In pyæmia it is generally impossible to disconnect the general symptoms which are indicative of the pulmonary affection from those which belong to the clinical history of pyæmia. In pyæmia it is rare that the different foci of inflammation in the lungs involve areas sufficient to give rise to characteristic physical signs.

The PROGNOSIS is almost necessarily fatal in pyæmic pulmonary abscesses. In other forms of pulmonary abscess, such as that secondary to pneumonia, the prognosis is comparatively favorable—more favorable than in gangrene of the lungs. The affection, however, is in all cases grave.

The TREATMENT is to be directed especially to sustaining measures. Quinine may be given in large doses. The air of the room should be kept pure, and inhalations of antiseptic vapors, such as thymol or carbolic acid, may be tried. A case of large abscess of the lung which was first diagnosticated as empyema was treated successfully by Teale by means of incision and free drainage.[1]

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[1] *Lancet*, 1884, vol. i. No. 2.

Pneumonokoniosis—Anthracosis—Siderosis, etc.

Under the generic name pneumonokoniosis, proposed by Zenker, are included the various affections of the lung produced by the inhalation of dust-like particles. A considerable number of such inhalation-diseases is already known. The one longest recognized is the lesion caused by the inhalation of coal-dust. A moderate amount of black coal-pigment is found in the lungs of all adults, more especially of those living in cities; but in the lungs of those who have been exposed for a long time to a dense atmosphere of coal-dust the carbon-particles accumulate to such an extent as to cause a lesion called *anthracosis* of the lungs. This lesion is produced especially in coal-miners. The particles of coal are carried by the current of inspired air into the air-vesicles or alveoli. Thence, either free or enclosed in cells, which are probably emigrated white blood-corpuscles, they make their way into the alveolar septa, and are still further conveyed through the lymph-spaces and lymph-vessels into the interlobular and peribronchial connective tissue. Some of the particles reach the bronchial glands, and exceptionally may even be carried to the cervical or abdominal lymphatic glands. The lungs and bronchial glands are black; the microscope shows the coal-pigment to be chiefly in the interlobular connective tissue and in the bronchial glands, but it is also present inside of cells in the alveoli and in the walls of the air-vesicles. Extensive anthracosis may exist without any changes in the lung other than chronic bronchitis and the presence of some large cells containing pigment in the alveoli. Frequently, however, it is combined with other chronic inflammatory changes, particularly with chronic interstitial pneumonitis. Dense fibrous bands and nodules deeply pigmented are found. The walls of the alveoli are thick, and their lumina obstructed or even obliterated by the new growth of fibrous tissue. Firm pleuritic adhesions are present. Combined with these chronic interstitial changes bronchiectatic and ulcerative cavities may be present. Anthracotic lungs may be the seat also of chronic pulmonary tuberculosis, in which case tubercles and tuberculous pneumonitis will be found.[1] To pulmonary anthracosis with chronic interstitial inflammation, with or without the presence of tubercles, the name coal-miner's phthisis is applied. The coal-particles, when of sufficient size, can be recognized as such under the microscope; often, however, they are not to be distinguished from melanotic pigment derived from the blood. The coal-particles are often present in the expectoration of those affected with anthracosis pulmonum.

The term *siderosis pulmonum* denotes the accumulation in the pulmonary structures of the oxide of iron. This oxide forms a very fine, brownish-red dust. It is inhaled by workmen employed in dyeing, polishing mirrors, and other mechanical arts in which it is used. Accumulating in the lungs, it causes a reddish pigmentation. Its morbid effects were first studied by Zenker.[2] They are essentially the same as in anthracosis, the color being red instead of black.

Chalicosis pulmonum is the name given to the pulmonary changes induced by the inhalation of stone-dust. It is also called stone-cutter's phthisis. The chemical examination of these lungs shows the presence of an excess of silica. The changes are similar to those of anthracosis and of siderosis, with the exception of the greater disposition to the formation of nodules and diffuse masses of fibrous tissue. These have a gray centre and dark periphery. The foreign particles can be recognized microscopically, but not so readily as in the previous affections.

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[1] It is claimed by recent observers that tubercle bacilli are usually absent in coal-miner's phthisis, in which case the process is not to be regarded as tuberculous. According to Cohnheim, coal-particles, as such, cause no irritative changes in the lung, and when inflammatory changes are present irritating substances are inhaled with the particles of coal.

[2] *Deutsches Archiv f. klin. Med.*, Bd. 2, p. 116.

Various other forms of pneumonokoniosis have been described, such as those due to the inhalation of particles of tobacco, of cotton, of grain, of various metals, etc.

The clinical history of the different varieties of pneumonokoniosis is that of bronchitis, which is generally chronic, associated, in a certain number of cases, with fibroid phthisis.

The removal of the patient from the continued operation of the cause is of course the first object of treatment. In other respects the indications relate to the diseases which are incident to the pneumonokoniosis.

Brown or Pigment Induration of the Lungs.

The lesion thus entitled, described by Virchow in 1847, is referable to the long-continued passive congestion incident to valvular disease of the left side of the heart, particularly to obstruction and to regurgitation at the mitral orifice. Lungs thus affected do not collapse on opening the chest as much as when they are healthy. They are heavy, inelastic, dry, crepitating but little on pressure, of a leathery consistence, and they have a peculiar reddish-brown, yellow, or salmon color. On section, reddish or brown spots may be seen, due to ecchymoses. The microscope shows that the essential morbid condition is a dilatation of the pulmonary capillaries, loops of which often project far into the alveoli and partially obstruct their lumina. The air-cells are still further obstructed by the presence of large epithelial cells containing yellow or brown pigment, which cells may accumulate in sufficient number to fill the alveoli. Pigment is also found within the capillary vessels, in the capillary walls, and in the interstitial tissue, both free and enclosed in cells. The pigment is derived from red blood-corpuscles which escape through the capillary walls by diapedesis, in part also by rupture of the walls. The walls of the air-cells and the interstitial tissue often appear hypertrophied. Rindfleisch describes also an hypertrophy of the smooth muscle-fibres of the pulmonary parenchyma in this affection.

The symptoms denoting the lesion are not definite, but it must increase the dyspnœa which is connected more directly with the cardiac disease.

Hyperæmia of the Lungs—Hypostatic Congestion—Hypostatic Pneumonitis.

Active congestion or hyperæmia of the lungs, irrespective of inflammation, is rarely if ever a primary morbid condition, and is hardly to be considered as an individual affection. It may be produced by increased power of the heart's action, arising either from transient causes, such as the ingestion of alcoholic stimulants and mental emotions, or from hypertrophy of the right ventricle. It may be caused by the inhalation of stimulating or irritating vapors. Whenever the circulation in a considerable portion of the lungs is interrupted, more or less abnormal determination of blood must take place in the portions to which the flow of blood is unobstructed, as in cases of emphysema, lobar pneumonia, pleuritis, etc.

Passive hyperæmia or congestion is of more frequent occurrence, but is never primary or idiopathic. It is an effect of mitral cardiac lesions causing either obstruction or regurgitation, or both combined. It is also consequent on feebleness of the action of the heart, especially of the right ventricle, as in cases of dilatation or of fatty degeneration. In cases of general dropsy it occasions a disproportionate amount of hydrothorax. It gives rise to brown or pigment induration of the lungs. (See p. 181.)

Passive hyperæmia occurring in the dependent portions of the lungs is called hypostatic congestion. The conditions which favor the production of hypostatic congestion are enfeebled heart's action and the maintenance of the body in one position for a long time. It is met with in acute infectious diseases, in the aged, and in the course of chronic diseases which occasion general debility: The higher grades of hypostatic congestion result in a transudation of bloody serum. The condition of lung thus produced has been called *splenization*. The affected lung-tissue is of a dark-red color, crepitates less than in health, and upon section a large quantity of blood and serum oozes out. In prolonged or in severe hypostatic congestion the so-called hypostatic pneumonia is frequently developed. It has been disputed whether this is to be regarded as an inflammatory affection or not. It seems to involve a low degree of inflammation. The lung-tissue is dark-red, friable, non-crepitant, and sinks in water. The whole lower lobe or only portions of it may be implicated. Microscopical examination shows the pulmonary capillaries widely distended with blood, and the air-cells filled with blood-corpuscles, desquamated epithelial cells, and sometimes fibrin. The inflammatory condition is more analogous to certain forms of lobular pneumonitis than to lobar pneumonitis.

Congestion of the lungs, whether active or passive, compromises the respiratory function—*first*, by diminishing the capacity of the air-cells; and *second*, by lessening the quantity of decarbonized and oxygenized blood returned from the lungs to the systemic circulation. The respirations are increased in frequency, and a sense of the want of air, or dyspnœa, is felt, *cæteris paribus*, in proportion to the degree and extent of the congestion. As regards physical signs, congestion, if marked or considerable, undoubtedly causes a certain amount of dulness on percussion; but this is not determinable if both lungs be alike congested, or in cases in which congestion of one lung follows certain affections in the other side of the chest, as in pleurisy with effusion, pneumohydrothorax, etc. Congestion also undoubtedly diminishes the intensity of the respiratory murmur; but if the murmur be changed in character—that is, if it be bronchial or broncho-vesicular—there is solidification from some cause. It is not probable that simple congestion gives rise to adventitious sounds or râles, nor does it occasion any vocal signs.

The TREATMENT of active hyperæmia must have reference, of course, to the causative conditions and the circumstances under which it occurs. The disturbance of respiration, not only when the hyperæmia is bilateral, but when unilateral, as when one lung is consolidated or compressed, may be so great as to call for venesection. In most cases, however, dry cups applied to the chest, revulsives, and cardiac sedatives suffice for relief.

In hypostatic congestion, on the other hand, measures to increase the power of the heart's action are indicated—namely, digitalis, alcoholics, and alimentation. As a preventive measure in cases of prolonged disease, especially if the patient be feeble, constant recumbency on the back is to be avoided; the position of the body should be changed at intervals. Deep inspirations from time to time should be enjoined. In this way hypostatic pneumonitis may be prevented. The occurrence of the latter in different diseases not infrequently contributes to a fatal termination. In all diseases accompanied by much prostration or of long duration, or when the subjects are feeble, important objects of treatment are the prevention and removal of hypostasis in the dependent portions of the lungs. These objects are often of prominent importance in cardiac diseases.

Œdema of the lungs will be considered under a separate heading. (Vide Chapter XI.)

Pleurodynia and Dorso-Intercostal Neuralgia.

A brief consideration of these affections is included in this chapter, because, from similarity as regards pain, they are liable to be confounded with either acute pneumonitis or pleuritis. Prior to the employment of physical exploration this mistake was by no means uncommon, and it is still made by those who depend on symptoms alone. The term *pleurodynia* is applied to a painful affection of the muscles entering into the composition of the thoracic walls. *Intercostal or dorso-intercostal neuralgia* denotes a neuralgic affection of the intercostal nerves.

Both affections may be characterized by pain resembling that of either acute pneumonitis or pleuritis; that is, a lancinating pain felt especially in the act of inspiration. They may be accompanied by a dry cough which is acutely painful. In case of pleurodynia there may be pyrexia, and this may accidentally exist in cases of intercostal neuralgia. How are these affections to be discriminated from inflammation of the pleura either primary or occurring as a complication of inflammation of the pulmonary parenchyma?

Intercostal neuralgia, as a rule, is not accompanied by pyrexia. But this affection has a diagnostic criterion which is readily available. It consists in the existence of tenderness usually in three isolated points—namely, behind, near the dorsal vertebræ; laterally, in one, two, or three intercostal spaces; and anteriorly, in one or more intercostal spaces near the sternum or over the epigastrium. Sometimes tenderness exists in only two of these situations. The tenderness is frequently confined to a very limited space in each situation, a space perhaps small enough to be covered by the finger. On the opposite side of the vertebræ, and between the tender points in the three situations, pressure or percussion is well borne. In addition to this diagnostic test, physical exploration fails to discover the evidence of either pleuritic or parenchymatous inflammation—namely, in primary pleuritis a friction murmur and the signs denoting pleuritic effusion, and in pneumonitis the crepitant râle and the signs of solidification. The affection occurs much oftener on the left than on the right side. Both sides may be affected, but this is extremely rare.

Pleurodynia lacks a similar positive test; that is, the tenderness is not limited to isolated points, but is more or less diffused. The diagnosis, therefore, must rest on the exclusion of intercostal neuralgia by the absence of the evidence just stated, together with the absence of the signs both of pleuritis and pneumonitis. Pain in pleurodynia and intercostal neuralgia is more marked, as a rule, in movements of the body than in the respiratory movements, the reverse being true of the pain in pleuritis and pneumonitis. This remark applies more especially to pleurodynia. The tenderness in both affections varies, being sometimes slight and sometimes exquisitely acute.

Of the two affections, pleurodynia is much the less frequent. Before the diagnostic criterion of intercostal neuralgia had been pointed out by Bassereau and Valleix, cases of this affection were doubtless often considered as rheumatic, and hence cases of the neuralgic affections have apparently increased of late at the expense of pleurodynia. Intercostal neuralgia is of frequent occurrence. Cases are often met with both in private practice and among patients treated at hospitals and dispensaries, but more especially the latter. The poorer classes are more liable to it than those in comfortable circumstances. It occurs oftener among females than males. Persons affected are frequently anæmic. It is liable to be developed during the progress of pulmonary phthisis. It occurs especially as a sequel of intermittent fever.

The degree of suffering from these affections varies much in different cases. They are sometimes so slight as merely to occasion annoyance, but in some cases the pain is sufficiently severe to shorten the inspirations, thus rendering the respirations frequent, as in acute pleuritis, and occasioning great distress. In the neuralgic affection exacerbations of pain are frequently experienced. They vary greatly in different cases, as regards duration, occurring sometimes as transient affections, but in some cases persisting for a long period. Pain and soreness in the chest are sometimes assumed by malingerers, but a ready proof of the reality of the affection is the diagnostic test—namely, the limitation of the tenderness to the three points on one side of the chest. This proof is reliable, unless the malingerer have had shrewdness enough to discover that this test is relied upon for the diagnosis.

The treatment of both affections embraces measures to relieve pain if it be severe or considerable. Opium may be required internally or hypodermically for this object. Local applications may suffice if the pain be slight or moderate; for example, liniments containing chloroform or aconite, or the belladonna plaster. In cases of intercostal neuralgia quinia is an effective remedy, whether the affection be a sequel of intermittent fever or not. The quinia should be given in doses sufficient to occasion slight cinchonism, and continued in such doses for one or two weeks. If anæmia exist, this condition claims appropriate treatment. The citrate of iron and quinia or the tincture of the chloride of iron may be prescribed with reference to this condition. In obstinate cases flying blisters are useful. Valleix considers these as most likely to prove effective. The application of dry cups will sometimes afford relief.

CHAPTER VI.

PULMONARY PHTHISIS.

Anatomical Characters; Clinical History; Pathological Character; Causation.

THE discovery by Koch, in 1882, of the bacillus tuberculosis has elucidated not only the etiology, but also the pathological anatomy, of pulmonary phthisis. The proof that the tubercle bacillus is the specific cause of tuberculosis may be considered complete. This proof has established Laennec's doctrine of the unity of phthisis—a doctrine which has been maintained in all previous editions of this work. In the presence of the tubercle bacillus we possess a certain criterion of the tuberculous nature of a morbid process. We now know that the characteristic inflammatory lesions of phthisis are as much the result of the tubercle bacillus as is the tubercle granulum itself.

ANATOMICAL CHARACTERS.—In most cases of pulmonary phthisis all of the structures which compose the lungs are the seat of morbid changes. There are lesions of the bronchi, of the alveolar passages and air-cells, of the blood-vessels, of the lymphatic vessels, of the interstitial connective tissue, and of the pleura. The characteristic lesions of phthisis are tubercles and inflammatory processes with a tendency to caseous degeneration. Other lesions which are often present are ordinary inflammations, congestion, emphysema, atelectasis, and œdema. Another morbid change of great importance is the formation of cavities. The multiplicity of lesions and of structures involved, and the differences in different cases in the combination and relative preponderance of these lesions, render the morbid appearances so manifold that it may be said that no two cases of phthisis are exactly alike.

The dominating element in the pathological anatomy of pulmonary phthisis is *tubercle*. For an account of the histological structure and the nature of this product the reader is referred to Part I. of this work (p. 47). Tuberculous nodules or granules are to be found, either with the naked eye or with the microscope, in the majority of cases of phthisis. When young, these nodules are gray or translucent, but they oftener appear opaque and yellowish, having undergone partial or complete caseous degeneration. Sometimes they are transformed into fibrous tissue, and then they are dense in consistence and grayish or slate-colored. The ultimate submiliary granula coalesce to make small and large nodules, varying in size from a millet-seed to a pea, and often larger. A favorite and early situation for the formation of miliary tubercles is in the walls of the small bronchi, especially in the terminal bronchioles where they open into the alveolar passages. Tubercles may be found in any part of the lung—namely, in the walls of the bronchi, of the blood-vessels, of the lymphatic vessels, in the interlobular and interalveolar tissue, in the air-cells, and in the pleura. Many tubercles are composed of groups of air-cells filled with epithelial and lymphoid cells with or without giant-cells. The walls of these air-cells are often, but not necessarily, thickened by an accumulation of epithelioid and lymphoid cells. In the majority of cases tubercles are formed primarily at the apex of the lung. Sometimes the same tissue which composes tubercles occurs, not in a nodular, but in a diffuse form.

In acute miliary tuberculosis, which will be described subsequently, miliary tubercles are found scattered throughout both lungs. In these cases the tubercle bacilli are conveyed to the lungs by the blood-current. In many cases of phthisis an eruption of miliary tubercles is found for a variable distance around a cheesy focus or an old tuberculous nodule. Here the tubercle bacilli are conveyed by the lymphatics. The usual mode of access of the tubercle bacilli to the lungs is by the respiratory passages. In this mode of access the first effect of the bacilli is to cause the production of tuberculous nodules in the air-cells, the alveolar passages, and the terminal bronchi, where the bacilli lodge.

The production of *cheesy pneumonia* is an effect of the tubercle bacilli not less important than the production of tubercles. This form of pneumonia has been called also tuberculous pneumonia, desquamative pneumonia, and infiltrated tubercle. The air-cells are filled with epithelioid cells, lymphoid cells, usually but not always fibrin, and sometimes red blood-corpuscles. The alveolar walls are frequently infiltrated with cells. The solidified portions of lung are at first moist and reddish-gray in color; later they become gray and dry, and finally cheesy. In rare instances caseous pneumonia involves a whole lobe; usually it is lobular in distribution. The smaller bronchi are generally involved. They contain caseous plugs and have thickened caseous walls (caseous peribronchitis).

Inasmuch as tubercles are often formed partly or wholly by accumulations of cells in the alveoli, it is manifestly difficult, and after cheesy degeneration has ensued often impossible, to draw any sharp line of distinction between tubercles and foci of cheesy pneumonia. This distinction, however, is less important than was formerly supposed, inasmuch as the tubercle bacilli are present in the caseous pneumonia as well as in the tubercles, and are the cause of both processes.

In most cases of rapid or the so-called galloping *phthisis* (phthisis florida) more or less extensive patches of caseous pneumonia form the greater part of the lesions. Tubercles, however, are usually present at the same time. When a large number of tubercle bacilli gain access to the lung through the respiratory passages, as often happens in children by the bursting of a caseous bronchial gland into a bronchus, then caseous pneumonia rapidly develops, and involves a large portion of the lung at once. A similar result may follow when a cheesy focus or a cavity in any part of the lung opens into a bronchus, and thus allows a large number of bacilli to be inspired into other bronchial tubes.

Changes in the blood-vessels of the lung are of importance in the pathology of phthisis. Tubercles are non-vascular structures, as has already been explained. The pre-existing vessels, where the tubercles develop, become obliterated, partly by thrombosis and partly by an accumulation of epithelioid and lymphoid cells in their lumen. A similar process of obliteration occurs when tuberculous pneumonia undergoes cheesy degeneration. The blood-supply of the lung is furthermore greatly impeded by an inflammatory thickening of the inner coat of many of the small and medium-sized arteries, by which process the lumen of the vessel is gradually encroached upon, and may be finally obliterated. This process is called *endarteritis obliterans.* Endophlebitis obliterans is also frequent. Tubercles may be found in all of the coats of the blood-vessels. Tubercles, and especially cheesy masses which invade the inner coat of the pulmonary veins, are of grave significance, for in this way the tubercle bacilli may enter the blood-current and be transported to all parts of the body, causing a general miliary tuberculosis. The obstructions to the blood-current which have been described undoubtedly favor the cheesy degeneration of tubercle and of pneumonic products, but probably to a much greater extent the cheesy degeneration is a direct effect of the action of the tubercle bacilli. This cheesy degeneration, as has already been mentioned (p. 48), is a form of coagulation necrosis.

Mention has already been made of the presence of tubercles and of cheesy inflammation in the coats of the bronchi. The mucous membrane of the bronchi is always inflamed in phthisis. Ulcers, usually of tuberculous origin, are frequent. The various forms of *peribronchitis*, called tuberculous, caseous, purulent, or fibrous, are sufficiently explained by these terms. Some form of peribronchitis is a constant attendant of phthisis.

Inflammation of the interstitial tissue of the lung, leading to the formation of new connective tissue, is called *chronic interstitial pneumonitis*, and is present in some degree in all cases of chronic pulmonary phthisis. The new tissue encroaches upon the pulmonary parenchyma, causing the obliteration of air-cells and shrinkage of the affected portion of the lung. New fibrous tissue may be formed directly from tubercle-nodules, from diffuse tubercle-tissue, or as a result of chronic interstitial inflammation. Fibroid phthisis, which is characterized by an excessive formation of fibrous tissue and retraction of the lung, presents sufficient clinical peculiarities to demand special consideration, which will be given in the next article.

Pulmonary phthisis is always accompanied by *pleuritis*, usually of the dry form. The pleuritis leads to the formation of vascular adhesions, which are, as a rule, most extensive and firmest at the apices of the lungs.

It is customary to find around tubercle-nodules and foci of cheesy pneumonia the air-cells filled with the ordinary products of inflammation. Patches of ordinary catarrhal or of fibrinous pneumonia may also occur in parts of the lung which do not contain tubercles or caseous masses. Pneumonia of this form does not undergo cheesy degeneration unless the affected part of the lung be invaded by tubercle bacilli.

Of *non-inflammatory changes* deserving mention, there are pigmentation with coal-particles and with melanin, particularly in newly-formed fibrous tissue; calcification of caseous material; atelectasis; compensatory emphysema; congestion and localized and general œdema.

After a variable period *cavities* are formed in the majority of cases of phthisis. These are of two kinds—those due to dilatation of bronchi, *bronchiectases*, and those resulting from the softening of cheesy material and its removal through the bronchi, *ulcerative cavities*. The bronchiectatic cavities are common, especially in connection with much increase of connective tissue. They do not attain a large size without leading to ulceration of the bronchial wall and the formation of excavations. The ulcerative cavities enlarge, partly by softening of caseous deposits in their walls and partly by suppuration of the walls. Cases differ much as regards the number and the size of the cavities. The greater part of a lobe, usually the upper lobe, may be converted into a single cavity. The cavities are generally irregular in form, with ridges of pulmonary tissue in their walls, and are often traversed by bands of fibrous tissue containing blood-vessels, the rupture of which may be the source of dangerous hemorrhage. In cases of fatal hæmoptysis the source of the hemorrhage is usually to be found in an aneurism of some artery in the walls of a cavity. These aneurisms are usually about the size of a pea and project from the side of the artery into the cavity. In recently-formed cavities the walls are often sloughy; in old cavities the walls are dense and firm, like the pyogenic membrane lining an old sinus. Cavities sometimes open into the pleural sac and give rise to pyo-pneumothorax. This accident is usually prevented by pleuritic thickening and the formation of adhesions between the pleural layers.

As the expectoration contains tubercle bacilli, it is common to find tuberculous lesions in the respiratory tract above the lungs, especially in the larger bronchi, the trachea, and the larynx. These lesions are usually in the form of tuberculous ulcers, and are in nearly all cases secondary to the pulmonary affection.

By swallowing sputum which contains the tubercle bacilli the tuberculous virus may gain access to the intestinal tract and cause tuberculous ulceration, which is found most frequently in the lower part of the ileum. From the intestine the bacilli may be readily conveyed to the peritoneum, the mesenteric glands, and the liver, which in many cases of phthisis are found to contain miliary tubercles.

The morphological and biological properties of the tubercle bacilli have been described in the former part of this work (p. 48). The number of bacilli present in different cases of phthisis is subject to considerable variation. In some cases they are abundant and occur in dense masses; and in other cases they are scanty and found only after long search. The bacilli are present in the bodies of cells, particularly of epithelioid and of giant-cells, and also free between the cells. They are always present in the beginning of tuberculous processes, and also when the tuberculous formation is in rapid progress. The bacilli are usually few or entirely absent in cheesy matter, but there are exceptions to this rule. When the cheesy substance is in communication with the atmosphere, as in the walls of phthisical cavities, the bacilli are generally abundant. Cheesy matter, even if bacilli are absent, usually contains the spores of the bacilli, as is evident from the possibility of producing artificial tuberculosis by the inoculation of cheesy substance which is free from bacilli. We are at present unable to demonstrate by coloring agents the free spores of tubercle bacilli.

The most important biological properties of the bacilli of tuberculosis are the development of spores, the slow growth of the bacilli, and the fact that the bacilli are incapable of development at a temperature below 28° C.

(82.4° F.) and above 42° C. (107.6° F.), the most favorable temperature for their growth being about that of the human body. In the ordinary conditions of nature, therefore, the bacilli do not develop outside of the bodies of human beings or of animals. The bacilli or their spores retain in the dry state their virulence, at least for many months, outside of the body, as has been proven by inoculation-experiments with dried phthisical sputum.

In most cases of pulmonary phthisis the primary infection takes place by inhalation of tubercle bacilli. These bacilli may lodge in the small bronchi or in the air-cells, or they may be carried into the substance of the lung by the lymph-current or by transportation in wandering cells. The first effect of the bacilli is to cause proliferation of the fixed cells by indirect division, as has been shown by the observations of Baumgarten. This cell-division results in an accumulation of epithelioid cells, which represent the beginning formation of tubercle. There is subsequently an emigration of white blood-corpuscles, which accounts for the appearance of lymphoid cells in the periphery, and later in the interior, of the tubercle-nodule. From this primary tubercular formation, which is most frequently in the apex of the lung, the bacilli make their way into the adjoining lung-substance through the lymph-spaces and the lymph-vessels and give rise to new tuberculous growths. From these primary tubercles, especially after they have undergone cheesy degeneration and have ruptured into the bronchi, the bacilli, often in large quantities, can enter the bronchial tubes, whence they are partly expectorated; but, unfortunately, they are often inspired into other bronchial tubes, possibly those of the opposite lung. This secondary invasion with bacilli from tuberculous foci already existing in the lung gives rise to new tuberculous growths, and especially to patches of cheesy pneumonia. Thus, partly by the peripheral growth of tuberculous nodules and infiltrations, partly by conveyance of the bacilli through the lymphatic vessels and spaces, and partly by the entrance of the bacilli into the bronchi, one area after another of the lung becomes the seat of tubercles or of caseous pneumonia. This extension of the tuberculous process is most frequently from the apex toward the base of the lung. The tubercle bacilli may also enter the blood-current, as has already been explained. In this way the tubercular virus may be carried to other organs of the body. It is readily understood that through the lymphatic current the bronchial glands often become tuberculous in cases of phthisis.

A simple and convenient division of phthisis into stages is based on the marked difference, as regards anatomical characters, before and after the formation of cavities. The first stage embraces the period during which the tuberculous products accumulate and soften, up to their evacuation in a liquefied state. The second stage extends from the time when cavities are formed to the termination of the affection. A stage of softening, distinct, on the one hand, from the period during which the caseous products are solid or semi-solid, and on the other hand from the cavernous stage, is superfluous, for the existence of such a stage cannot be predicated with any degree of certainty on either symptoms or signs. As regards the two stages—namely, before and after excavation—it is to be borne in mind that frequently, if not generally, both are represented at the same time in different portions of the lungs. After cavities have been formed in certain portions more recent tuberculous products in other portions are still either firm or are undergoing the process of softening.

In this division into two stages it is assumed that the affection goes on to the formation of cavities. This is the rule, yet exceptions to the rule are not very infrequent. If the tuberculous products be scanty and disposed in small disseminated nodules or tubercles, they may be absorbed. In some cases the animal constituents of the deposit may be infiltrated with lime salts and become hard, calcareous bodies, which may remain imbedded in the lung, or, ulcerating into the bronchial tubes, they are expectorated. In these modes a cure of the local affection takes place in a certain proportion of cases. But a cure may also take place after the affection has gone on to the second stage. It is well ascertained that cavities, even of considerable size, may gradually contract, and at length perfectly cicatrize. In some cases in which cicatrization does not take place, cavities become lined with a non-secreting membrane, and they may remain nearly innocuous for an indefinite period.

CLINICAL HISTORY.—In sketching the clinical history of this affection the symptoms attending its development and presented during the first stage will, in the first place, claim attention.

The affection is often remarkably insidious in its development. In a pretty large proportion of cases the pulmonary symptoms which may be considered as denoting the tuberculous affection are neither preceded nor accompanied by ailments of any description which lead the patient to suspect the existence of any disease. In the majority of cases, when the patient first comes under the observation of the physician the previous history renders it probable that the affection has existed for several weeks or perhaps months.

Of the pulmonary symptoms, the first which occurs is cough. Unless the affection be developed coincidently with an attack of bronchitis, which is not common, the cough is at first and for some time slight, dry, hacking, and perhaps it excites little or no attention. Occasionally, however, it is from the outset a prominent symptom. The expectoration, for some time wanting, is at first small, and consists of frothy, glairy mucus. It progressively increases, becoming gradually more abundant, and at length solid and opaque. These symptoms proceed from irritation and circumscribed bronchitis. Hæmoptysis frequently takes place shortly after the occurrence of cough and expectoration. In some cases it precedes these symptoms. It is often the event which first excites any apprehension of serious disease and leads the patient to seek medical advice. If cough and expectoration have existed for some time prior to this event, the patient had not heeded them, thinking they were due to a slight cold and that they would shortly disappear.

Pain in the chest does not belong to the disease *per se*. Pain, however, is liable to occur at times more or less frequently during the first stage of the disease, and is due to circumscribed pleuritis. The pain is sharp, lancinating, or stitch-like, referred to the summit of the chest, and frequently shooting beneath the scapula. It varies at different times and in different cases in severity, being rarely sufficient to keep the patient in bed, or even within-doors, and it may continue for one, two, or three days. In the progress of the disease the pain in these attacks is referred successively to both sides of the chest, and it may be more severe and frequent on the side in which the amount of disease is the smaller. The attacks are considered by patients to be rheumatic or neuralgic. They possess considerable diagnostic significance, but they are of very little importance as indicating the amount or the progress of the disease. Intercostal neuralgia occasionally occurs, and is to be recognized by the diagnostic points which have been presented in treating of that affection.[1] The respirations are usually increased in frequency, and the increase, other things being equal, is in proportion to the amount of the phthisical disease. This symptom may not attract the notice of the patient. It is apparent especially on exercise. Even when, owing to the amount of disease, lividity is marked, dyspnœa is not always complained of.

Among other than pulmonary symptoms the condition of the circulation, as represented by the pulse, holds an important place. During the first stage more or less acceleration of the pulse is the rule, and this acceleration, although not great, is an important diagnostic symptom. The acceleration differs much in different cases. In general, it is in proportion to the activity of the disease. If the pulse be frequent the disease is likely to be rapidly progressive.

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[1] Vide p. 183.

The temperature is more or less raised in proportion as the disease is progressive. The thermometer thus aids in the diagnosis of the disease, and if the diagnosis be not doubtful it affords evidence of the disease being progressive or otherwise. When it is a question as to the diagnosis, increase of temperature is evidence for, and absence of any increase is evidence against, the existence of the disease. In well-marked cases the increase is a measure of the activity of the disease, whereas a normal temperature denotes that the disease is non-progressive.

Daily exacerbations of fever may occur in the first stage. Toward evening the acceleration of the pulse is increased, with increased heat of skin, and sometimes a marked sensation of burning in the soles and palms; the cheeks may present a bright circumscribed flush, and after some hours the exacerbation goes off with perspiration. The exacerbation is sometimes preceded by a chill. Chills also occur without being followed by notable febrile movement. They may be pronounced and accompanied by rigor, or they are slight and transient, consisting in some cases of occasional shiverings or a sensation as if water were trickling down the back. Well-marked chills, followed by increased heat and perspiration, may occur with such regularity that the patient is supposed to have intermittent fever, and under these circumstances, if the pulmonary symptoms be slight, the affection of the lungs may be for some time overlooked. I suppose there are few physicians of much experience in situations where they are liable to meet with intermittent fever who cannot recall cases in which this error of diagnosis has been made. Fever of limited duration may be due to an intercurrent circumscribed pneumonitis.

As regards appetite and digestion, there is much diversity in different cases. In general, the desire for food and the ability to digest it are diminished. In some cases there is almost entire loss of appetite, and even a repugnance to food. This is an unfavorable feature of the disease. Disorder of digestion is sometimes so prominent as to appear to be the chief malady. The physician should be on his guard against considering the cough in such cases as merely dyspeptic or sympathetic. Antipathy to fat is by no means constant; some patients take fatty articles of food freely without repugnance and with relish; and on the other hand not a few patients who are not phthisical have an antipathy to fat.

Diarrhœa occurs in the first stage in a certain proportion of cases. It may exist with and without a tuberculous affection of the intestinal canal, usually seated in the small intestine. Persisting or frequently recurring diarrhœa, accompanied by cough, be the latter never so slight, should excite suspicion of pulmonary phthisis, and the existence of this affection being ascertained, a tuberculous affection of the intestinal canal as a secondary affection is probable. This affection results from swallowing the expectoration. Diarrhœa having this significance is an unfavorable symptom, diminishing the prospect of improvement. The seat of the affection is usually the Peyerian and solitary glands. It leads to ulcerations differing from those which are characteristic of typhoid fever in a tendency to extend in a transverse direction and to occupy only portions of the elliptical patches; also in having hardened and raised margins. Tuberculous ulcerations are sometimes found in the large as well as in the small intestine, and they may be confined to the former. The ulcerations may lead to circumscribed peritonitis, and the peritoneal inflammation sometimes becomes general. Perforation of the intestine occasionally takes place, giving rise to general peritonitis, provided the peritoneal surfaces over the ulcerated space within which the perforation occurs have not become adherent from previous circumscribed inflammation. Intestinal hemorrhage sometimes takes place, and I have known it to prove the immediate cause of death.

Impaired nutrition, denoted by diminished weight, is frequently one of the earliest symptoms. As a rule, reduction in weight continues so long as the disease is progressive; on the other hand, the disease may be considered as non-progressive if the patient be not losing weight, and still more if he be gaining in flesh. Defect in the chylopoietic processes is shown by anæmia, which in most cases is an early symptom and is frequently marked.

The muscular strength progressively diminishes if the disease be progressive, but here there is much variation in different cases. Patients are rarely compelled to keep the bed in the first stage, and they are generally able to be out of doors. Deficiency of breath in some cases precludes an amount of active exercise or labor for which muscular strength is sufficiently retained.

Suppression of the menses is a symptom significant of the progress of the disease; the menstrual discharge diminishes in quantity, and in the course of the disease ceases. Patients often attribute importance to the suppression as a cause of their other ailments. It is, however, an event incidental to the disease, and does not claim special treatment. Facts do not show that suppression of the menses from other causes exerts an influence in the development of phthisis.

This disease does not tend directly to impair the intellectual faculties. The intellect frequently remains bright and active notwithstanding the progress of the disease. The feelings are usually cheerful and buoyant. Patients are hopeful respecting their condition; they depreciate their symptoms and are reluctant to admit that they have a serious malady. This state of mind sometimes amounts to an infatuation which renders it difficult to obtain from the patient a fair account of the symptoms, and it is necessary for the physician to rely chiefly on the statements of others and his own observations. To such an extent is the absence of anxiety characteristic of the disease that an opposite frame of mind militates somewhat against the existence of phthisis. Patients are readily persuaded that they are improving, and hence they fall an easy prey to quacks. They sometimes dislike to be told the truth, and take offence at an intimation that they are consumptive.

Proceeding to notice the points in the clinical history which distinguish the second stage, it is to be borne in mind that it is not easy, nor is it practically important, to draw an exact line of demarcation between the two stages. While the local affection has advanced to the second stage in one part, usually near the apex, the different gradations of the first stage may be represented in other parts. The disease is to be considered as having advanced to the second stage when it has led to the formation of a cavity or of cavities of sufficient size to give rise to certain distinctive signs and symptoms. The symptoms of the first stage continue into the second, in most cases being increased and modified by the existence of cavities.

The cough in the second stage usually becomes more prominent. It may be less irritable, and more exclusively for expectoration. It is troublesome and difficult in proportion as the contents of the cavities are not easily evacuated, owing to their size, want of free communication with the bronchial tubes, etc. The expectoration consists of liquefied phthisical products, matter furnished by the lining membrane of the cavities, and secretion from the bronchial mucous membrane. The quantity of expectoration is very variable, amounting in some cases to only a few ounces, and in other cases to a pint or more daily.

The appearances of the expectoration vary. It is oftenest muco-purulent, with more or less predominance of the characters of pus. In proportion as it is purulent the sputa do not remain distinct, but run together to form a homogeneous mass. Sometimes a collection of the liquid expectorated presents a thick, opaque appearance, not unlike that of pea-soup or gruel. The sputa are sometimes solid, remaining distinct, and in falling upon the bottom of a vessel assume a flat, round appearance like that of a coin, and hence are called *nummular sputa.* The sputa occasionally present yellow *striæ,* which consist apparently of pus. The edges of the sputa are sometimes ragged; another appearance is that of small particles, resembling boiled rice, contained in the matter expectorated. It is very rare for a semi-softened, cheesy, morbid product to be expectorated; but in a case under my observation several masses of this description were raised, evidently due to an unusually large ulcerated orifice being established between a collection of tuberculous products and a bronchial tube of considerable size. In this case the signs of a cavity were developed directly after the expectoration of a large amount of matter which contained the partially softened masses. A sudden expectoration of matter in considerable quantity, presenting a purulent or pyoid appearance, denotes the opening by ulceration of a so-called tuberculous abscess. Not infrequently this occurs repeatedly during the progress of the disease, but in many cases the ulcerated opening is at first small, and the liquefied matter escapes gradually into the bronchial tubes. The constituents, as shown by the microscope, are pus-corpuscles, epithelium, fatty granules, blood-discs, the yellow elastic fibre, and tubercle bacilli.

The quantity of matter expectorated is, in general, in proportion to the number and size of the cavities. It is usually more abundant in the morning than at any other time, owing to its having accumulated during sleep. Toward the close of life the act of expectoration becomes difficult on account of muscular feebleness, and the consequent accumulation in the air-passages not infrequently hastens death. Occasionally the matter expectorated and the breath of the patient emit considerable fetor, due to putrefaction of the contents of cavities.

Hæmoptysis occurs less frequently in the second than in the first stage; and occurring in the second stage it may be due to the rupture of the bands of pulmonary tissue which so often traverse tuberculous cavities, these bands sometimes containing vessels of sufficient size to furnish an abundant hemorrhage, or more frequently to rupture of the minute aneurisms in the walls of the cavities, to which reference has been made in the account of the anatomical characters. Sometimes the loss of blood in this way is sufficient to destroy life, but such instances are rare. The hemorrhage, however, may contribute to a fatal result.

Hoarsenesss or huskiness, and sometimes extinction of the voice, denote laryngitis, which may be developed in the first as well as in the second stage. Its occurrence is presumptive proof of pulmonary phthisis, the latter, as a rule, existing prior to the laryngitis.

Lancinating pains incident to circumscribed pleuritis recur in the second as well as in the first stage from time to time. Occasionally, both in the first and second stage, the pleuritis becomes general and occasions more or less liquid effusion. So far as my observations go, they confirm the statement by Louis, that double pleuritis with effusion denotes the pre-existence of tubercle. Acute pain, suddenly developed, with embarrassed breathing, acceleration of pulse, and prostration, point to perforation of the lung, and the signs of pleurisy with pneumothorax are to be sought for.

le, is more accelerated in the second than in the first
erbations are oftener present and more marked, with
and palms. The night-perspirations are often copious,
great discomfort.

igestion are more or less impaired, cases differing greatly
ms connected with the ingestion of food. Diarrhœa is
ent, and is a more prominent symptom in the second
d often with intestinal ulcerations. If profuse and per-
istinguished as *colliquative diarrhœa*. Colliquative diar-
lvanced period of the disease, although tuberculosis of
ot exist. It is then due to defect of intestinal digestion
from impoverishment of the blood. With the progress
ody progressively diminishes in weight, and the patient
an extreme degree of emaciation. The strength varies
. The patient may be confined to the bed for a greater
death, but not infrequently strength is preserved suffi-
be up much of the time, and even to go out of doors,
or even a few hours of death.

Bulbous enlargement of the ends of the fingers, with incurvation of the nails, forming what are called *clubbed fingers*, is occasionally observed. This peculiar appearance is characteristic of phthisis, but it is not pathognomonic. It may be strikingly marked in connection with organic disease of the heart without phthisis. It occurs in cases of fibroid phthisis oftener than in the form of the disease under present consideration. The toes may present a similar appearance, but in a less marked degree. These appearances occur only in cases in which the disease has existed for a considerable period. Of 1776 cases analyzed by Pollock, the fingers were more or less clubbed in 654, being a fraction under 27 per cent.

The mental condition in the second stage is frequently not less characteristic than in the first. The expectation of improvement, prolonged life, or even recovery, amounts in some cases to an insane delusion. Patients are sometimes occupied in forming plans for the future when it is obvious to any observer that they are on the verge of the grave. They may insist to the last that there is no danger. Over and over again in the hospital wards I have been asked by patients reduced almost to skeletons, and too feeble to sit up for a moment, if there was any danger or if they would recover. But whenever patients affected with this disease are satisfied that there is little or no hope of recovery, they generally become quickly and completely resigned.

Œdema of the feet and ankles is a symptom of the second stage, denoting notable failure of the circulation. For a time this may occur only after sitting or standing, disappearing after the recumbent posture has been maintained for some hours; but at length it is permanent, and may increase and extend over the whole of the lower extremities, rendering them unwieldy. When the œdema becomes thus marked, extensive, and permanent, thrombosis of the iliac or femoral veins is to be suspected—an event which is liable to occur in this disease. Coagulation in the vein on one side will account for the œdema in some cases in which it is either limited to or is much greater in one of the lower limbs. If the œdema be diffused over the body, constituting anasarca, coexisting disease of the kidneys is to be inferred; but the latter is a rare complication of pulmonary phthisis.

The clinical history is affected in no small degree by complications and coexisting affections. The occurrence of tuberculous disease of the intestines, tuberculous laryngitis, pleuritis with pneumothorax, and disease of the kidneys has been already referred to. Chronic peritonitis is an occasional complication. The researches of Louis demonstrated that chronic peritonitis, not traumatic, is almost invariably dependent on tuberculosis in this situation, and hence, from the existence of the peritoneal affection, pulmonary phthisis may be inferred. Acute peritonitis from perforation is sometimes incident to the tuberculous affection within the intestinal canal. Meningitis, usually tuberculous, occurs as a rare concomitant in the adult, but is not infrequent in early life. This coexisting affection accounts for the development of delirium and coma in certain cases. Perineal abscess, leading to fistula, is another complication occurring in a certain proportion of cases, proceeding, as microscopical examinations have shown, from tubercle in this situation. Clinical observation appears to show that, as a rule, when this complication occurs the amount of tubercle in the lungs is small or moderate and the progress of the pulmonary affection is slow. An accumumulation of fat within the liver-cells is a morbid condition observed in a considerable proportion of cases, occurring oftener in women than in men. Authors of works on the diseases of the skin have noted the occasional occurrence of tinea versicolor in phthisical patients.

The duration of this disease is extremely variable. In the vast majority of cases it is essentially a chronic affection, but sometimes it runs a rapid course, passing through its changes and destroying life in a few weeks. The disease in such cases has been known as "galloping consumption," or phthisis florida. It is different from that known as *acute miliary tuberculosis*, which will be noticed under a distinct heading. Distributing fatal cases into groups according to the duration of the disease, exclusive of the rapid cases just referred to, one group will consist of cases in which the disease continues from three to six months; in another group death takes place between six months and a year; in a third group the career extends from one to two years; and in another group the fatal termination is held in abeyance for many years.

Limiting attention to the cases in which sooner or later the disease proves fatal, its march is extremely irregular. It is not uncommon for little or no progress to be apparent for several successive weeks, months, or even years, and then the disease to advance with more or less rapidity. Distinct developments occurring at successive periods are frequently points of departure for the onward march of the disease; but cases differ much as regards the local destruction and the rapidity with which softening, ulceration, and the formation of cavities take place. (Further remarks on the march of the disease will be offered under the head of Prognosis.)

PATHOLOGICAL CHARACTER.—The pathological character of tuberculosis has been considered in Part I. of this work (p. 48). In a practical point of view pulmonary phthisis involves the co-operation of two pathological factors. One of these is the bacillus tuberculosis. This is an essential factor. The colonization of this micro-organism within the lungs is the primary step in the production of pulmonary phthisis. Certain local conditions favorable for the multiplication of the parasite are not less essential than the presence of the latter. The nature of those local conditions which form the soil suited for the cultivation of the parasite is unknown. Certain conclusions deduced from clinical observations will be noted in connection with the causation of the disease. These unknown conditions constitute a predisposition to this disease. This term, "predisposition," in its application to pulmonary phthisis, designates what has hitherto been expressed by the terms tuberculous constitution or diathesis.

CAUSATION.—The causes of pulmonary phthisis relate, first, to the specific micro-organism, the bacillus tuberculosis; and second, to the conditions which constitute a tuberculous predisposition or diathesis. The disease is never produced without the parasite, but the efficiency of this agent depends on the predisposition or diathesis. The latter alone is incapable of producing the disease. If the introduction of the parasite into the air-passages could be prevented, the disease would not be produced, no matter how strong the predisposition; and, on the other hand, if the predisposition be wanting the disease will not be produced, no matter how great the exposure to the specific cause. Absolute prevention of exposure to the specific cause is only possible in situations in which tuberculous disease does not exist, either in man or lower animals. The exposure is greater, other things being equal, the greater the prevalence of the disease, and in proportion as circumstances favor proximity to patients affected with it. When it is considered that about one-seventh of all human beings die of phthisis, it is not difficult to account for the wide distribution of the tuberculous virus. The fact that protection against the introduction of the parasite is impossible in most places renders the causes of the tuberculous predisposition especially important.

The bacillus tuberculosis is not capable of multiplication outside of the animal body under the ordinary conditions of nature, and hence pulmonary phthisis is not only an infectious but a contagious disease. The parasite is the contagium. The contagiousness of this disease—which has long been a moot question—is not easily established by clinical evidence, the instances being so many in which circumstances favorable to communicability are present without its being communicated. Moreover, the doctrine of chances explains the occurrence of a disease so prevalent as this in a certain number of those who are brought more or less intimately into close proximity to phthisical patients. That the tubercle bacillus is the living contagium of tuberculosis the experiments of Koch and others have established demonstratively by the inoculation of certain of the lower animals with pure cultures of this micro-organism. The disease is never produced without the agency of this contagium; but many persons are exposed thereto with impunity, for the reason that in them the tuberculous predisposition does not exist. The apparent incongruity between the proof of contagiousness afforded by inoculation in certain of the lower animals, and the uncertain evidence obtained by clinical observation, is to be accounted for by the fact that for the contagion to be effective the predisposition is requisite; and this predisposition does not exist in all persons or at all times in the same person.

The most fruitful source of tuberculous infection is to be found in dried phthisical sputum. Experiments have shown that currents of air do not carry bacteria off from fluids or moist surfaces, but when the substances containing the bacilli are dry, these organisms are readily transported with particles of dust in the atmosphere. Tuberculous infection may come from the lower animals, but probably much less frequently than from the human body. That it may come from the milk of tuberculous cows is certain. In children especially it is liable to be derived from this source.

Facts show the tuberculous predisposition or diathesis in a certain proportion of cases to be congenital and inherited. Why a peculiarity of constitution rendering a person specially liable at a certain age to this disease should be transmitted from parent to child, cannot be explained, any more than the fact that peculiar traits of physiognomy or of mental character are inherited. The conclusion rests on observation. It is a matter of common remark that the offspring of consumptive parents often become tuberculous. But it is to be borne in mind that owing to the frequency of the disease a tolerably large proportion of phthisical patients must have a consumptive parentage from mere coincidence; and it may be true that, sufficient consideration not having been accorded to this fact, hereditary influence has been overrated. Facts show that the tuberculous predisposition is sometimes inherent in the constitution and inherited. To what extent a congenital influence is involved in cases in which circumstances do not render it evident must be left for conjecture. It is not irrational to suspect its existence in a large proportion of the cases in which proof is impossible.

The predisposition has relation to age. The disease is most liable to be developed between twenty and thirty. The other decennial periods of life are arranged in the following series, according to the rate of predisposition: thirty and forty, ten and twenty, forty and fifty, fifty and sixty, birth and ten, sixty and seventy, seventy and eighty, eighty and ninety, ninety and a hundred. No age is exempt from a liability to the disease, and it may affect the fœtus *in utero*. Tuberculous cavities are rare in infancy. Dr. Leuf of Brooklyn, N. Y., has communicated to me a case in which cavities existed in a child not quite six months old.

Climate appears to exert an influence either for or against the development of the disease. The prevalence of the disease is less in climates either uniformly warm and dry or uniformly cold and dry than in those which are moist and subject to frequent alternations of cold and warmth. A high altitude seems to afford protection against the disease. Jaccoud states that observations for fifteen consecutive years warrant him in asserting that in Alpine situations elevated 4000 feet tuberculosis is almost unknown; and especially is this true of villages at an elevation of 5500 feet, where, in the language of the country, there are "nine months of winter and three months of cold weather." According to Dr. Hjaltelin, who resides in Iceland, the inhabitants of that country enjoy exemption from phthisis. The disease prevails to a much greater extent in cities than in the country. The researches of Bowditch go to show that in the State of Massachusetts cases occur more frequently in places in which the atmosphere is rendered humid by streams or marshes. Dr. Buchanan's observations show that in England cases are comparatively rare in dry, elevated situations. There is no foundation for the opinion that malaria affords any protection against the disease. Newly-settled places are frequently exempt for some time from its prevalence to much extent; but the explanation of this fact is that the pioneers who compose the early population in these places are persons generally of sturdy, vigorous health, and the habits of life in such a population are protective against this disease. As regards season, the disease is oftener developed during the spring months and the hot months of summer than at other seasons of the year. The disease prevails more among those whose pursuits are sedentary than among those whose occupation involves outdoor life. Want of exercise, defective ventilation, deficiency of light, and the depressing emotions apparently contribute to the production of the tuberculous predisposition.

The infrequency with which the disease is found in the post-mortem examinations of those who have died from intemperance has led to the supposition that the use of alcohol antagonizes the tuberculous predisposition.[1] There is reason to believe that habits of diet unduly restricted as regards variety and quantity, either from choice or necessity, may contribute to the development of the disease.

Pregnancy has been supposed to antagonize the tuberculous predisposition, and marriage has sometimes been advised as a prophylactic and even curative measure. Facts, however, do not afford support to this supposition. In the collection of cases which I have analyzed, 87 were married and under forty years of age. Of these 87 cases, the disease was developed during pregnancy in 10, and in 12 within a few weeks or months after confinement.[1] The inference is, that pregnancy, either directly or indirectly, has a decided and considerable influence in the etiology of the disease. The researches of Grisolle and Dubreuil show that the development of the disease not infrequently takes place during pregnancy, and when tuberculous patients become pregnant the progress of the disease is hastened rather than retarded.[2] Clinical facts are opposed to the notion that the progress of the disease is retarded by lactation.

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[1] Hérard and Cornil, *op. cit.*

Certain diseases exert an influence on the one hand to promote, and on the other hand to prevent, the development of tuberculosis. It rarely occurs in persons affected with pulmonary emphysema. Persons affected with cardiac lesions which interfere with hæmatosis rarely become tuberculous. On the other hand, measles and typhoid fever leave the system in a condition favorable for the development of tuberculosis. Are they who suffer in early life from a scrofulous affection of the lymphatic glands of the neck especially prone to pulmonary tuberculosis in after-life? I have been led to think that this question may be answered in the negative. I have collected a number of cases in which young and middle-aged persons presenting the characteristic cicatrices on the neck were free from tuberculous disease of the lungs; and on the other hand it is rare to find these cicatrices in persons who are affected with pulmonary tuberculosis. Assuming tuberculosis and the scrofulous cachexia to be identical, as has been proved by the presence in both of the bacillus tuberculosis, it would seem that the predisposition is, as it were, exhausted by the affection in the glands of the neck, and that afterward it is not likely to recur. Contrary to what might be expected *a priori*, anæmic persons rarely become tuberculous. The association of anæmia with pulmonary tuberculosis, except as a complication or an effect, is rare. It is rare for phthisis to follow an attack of lobar pneumonitis or acute pleuritis. Statistics show that these affections have not a causative relation to pulmonary phthisis.

Bronchitis, acute or chronic, has no tendency to eventuate in phthisis. This assertion contravenes not only a prevailing popular notion, but a traditional opinion still held by not a few physicians. I make the assertion, however, with the utmost confidence in its correctness, inasmuch as it is based on the study of a large number of cases with reference to the earliest symptoms and signs. Moreover, it accords with the clinical researches of Louis, which, so far as they go, are not less valuable to-day than when they were made, more than half a century ago.

Bronchial hemorrhage (*bronchorrhagia*) was considered by older writers, and recently by Niemeyer, as causative of phthisis. This is an error. Bronchial hemorrhage may occur irrespective of any connection with tuberculous disease, but when a connection exists the latter precedes and is causative of the former.

To the conformation of the thorax was formerly attributed considerable causative agency. This doubtless arose in part from observing that phthisical patients often present contraction of the chest, occurring as an effect of the disease. Persons with great deformity of the chest are not specially liable to phthisis. So also the idea that the exercise of the voice may be a predisposing or exciting cause has become obsolete; and observation shows that if any influence be derived from this source it is conservative rather than causative.

The opinion appears to be entertained by some writers that chronic pharyngitis, which is an exceedingly common affection in this part of the world, has a tendency to eventuate in pulmonary phthisis. This opinion is erroneous. After a large opportunity of observing cases of chronic pharyngitis, I have come to the conclusion that it generally denotes a condition of the system unfavorable to phthisical disease. The sebum-like matter which is liable to accumulate in the glands of the tonsils, and to be ejected by coughing in the form of round hard pellets, is sometimes mistaken for tubercles. These may be distinguished from obsolete tubercles or pulmonary calculi by their being unctuous to the touch and emitting when touched a fetid odor.

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[1] *Phthisis, in a Series of Clinical Studies*, Philadelphia, 1875.

[2] *The Elements of Prognosis in Consumption*, by James Edward Pollock, M. D., London, 1865.

In conclusion, of the agencies concerned in the causation of the tuberculous predisposition, it is difficult to estimate their relative importance. In view of the necessary existence of this predisposition in the development of pulmonary phthisis, this is a diathetic disease; but it is impossible to determine whether the diathesis be always innate, or whether in a certain proportion of cases it is acquired. Do the causes which contribute to the development of the disease produce the diathesis, or do they act as auxiliary causes, the diathesis already existing? Our present knowledge does not enable us to answer this question. Yet the limited knowledge which we possess is of much value in its practical application to prophylaxis and treatment.

We cannot define anatomically the tuberculous predisposition; in other words, in the present state of our knowledge we are ignorant of the anatomical conditions on which this predisposition depends. We can think of it only as a certain quality of lung-tissue which makes the latter a favorable soil for the multiplication of the tuberculous bacilli. Using the language of metaphor, we may say that the cells are unable to conquer in a struggle for existence with the bacilli. It is probable that those who are naturally resistant to the action of the bacilli may become tuberculous when exposed to the virus in large quantity, as in the case of a wife or a nurse constantly in close proximity to a tuberculous patient.

CHAPTER VII.

PULMONARY PHTHISIS (CONTINUED).

Diagnosis; Prognosis; Treatment.

DIAGNOSIS.—The diagnosis of pulmonary phthisis rests mainly upon an examination of the sputum and upon physical signs. The discovery of tubercle bacilli in the sputum is almost certain evidence of the existence of pulmonary tuberculosis. The possibility of these bacilli being accidentally present in the sputum of persons unaffected by tuberculosis is so slight that it need hardly be considered, and certainly any possible error from this source will be eliminated by examinations of the sputum repeated from time to time. It very rarely happens that bacilli are absent for any considerable length of time from the sputum of phthisical patients. Failure to find the bacilli, however, is less satisfactory as an evidence of the absence of phthisis than is the recognition of the bacilli as an evidence of the existence of phthisis. Nevertheless, when the sputum is repeatedly examined for a long time without the discovery of bacilli, the existence of phthisis is improbable. Cases which are sometimes described as non-bacillary forms of phthisis are generally, in so far as there is no error of observation, cases of some non-tuberculous form of chronic pulmonary disease, particularly chronic bronchitis with bronchiectatic cavities. Bacilli are sometimes very abundant in phthisical sputum; at other times they are few and are discovered only after long search. No definite rules can be laid down as to the relation between the number of bacilli and the rapidity of progress and the extent of the tuberculous disease.

On account of the diagnostic importance of the subject, directions will here be given for staining the tubercle bacilli in the sputum, but it must be mentioned that considerable familiarity with microscopical technique and observation is required for the successful employment of the methods described.

The best method of staining the tubercle bacilli is that devised by Ehrlich. The staining fluid is prepared as follows: Shake thoroughly aniline oil and distilled water in the proportion of about 5 parts of the oil to 100 parts of water. After about ten minutes, filter the mixture through a filter moistened with distilled water. If drops of oil be still present in the filtrate, it is necessary to filter again. To the clear filtrate add enough of a saturated alcoholic solution of fuchsin or of gentian-violet to produce a dark color, with a slight opalescent scum on the surface. (The alcoholic solution of the staining dye is saturated when after standing a number of days some of the staining substance remains undissolved at the bottom of the bottle. This alcoholic solution can be kept permanently.) The filtered mixture of aniline oil and water may be prepared fresh every time it is used, or a staining fluid of the following composition may be preserved for about two weeks: 100 parts of filtered aniline oil and water, 11 parts of saturated alcoholic solution of fuchsin or of gentian-violet, and 10 parts of absolute alcohol.

In order to prepare the specimen, portions of sputum should not be taken at random, but, if they be present, little whitish or yellowish particles should be selected from the sputum and transferred to a cover-glass, where such a particle is to be spread over one surface of the glass in as thin and as uniform a layer as possible. This is accomplished either by means of a scalpel or needle or by pressing two cover-glasses together and then drawing them apart. The specimen should be allowed to dry thoroughly, and then the cover-glass, with the specimen uppermost, is passed with moderate rapidity three times through the flame of a Bunsen burner or of a spirit-lamp. In this process the specimen should not be burned.

The cover-glass, thus prepared, is now placed, specimen side downward, upon the surface of the staining fluid, where it remains for from twelve to twenty-four hours. The staining process may be hastened, although the results are somewhat less satisfactory, by heating the fluid to beginning ebullition. In this way a specimen may be stained in ten minutes.

The cover-glass, on which the specimen is now deeply stained, is transferred to a mixture of 1 part of pure nitric acid and 3 or 4 parts of water. Here the specimen remains a few seconds, at the most half a minute, after which most of the color is extracted.

From the acid the cover-glass is transferred to alcohol (60–70 per cent.), where most of the remaining color is removed. A few seconds to half a minute in the alcohol suffices. The cover-glass is now removed from the alcohol, and, if necessary, after a second washing in fresh alcohol it is allowed to dry. After it is dry the specimen may be mounted in Canada balsam or it may be examined in xylol, oil of cedar, or, without previous drying, in glycerin, or even in water. After removing the specimen from the alcohol a complementary stain, although it is not necessary, may be obtained by placing the cover-glass for a few seconds in a weak aqueous solution of Bismarck brown (if gentian-violet be employed) or of methylene-blue (if fuchsin be employed).

For recognizing the bacilli it is very desirable to make use of an oil-immersion objective and an Abbé's illuminating apparatus. The tubercle bacilli appear as slender rods about one quarter to one half the diameter of red blood-corpuscles, and frequently curved or slightly bent. They are stained either blue or red according to the staining dye used. The bacilli of leprosy stain by the same procedure as that adopted for tubercle bacilli, but an error from this cause is not likely to occur.

It is particularly important to examine the sputum for bacilli in the early stages of phthisis before the phthisical signs are distinctive. By the discovery of the bacilli in cases of early hæmoptysis great assistance has been rendered in diagnosis.

Elastic fibres are often present in phthisical sputum, even in an early stage of the disease. They afford evidence of destruction of lung-tissue. They may be readily discovered by boiling the sputum for a short time with a solution of caustic potash or of caustic soda (1 to 6), as recommended by Fenwick. The sediment may be collected in a conical glass, and after settling a portion of the sediment may be transferred to a slide by a pipette. The fibres may be recognized by their glistening appearance and curled shape. Sometimes they preserve their alveolar arrangement.

The physical conditions when the local affection is considerable or when the disease is advanced to the second stage are represented by well-marked physical signs. The diminished volume of lung at the apex causes a depression at the summit of the chest, and, owing to pleuritic adhesions, the expansion at the summit is less than on the opposite side. The scapula on the side most affected is restrained in its movement upward in inspiration, contrasting in this respect with the opposite scapula. These signs, obtained by inspection, are available in a certain proportion of cases. Percussion elicits relative dulness, and sometimes even flatness. Exceptionally, over the solidified portion of lung the resonance is tympanitic. A tympanitic resonance over a circumscribed space and the varieties of this resonance called cracked metal and amphoric denote cavities. The respiratory sound obtained by auscultation is either bronchial or broncho-vesicular or cavernous, and not infrequently these different signs are found in different situations in the same case. The first represents considerable or complete solidification, the second moderate or slight solidification, and the third a cavity or cavities. The cavernous respiration in some cases has an amphoric quality. The correlative vocal signs are usually present—namely, either bronchophony with the loud and whispered voice, or exaggerated vocal resonance, and in rare cases pectoriloquy. More or less of these auscultatory signs are available in the great majority of cases.

It is only in cases in which the local affection is moderate or small, the symptoms being at the same time less marked, that the diagnosis from physical signs is difficult; and the difficulty in such cases is rarely great, provided the physician have a good practical knowledge of physical exploration. Inspection may in these cases furnish the same signs, but less marked than in cases of a considerable local affection. As a rule, slight or moderate but distinct dulness on percussion may be ascertained. The exception to this rule is when more or less of the pulmonary lobules become emphysematous, and then the resonance is altered, although not diminished; it is vesiculo-tympanitic. A comparison of the respiratory murmur on the two sides at the summit will be likely to show abnormal changes—namely, the characters of the broncho-vesicular respiration. So also on a comparison as regards vocal signs, the vocal resonance and the bronchial whisper may be found to be increased. The same is true of vocal fremitus.

In addition to these signs there are several occasionally present which inferentially are evidence of phthisis. These may be distinguished from those already enumerated as the accessory signs of the disease. Fine bubbling or the subcrepitant râles heard within a circumscribed space at the summit of the chest on one side are highly significant of tubercle. They proceed from secondary circumscribed bronchitis. A crepitant râle, in like manner limited to a small space at the summit on one side, has the same significance, being due to secondary circumscribed pneumonitis. Crumpling and crackling sounds are significant, provided they be limited to the summit on one side. A pleural friction sound limited to the summit on one side denotes a secondary circumscribed pleuritis. Other accessory signs are an abnormal transmission of the heart-sounds and an interrupted or jerking respiration. The diagnostic significance of all these signs, it will be observed, depends on their situation at the summit of the chest on one side. Separately, they are not sufficient for the diagnosis, but coexisting with the direct signs—namely, dulness on percussion, broncho-vesicular respiration, exaggerated vocal resonance, and increased bronchial whisper—they serve to confirm these signs and render the diagnostic evidence positive.

The diagnostic points pertaining to symptoms to which attention is to be directed are as follows: (*a*) Cough and expectoration, not succeeding an attack of acute bronchitis and not connected with chronic pharyngitis, the cough at first dry, and afterward an expectoration at first small and transparent, and becoming gradually more abundant and opaque. (*b*) Stitch-pains at the summit, not connected with intercostal neuralgia. (*c*) Chills not referable to malaria. (*d*) Hæmoptysis: this is always a symptom of great significance, but the fact is to be borne in mind that it occurs in a certain proportion of cases without being associated with tubercle. (*e*) Accelerated breathing. (*f*) Loss of weight. (*g*) Pallor or anæmia not otherwise explicable. (*h*) Hoarseness or huskiness of voice proceeding from chronic laryngitis. (*i*) Chronic peritonitis not traumatic. (*j*) Suppression of the menses. (*k*) Buoyancy of mind instead of despondency.

In certain cases the solidification represented by physical signs at the time of an examination of the chest depends to a greater or less extent on an intercurrent, transient, circumscribed pneumonitis. This can only be ascertained by repeated examinations of the chest after certain intervals of time. The solidification, so far as due to superadded simple or ordinary pneumonic inflammation, will be found to disappear, leaving that which is caused exclusively by the phthisical affection. It is important to bear in mind the liability to a temporary increase of solidification of lung in cases of phthisis from an intercurrent pneumonitis which may not reveal itself by any distinctive symptoms.

PROGNOSIS.—The prognosis is extremely unfavorable, in view of the fact that in many if not most parts of the world the mortality from this disease exceeds that from any other, exclusive of fevers and affections which prevail epidemically or endemically. Yet the mortality from the disease has undoubtedly diminished within the past half century. This must be obvious to medical observers whose professional experience extends back for that period. The fact is also shown by the statistics published by the registrar-general of Great Britain and by the mortuary reports of the large cities in this country. The explanation of the fact is to be found in improved views as regards the management of the disease. The diminution which has already taken place in the death-rate from this disease affords ground for the hope that its formidable character may be still further mitigated.

The different modes in which the disease may pursue a favorable course have been already stated. The generations of bacilli may die out or be destroyed, and the tuberculous .products may either be absorbed or calcification may occur, forming the so-called obsolete tubercles or calculi; and these may remain quiescent or they may find their way into the bronchial tubes and be expectorated. Cavities may completely cicatrize. In these modes complete recovery may take place. This, of course, is the most satisfactory termination. Next to this is the persistence of cavities without any fresh products, the cavities giving but little inconvenience for an indefinite period, and even through a long life. According to these different modes in which the course of the disease is favorable, cases may be divided into those in which an arrest takes place without recovery and those in which the arrest is followed by recovery. Whether recovery follow or not depends on the extent of the local affection and other circumstances. Not infrequently the disease ceases to progress, with or without recovery, and after the lapse of months or years a recurrence occurs. The latter, then, is not properly a continuance of the disease, but a renewed invasion by bacilli.

The facts relating to the proportion of instances in which the disease ceased to progress, either with or without complete recovery, as developed by the analytical study of the collection of cases recorded by me during a period of thirty-four years, are as follows: Of 670 cases, the list embracing a few cases of acute tuberculosis and interstitial pneumonia, 44 ended in recovery. In my work on *Phthisis*, published in 1875, the details of the history of each of these cases are given sufficiently to render evident the correctness of the diagnosis and the fact of recovery. In 31 cases the disease was either arrested or ceased to progress, complete recovery from the lesions incident thereto not taking place. Thus, in 75 cases either recovery took place or the disease became non-progressive. The ratio of recoveries (1 to 15) is not fairly shown by these facts, inasmuch as the termination in a considerable number of cases was not ascertained. This statement of course applies equally to the ratio of cases in which the disease is either arrested or its progress ceases without recovery. A fair conclusion is that in not an extremely small proportion of cases complete recovery may be hoped for, and that this conclusion is equally true in respect to the disease becoming non-progressive without complete recovery. A recurrence of the disease after periods varying from one and a half to more than six years was noted in 6 cases. In 5 of these cases the recurrent disease terminated fatally. In the single case in which the recurrent disease did not prove fatal a second recurrence took place more than twenty years ago, the patient now living and being free from any pulmonary affection.

I may claim, in behalf of my clinical studies in relation to phthisis, the establishment of the fact that in a certain proportion of cases this disease is self-limited; in other words, it ends in recovery from an intrinsic tendency thereto. Of the 44 cases ending in recovery, in 23 there was no medicinal treatment to which an arrest of the disease could be attributed. In several there was no medicinal treatment whatever, and in the remainder the treatment consisted of simple tonics, cough palliatives, or remedies to meet other symptomatic indications. In none could the treatment be considered as curative. Of the 31 cases in which the disease became non-progressive without complete recovery, in 15 there was no medication by which, as might be supposed, the disease was controlled, and in several none whatever. In respect to hygienic treatment, in some cases of both groups there was no change whatever in the habits of life; in other cases there were changes involving more favorable circumstances pertaining to hygiene; but in a considerable number those changes were not of such a character that a potential influence could be attributed thereto. It is probably correct to say that the changes may have favored recovery or non-progression, but were inadequate to cause an arrest of the disease. The histories in these ten groups substantiate self-limitation in cases of phthisis. The disease may cease to progress and end in recovery because it is self-limited.

Assuming that phthisis in a certain proportion of cases is a self-limited disease, the practical bearing on the conclusions to be drawn from medicinal and hygienic treatment is important. If the disease may end in recovery exclusively from self-limitation, this must be more or less operative in the cases which recover under different measures of treatment. Self-limitation is a factor co-operative in certain cases with curative measures, and, it may perhaps be added, sometimes effective in spite of measures which tend to obstruct its operation. On the other hand, when this factor is feeble or wanting curative treatment is not likely to prove of much avail. Evidently, in drawing conclusions respecting the curative effect of remedies allowance is to be made for this factor. The extent of its co-operation doubtless differs much in different cases, in some being sufficient in itself, and in others either considerable, moderate, or slight. Considering that its agency has not been hitherto recognized, it is easy to explain the well-known fact that from time to time various methods of treating phthisis apparently successful in some striking instances have failed speedily to meet the expectations excited by their apparent success. Whatever was really due more or less to self-limitation was attributed solely to the methods of treatment, and when the former was feeble or wanting the latter gave little or no evidence of curative power. This is the rationale of the "*post hoc, propter hoc*" fallacy which has always had much to do with false deductions from therapeutical experience.[1]

The most rational explanation of the manner in which this disease ends by self-limitation is to suppose that the conditions which are essential for the multiplication of the bacilli, and which constitute the tuberculous predisposition or diathesis, after a time cease to exist. The vegetable parasite is destroyed because the soil becomes incapable of maintaining longer its existence. These conditions, it may be supposed, are greater and more enduring in some persons than in others, and the variation in this respect explains the differences in different cases of phthisis as regards the period in the progress of the disease when the ending by self-limitation takes place. In our present ignorance of the nature of the conditions on which depend the life of a parasite, our information in respect to the foregoing question must be derived from clinical observation.

The symptoms which warrant hope, and sometimes an expectation of a favorable course and termination, relate especially to the circulation, the body-heat, alimentation, and nutrition. Persistent frequency of the pulse, fever, anorexia, and progressive emaciation oppose reliance on self-limitation. *Per contra*, encouragement on this score is warrantable when the pulse is but little if at all more frequent than in health, the temperature of the body not much if any raised, the appetite in a great measure retained, and loss of weight inconsiderable. These and other symptomatic conditions which render it possible if not probable that the disease tends intrinsically to recovery may be summed up in one word—tolerance. In proportion as phthisis is well tolerated there is room for hoping that it will prove self-limited. If the tolerance be deficient, self-limitation is proportionably weak or wanting. Other things being equal, the chances of recovery by self-limitation are greater the smaller the amount of phthisical lesions and the space in which they are localized. I have been led to believe that phthisis not infrequently ends by self-limitation before it has made sufficient progress to develop well-marked physical signs. In other words, there are abortive cases of this as of other diseases. How often are the traces of an old, small phthisical affection found in bodies dead with various diseases! I have met with not a few instances of, as I supposed, abortive phthisis. These cases are likely to be misapprehended, the more because the general belief is that exceptions to the rule of progress are extremely rare.

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[1] Vide paper entitled "Self-limitation in Cases of Phthisis," by the author, in the *Archives of Medicine*, New York, June, 1879.

I have learned, however, from histories which I have recorded and reported, that self-limitation may be exemplified notwithstanding a large area of solidification followed by cavities of considerable size. The most reliable of the points on which a hopeful prognosis is to be predicated in these cases is the confinement of the affection within circumscribed limits; that is, absence of signs denoting progressive extension or general diffusion. The extent and degree of lesions, if circumscribed, do not prevent the disease from being self-limited, but they stand in the way of complete recovery. In most of the cases of non-progressive phthisis without recovery the lesions do not admit of restoration, although the phthisical disease may be said to have ceased; the lesions in these cases remain as sequels, like, for example, the intestinal ulcerations resulting in tropical climates from acute dysentery.

It is doubtful if any definite conclusion can be drawn from the number of bacilli in the sputa as regards the tendency to self-limitation. The complete disappearance of bacilli, however, in a series of specimens is proof that the tuberculous disease is arrested.

Aside from the symptoms and signs to be taken into account in forming a judgment respecting self-limitation, my clinical studies have shown that age and sex have no special significance. They show, what could not have been anticipated, that heredity is not incompatible with an intrinsic tendency to recovery. I will add that my histories afford evidence that profuse and repeatedly occurring hæmoptysis, chronic laryngitis, pleurisy with effusion, and perineal fistula are by no means in all cases unfavorable as regards prognosis based on self-limitation.

The disease proves fatal generally by asthenia. It is rare for the destruction of lung to be so great as to destroy life by apnœa. The patient is gradually worn out by the protracted disturbance occasioned by the disease, conjoined with progressive emaciation and exhaustion. The development of pneumo-hydrothorax from perforation may prove fatal in a short time, and then sometimes by apnœa. Acute peritonitis from perforation has been known to occur, and this affection is likely to prove rapidly fatal. Meningitis is another complication which hastens the fatal termination.

Hæmoptysis is very rarely an immediate cause of death. A few such instances, however, have fallen under my observation. A profuse and rapid flow of blood may occasion death by suffocation, the blood filling the bronchial tubes. A case of this kind has fallen under my observation. In two cases among those which I have analyzed death was attributable to profuse hemorrhage from a phthisical cavity. The analytical studies of Ware and Walshe proved that, as a rule, hæmoptysis is a favorable event as regards prognosis. My own clinical studies led to the conclusion "that cases in which hæmoptysis occurs show a larger number of recoveries, and a notably greater proportion of instances of arrest and of tolerance, than cases in which hæmoptysis does not occur."

Dunin has reported the sudden death of a patient affected with phthisis, in which air was found in the blood-vessels, the source of the air not being discoverable and no other cause of death being apparent.

Vogel has reported a case in which a cheesy affection of a bronchial gland led to a communication between a bronchial tube and the right subclavian vein, causing sudden death.

Duguet has reported a case in which a thrombus from the crural vein gave rise to embolism of the pulmonary artery and sudden death, the symptoms of phlegmasia dolens not having been present.[1]

TREATMENT.—Regarded from a rational standpoint in the light of the parasitic doctrine, the treatment of phthisis has reference to two objects. The first object is the destruction of the parasite by the direct agency of some substance which is either inhaled or introduced into the blood. Various agents destructive out of the body to bacteria have already been employed by inhalation, but thus far without encouraging results. The great obstacle in the way of thus directly destroying the parasite is the difficulty of the conveyance of the parasiticide in the form of either an injectable powder or in vapor to the colonies of the parasite, without the employment of this agent in a quantity sufficient to prove a source of danger. It remains for further experimental observations to furnish data for determining whether the progress of this disease can be arrested by disinfecting agents brought into contact with the parasite by means of the circulation. It is possible that some of the remedies which experience has shown to be useful may act as parasiticides. The second rational object of treatment is the removal of the tuberculous predisposition or diathesis. In the present state of our knowledge the prospect of success in arresting the disease or retarding its progress depends on the successful employment of measures for this object. In our ignorance of the nature of the conditions expressed by the terms tuberculous predisposition and diathesis our present knowledge of these measures must be derived wholly from clinical experience.

It is of course immensely desirable for the arrest to take place as quickly as possible while the local affection is limited and the pulmonary danger proportionably small; and with a view to a speedy arrest the importance of an early diagnosis can hardly be overrated.

Is the tuberculous predisposition or diathesis removable by any known special remedy or remedies? This question is to be answered in the negative. Various remedies have been supposed to exert a specific curative influence. Thus within the last half century wood-naphtha, cod-liver oil, phosphate of lime, the hypophosphites, the chlorate of potassa, and the benzoate of soda have successively been advocated as effecting a cure in cases of pulmonary tuberculosis. Few if any at the present moment consider these or any other remedies as specifically curative. This, however, is by no means saying that remedies are not useful in cases of pulmonary phthisis. Important indications are to be fulfilled by remedies, but in the present state of our knowledge more reliance is to be placed on hygienic than on medicinal measures of treatment.

Certain therapeutic measures heretofore employed, to a greater or less extent, under the guidance of false pathological views, do harm rather than good. In this category belong bloodletting, either general or local, blisters and other severe modes of counter-irritation, mercurialization, antimonial and other nauseating preparations, emetics, and cathartics. These are contraindicated. In general terms, everything is to be avoided which tends to impair the appetite, disorder digestion, and lower the vital powers.

The measures which clinical experience has shown to be useful, and sometimes effective, in either arresting or retarding the progress of the disease, are those which strengthen and invigorate. These terms, although lacking scientific precision, have a practical significance sufficiently intelligible. Directing attention to the measures which fall under the head of hygiene, these relate to diet, exercise in the open air, clothing, mental encouragement, and change of climate.

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[1] *Virchow u. Hirsch's Jahresbericht*, 1882.

The diet should be generous as regards quantity, quality, and variety. The articles should be highly nutritious and adapted to the digestive powers. All the varieties of wholesome food which the patient is able to take with relish should be allowed, and the appetite should be encouraged as much as possible. With respect to the kinds of food to be particularly recommended, there are no general rules applicable to all patients. Meats, milk, farinaceous articles, and the different vegetables are to be combined in relative proportions according to the choice, habits, and experience in individual cases. It is desirable that cream, butter, and other fatty articles should enter into the diet as largely as possible without inducing disgust and disorder of digestion. The same remark applies to sugar. The intervals between taking food should be such as will secure the largest amount of nutriment which can be taken and digested during the twenty-four hours. In short, it is far from desirable to arrange any fixed system embracing details of dietetics to which all patients are expected to conform. The object is to nourish the body to the fullest possible extent, and this requires in every case the co-operation of the physician and patient. Deficiency of appetite, especially for nutritious articles of diet, and weak or disordered digestion, are serious difficulties in the way of successful management. Much may be done by persevering efforts on the part of the patient, and he should therefore be made to understand the object which it is desired to attain.

A diet consisting largely, or even chiefly, of fresh milk has undoubtedly proved sometimes successful, and a trial of it may be advised when the appetite is deficient or when other articles of food occasion dyspeptic disorders. Goat's milk, mare's and ass's milk, are rarely used in this country. They have heretofore been, and are still to some extent, in European countries considered as preferable to the milk of the cow. In order to carry out effectually the "milk cure," as it is sometimes called, milk should not only constitute the chief article of diet, but it should be taken largely.

Exercise in the open air—or, as I should prefer to say, out-of-door life—is, of all measures, the most important. In 1863,[1] I analyzed the recorded histories of 62 cases in which an arrest took place, with a view to the points in the treatment which were common to all or a greater or less number. In 20 of these cases the arrest took place under hygienic measures without medicinal treatment. In these cases the most important point of agreement related to change of habits as regards exercise and out-of-door life. In respect to this point there was also an agreement in the cases in which medicinal measures were employed. The following extract from the paper contains a summary of the facts, with the general conclusions: "The most striking and valuable of the results of the analytical study of these cases is their almost uniform agreement as regards change of habits with respect to exercise and out-of-door life at the time of the arrest. Excluding 7 cases in which the arrest was attributable solely to self-limitation, and 2 cases in which the facts with respect to this point were not noted, of the remaining 53 cases, in all save 3 the histories show a greater or less change of habits to have been made; and in many cases the change consisted in relinquishing sedentary callings for other pursuits in order to carry out more effectually the desired reformation. There are grounds for believing that the advantage of a change of climate mainly consists in its being subsidiary to a change of habits as regards exercise and out-of-door life."

Phthisical patients should sleep in well-ventilated rooms. In conformity with popular notions they frequently fear the cool or cold air of night. A fire may be kept in sleeping-rooms in the winter season, but the windows should be kept open. Dr. James Blake of San Francisco, California, has reported several cases in which most favorable results followed living in the open air during the summer months, at an elevation of from 3000 to 5000 feet above the sea, in the Coast Range of the mountains of California, where the temperature is very equable and no rain falls for five or six months. The patients did not even sleep in tents, but were in the open air during night and day.

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[1] *Trans. N. Y. Acad. of Med.*, 1863.

Exercise in the open air should be accompanied by either mental recreation or occupations which interest the mind. It should, as far as possible, be incidental to pursuits which engage the attention. Adopted simply as a hygienic measure, it will rarely be persisted in. It is often essential, therefore, for patients to make a radical change in business, or, if they be able to devote their time to the restoration of health, hunting, sporting, travelling, etc. are to be resorted to as means of securing the union of out-of-door life with an agreeable exercise of the mental faculties. Over-exercise of the muscles is to be avoided. Patients sometimes, supposing that the benefit from muscular exercise is proportionate to its amount, carry it to the extent of producing a degree of fatigue and exhaustion which is injurious. It is not from the amount of physical exertion, but of out-of-door life with such exercise as is well borne, that benefit is to be expected.

The body should be protected against atmospherical changes, the temperature and functions of the skin being maintained by warm clothing. The garments worn next the surface should be made of a material which is a non-conductor of heat—namely, either wool or silk; but a superfluity of clothing is to be avoided. The object is to provide adequate protection without an undue accumulation of heat and perspiration. In a cold or temperate latitude during the winter season a waistcoat of buckskin or chamois leather, worn during the daytime over a light woollen or silk shirt, is to be recommended to patients of either sex as securing proper protection without the inconvenience of being bundled with an overplus of garments. The feet should be well guarded against cold and wet. Properly clothed, the patient, if he be strong enough to go out of doors, should rarely be kept within doors by the state of the weather, but should resolutely keep up habits of out-of-door life despite the ordinary changes of temperature and winds, remaining in the house only on inclement days.

I have frequently known the sponge-bath to be taken daily by tuberculous patients with apparent benefit. It may be safely tried and continued, provided it be followed by an agreeable glow. The temperature of the water used should be from 64° to 68° F. It is to be borne in mind, with reference to this measure as well as exercise in the open air, that the susceptibility to attacks of bronchitis is less the more the exposure, and also that an attack of bronchitis does not usually exert any unfavorable influence on the tuberculous disease. Many patients are deterred from going out of doors when the weather seems unfavorable, by needless apprehension of taking cold.

The proportion of cases in which an arrest of the disease is apparently either effected or promoted thereby is sufficient to authorize the physician to encourage patients strongly to employ the hygienic measures which have been briefly considered. The influence of the disease on the mind is to induce either an expectation of recovery without effort or resignation to death. It is important to make patients understand that very much depends on their own exertions. One of the difficulties in the way of successful management is a passive, patient, tranquil frame of mind which cannot be aroused to vigorous efforts. Persons endowed with resolution, energy, and perseverance, other things being equal, are more likely to struggle successfully with the disease than they who are deficient in these mental qualities. The measures which have been considered, if not successful in effecting an arrest of the disease, tend to retard its progress and render the condition of patients most comfortable while life lasts.

Change of climate, as a means of effecting a cure of pulmonary tuberculosis, has been a fruitful topic of discussion. It does not fall within the scope of this work to enter into a full discussion of this question. I shall present briefly certain considerations in connection with the question in individual cases, "Shall I try change of climate? and if so where shall I go?"

It may be assumed that a change of climate is frequently useful, and it appears sometimes to effect an arrest of the disease. The judgment of patients or their friends in the matter is not of much value, because it is usually based on the knowledge of a few cases, or perhaps of a single case, in which benefit may or may not have been apparently obtained.

It may be taken for granted that there is no specific influence in any climate. It would be therefore needless to inquire if there be one particular place on the globe to be preferred above all others. Observation shows that different climates are suited to different cases. In general, the advantages which belong to climate pertain to altitude, uniformity, and dryness. As regards the latter, while a dry climate is generally preferable, some patients appear to do best in situations in which the atmosphere is warm and humid. With respect to temperature, a uniform, cold climate is best for some cases, and a uniform, warm climate, for other cases. Of late years the custom has been gaining ground of sending patients to a cold latitude, and I have known of a number of instances in which the climates of Minnesota and Colorado have proved highly serviceable. Altitude is a marked element in these climates, as in St. Moritz and Davos in Switzerland. On the other hand, I have known of many instances in which a change to a warm climate has appeared to accomplish all that could be desired. What circumstances are to guide in deciding whether a cold or a warm climate will be likely to prove more advantageous? The feelings and choice of the patient are to have considerable weight. If when in health more vigor and enjoyment are experienced in summer than in winter, a warm climate will probably be better, and if the reverse be true a cold climate is to be preferred. The condition of the patient as regards feebleness is an essential point. If so feeble as not to be able to live out of doors in cold weather, or if the reaction from the impression of cold be slow and imperfect, a warm climate is more suitable. It is rarely proper to send patients to a cold climate if the disease be considerably advanced, and, as a rule, a cold climate is better suited to men than to women. These remarks with respect to temperature will necessarily apply to climates of which a high altitude is a prominent feature.

With reference to the particular place to be selected, it should contain resources for occupation and mental interest. There must be inducements for out-of-door life. I have known patients accustomed to active habits to suffer intolerably from ennui in going to places where the supposed excellence of climate was the only inducement. It is often better to move from one place to another than to remain stationary; as soon as a place becomes tiresome it is best to leave it. Travelling in foreign countries without any special regard for climate is often the best plan, the advantage consisting in the interest and inducements to out-of-door exercise derived from a succession of new scenes. A sea-voyage in warm latitudes is generally useful, and if patients be fond of the sea a long voyage, if practicable, may be advised. Many patients under my observation have resorted to sea-voyages; and I can affirm that while in not a few instances the benefit has been marked, I have never known any harm to ensue. I have known several instances in which patients in an advanced stage of the disease, the strength being much reduced, have taken long voyages with notable improvement. A change is often useful when there is no superiority on the score of climate, because it is in this way only, in certain cases, that relief from the cares and anxieties of business can be secured. As a rule, it is desirable to avoid places which are specially resorted to by phthisical patients, for the reason that seeing constantly examples of the different phases of the disease is likely to have an unfavorable moral effect. On this score sanitaria for phthisical patients are open to objection. Well-managed sanitaria, however, may have advantages relating to the systematic employment of regiminal measures, to diet, etc.

The habits and tastes of the patients are to be considered. Persons who are dependent on the associations and comforts of home and friends for their happiness will not be likely to be benefited by being sent away, especially if alone and among strangers. The stage of the disease and the rapidity of its progress are points of great importance. It is cruelty to send to a distance patients who are in a condition admitting of but little prospect of improvement, and who will probably not live to return. The symptoms and the amount of damage which the lungs have sustained, as determined by physical signs, are to be taken into account before deciding with regard to change of climate; and if a change be made and prove effectual, it becomes an important question whether, if practicable, the change shall not be permanent. The safest course, undoubtedly, is to take up a residence in a climate in which the disease is less liable to be reproduced than in the climate in which it has been already developed. Finally, in forming an opinion of the importance of change of climate it is to be considered that the progress of the disease will be unfavorable in a large proportion of cases, whatever may be the change or changes made. An arrest of the disease is not to be expected, save in a minority of cases. The fact that of the persons who make trial of change of climate the number who are not apparently benefited greatly exceeds the number who find this measure either successful or beneficial should not discourage resorting to the measure so long as in a small proportion of cases it proves effective. Another consideration is to be added—namely, a change of climate not infrequently appears to retard the progress of the disease, rendering the patient more comfortable and prolonging life, although an arrest of the disease be not effected.

In my series of clinical studies relating to phthisis[1] I endeavored to deduce some conclusions from the cases in which change of climate entered into the histories. In 74 cases this was an important, and in some instances the chief, measure of treatment. Of these 74 cases, in 9 the disease ended in recovery, and in 13 it became non-progressive. The proportion of 22 out of 74 cases is large enough to warrant the conclusion that a salutary influence was due to climate. Moreover, studying the histories closely, it appeared that in 34 additional cases there was evidence of improvement, leaving only 11 cases in which no benefit followed the change of climate. While self-limitation doubtless played an important part, it cannot account for these results to the exclusion of climatic influence.

The results relate to different climates collectively. A great number of places were resorted to by different patients—Minnesota, Colorado, California, New Mexico, the Southern States of the Union, Nice, Mentone, Egypt, South America, etc. My cases were insufficient to determine the comparative value of different climates. In undertaking to do this we have to deal with a complex problem. A fair comparison would require the histories of a considerable number of patients in each one of the various resorts embraced within the range of inquiry. There must at least be an approach to uniformity in all the cases as regards the stage of the disease, the extent of the local affection, and other circumstances. No single observer can accumulate data sufficient for such an undertaking. It would, however, be fair to take the results of all climates as a standard for reference, and bring into comparison the results of the analytical study of a considerable number of cases observed at any one or a greater or less number of the different places of resort. This might be done by observers residing in these different places. In this way very valuable data could be obtained for judging of the comparative advantages of different climates. The value of such data would be incomparably greater than any theoretical opinions in regard to altitude, dryness of the atmosphere, temperature, the presence of ozone, pine-woods' exhalations, etc. If, in addition to comparing results in particular places with those of different climates collectively, a comparison were to be made with the proportion of cases in which there is either recovery or cessation of progress without change of climate, it would be practicable to estimate, with some approximation to accuracy, climatic influences disconnected from the accessory circumstances, prominent among which is self-limitation. Single examples of recovery, however remarkable, are entitled to little weight as evidence of the curative effect either of climate or of any particular measure of treatment. In some of the most striking of the instances which have fallen under my observation the recovery took place without change of climate and without any important medication.

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[1] *Op. cit.*

In conclusion, while it must be a great relief to physicians to have formed convictions which prompt a ready and positive answer to the question so often asked, "What part of the world is best suited for phthisical cases?" it will be long before such convictions can have as a basis adequate numerical facts. Meanwhile, they whose minds are not committed to a belief in the superiority of a particular climate over all other climates must exercise judgment according to circumstances in individual cases as regards, first, the propriety of a change; second, the climate to be selected; and third, whether or not a change should be permanent.

As coming within the scope of hygienic management, marriage may be referred to. Physicians are sometimes consulted respecting the propriety of fulfilling a marriage engagement when one of the parties is affected with phthisis. My clinical studies (*op. cit.*) go to show that the effect of marriage on a phthisical patient, irrespective of pregnancy, is not unfavorable—a conclusion perhaps at variance with the opinion generally held. Regarded from a purely medical standpoint, the objections relate to the unfavorable influence of pregnancy if the wife be affected, the risk of phthisical offspring, and the possible communication of the disease from the husband to the wife and *vice versâ*. The physician should state these objections. Whether, regarded from other points of view, they should prevent marriage in particular cases is not a question for him to decide. My experience is that, whatever advice be given, the sentiments determine the action in these cases.

Passing to consider remedies which enter into the management, I place under this head alcohol, without discussing the question whether it be properly so regarded, or whether, as was claimed by Todd, it be alimentary rather than medicinal. Clinical experience shows that in a certain proportion of cases alcohol has a salutary effect. In some cases this effect is marked; in other cases alcoholics in any form are not well tolerated, and if their use be persisted in they may prove hurtful. If the immediate effect be that of a cordial—that is, if they produce a sense of comfort; if they be followed by a feeling of increased strength and a greater disposition to exercise; and if they do not excite the circulation or nervous system,—benefit may be expected from their use. *Per contra*, if their immediate effect be discomfort; if they be followed by a feeling of increased weakness and less disposition to exercise; and if they excite the circulation or nervous system,—they will not do good, and they may do harm.

As regards quantity, they may be given as freely as they can be taken without discomfort, without a sense of weakness or indisposition to exertion, and without excitation of the circulation or nervous system. The limitation in quantity is to be determined by the same circumstances which indicate their use, or, in other words, by their immediate effect. And, guided by these circumstances, it will be found that some patients will tolerate a large, some a moderate, and some only a small quantity. Phthisis is one of the diseases which in certain cases induce a remarkable tolerance of alcohol. Some patients who cannot take, without excitation, alcohol in any form in considerable doses, are able to take it without this effect, and with benefit, in a very small quantity repeated after short intervals.

The form to be used is another practical point of inquiry. Whatever be the form used, I believe the remedial principle to be alcohol. But no one form is equally applicable to all cases. Some patients are benefited especially by spirits, some by wine, and some by malt liquors. The selection of the form to be used in individual cases must be decided by the preference of the patient and experimental trials. As a rule, food should be taken with alcoholics.

The extracts of malt now in vogue cannot be regarded as substitutes for alcoholics, and it seems absurd to attribute to them anything beyond a certain amount of alimentary value.

With regard to cod-liver oil, without discussing the propriety of classing it among remedies, I proceed to state the practical points connected with its employment in cases of phthisis. All clinical observers unite in according to it more or less value. Its usefulness in a certain proportion of cases can hardly be doubted. Of the cases of arrested or non-progressive phthisis which I have analyzed, in several the treatment consisted exclusively in the use of cod-liver oil and hygienic measures. How much importance is to be attached to an intrinsic tendency to recovery and how much to hygiene in these cases cannot be determined, but an examination of the circumstances belonging to the individual histories goes to show that a certain amount of salutary influence belongs to the oil. Practically, the question of the use or non-use of the oil relates to individual cases. Clinical observation shows that it is not useful in all cases. I shall give, briefly, the rules which my own experience has led me to adopt respecting its use and non-use.

If it be taken without great repugnance, if it do not impair the appetite or digestion, or occasion derangement of the bowels, it may be expected to do good. If it produce any of the effects just named, its propriety is questionable. It is best to begin with a small quantity and increase to the maximum quantity, which is from half an ounce to an ounce three times daily, taken near the time of meals, either before or after, as choice or experience may dictate. It is generally best tolerated after meals. Of the different oils, some patients tolerate best the pale and others the brown variety. I have repeatedly known patients to take by preference the crude oil obtained at fish-markets. If the pure oil be not well tolerated, some one of the preparations containing lime or other ingredients to divest it of its repulsiveness may be substituted. Of course, in a given volume of the latter patients get a quantity of oil diminished by the bulk of the added material. Observations made by Dr. A. H. Smith and other members of a committee of the New York Therapeutical Society show that the addition of fifteen minims of ether to each half ounce of oil enables patients to take this remedy when, without the addition of the ether, it disagrees. The ether may sometimes be given with advantage half an hour after the oil has been taken.[1] Caoutchouc dissolved in the spirits of turpentine and prepared as a confection, has been largely used by Dr. T. R.

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[1] Vide *N. Y. Med. Journ.*, March, 1879.

Varick, instead of the cod-liver oil, with apparent benefit.[1] Glycerin has been thought to be useful as a carbonaceous medical food.

Arsenic is supposed by some to exert a certain influence on phthisis. The testimony based on clinical experience by different observers is discrepant. It is a fair conclusion from this fact that the remedy is sometimes useful and sometimes without any appreciable utility. My own experience leads to this conclusion. In a certain proportion of cases it appears to be useful. In order to secure benefit from it the remedy should be given in small doses—one or two minims of Fowler's solution—repeated three or four times daily. The doses should not be increased. The remedy in small doses may be continued for a long period, but if the dose be increased its toxical effects are liable to occur and its discontinuance becomes necessary. This is an essential point in the use of this remedy in cases of phthisis.

The hypophosphites have been much used in the treatment of phthisis within the last ten years, and as regards their utility there is the same discrepancy of testimony as with respect to arsenic. The same conclusion may be drawn from this fact as in the instance of arsenic. They are useful in a certain proportion of cases. Arsenic and the hypophosphites may be given in combination.

The bichloride of mercury is another remedy which, like the two foregoing remedies, is considered by some observers to have a curative influence. It is possible that whatever efficacy pertains to arsenic, phosphorus, and mercury is due to a direct effect upon the parasite; that is, they act as parasiticides. This remark will perhaps also apply to alcohol. It is, however, a more probable supposition that these remedies modify favorably, in some manner, the conditions on which depends the existence of the parasite. Arsenic is not destructive to the bacillus tuberculosis out of the body.[2]

All remedies which improve appetite, digestion, assimilation, and nutrition are in a greater or less degree useful. Hence quinia, salicin, strychnia or nux vomica, the bitter tinctures or infusions, and the mineral acids meet indications in cases of this disease. The chalybeate tonics are not without utility. That they favor the occurrence of hæmoptysis is an opinion not well grounded, and had they this effect it might be evidence for rather than against their utility.

Certain pulmonary symptoms claim attention. Expectorant remedies, as a rule, are not called for. They diminish appetite, occasion nausea, and disturb digestion. But remedies to allay superfluous cough—that is, cough not required for expectoration—are useful. For this end the succedanea of opium—namely, hyoscyamus, conium, belladonna, hashish, chlorodyne, and hydrocyanic acid—may be prescribed, but if these prove inefficient, opium in some form is required. The paregoric elixir will often suffice. Patients should be instructed not to yield to the disposition to cough, but to resist it whenever it is not effective. The habit of coughing unnecessarily is, in a great measure, under voluntary control. Soothing inhalations are sometimes useful in allaying the irritation which excites cough. The vapor of water or spray impregnated with conium or opium may be inhaled. I have known the vapor of chloric ether to be highly useful as a palliative. All irritating inhalations are hurtful. The oxalate of cerium, in doses of from 5 to 10 grs. three times daily, has been employed with much success as a cough-palliative. Its effect upon the stomach is that of a tonic.[3]

With reference to the injection of tuberculous cavities with a solution of the nitrate of silver or other medicated liquids through the larynx and trachea, it is sufficient to say that, aside from the difficulty of the operation and the impossibility of limiting the application of the injected liquid to cavities, a palliative effect is all that can be expected from this measure were it never so successfully executed. This statement applies to the injection of cavities after making an opening into them through the chest-wall. Prof. Pepper has reported 12 cases in which two hundred and ten injections into cavities were made, the objects being disinfection of the cavities and the promotion of cicatrization. In no instance was harm done, and the apparent results in several instances were beneficial.[1]

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[1] Vide *N. Y. Med. Record*, Nov. 1, 1873.

[2] Vide *Verhandlungen des Vereins für innere Medicin zu Berlin*, 1884.

[3] Vide article by Dr. Robert Cheesman, in the *N. Y. Med. Record*, June 12, 1880.

Certain complications furnish indications for treatment. Attacks of dry, circumscribed pleurisy call for mild revulsive and soothing applications, such as sinapisms, liniments, or the belladonna plaster. Local bleeding by leeches or cups is never required. The pain incidental to these attacks generally passes off in a few days at farthest. The treatment of coexisting laryngitis will be considered in Chapter XII. Diarrhœa dependent on tuberculous enteritis is to be kept in check by astringents and opiates, the latter being used with as much reserve as is consistent with the object to be attained. Chronic peritonitis claims only palliative measures.

In the progress of the disease symptoms other than those relating to the chest require palliative measures. The night-perspirations are frequently a source of great discomfort. The mineral acids, especially the dilute sulphuric acid and gallic or tannic acid, frequently afford relief. The external application of an astringent, such as alum dissolved in spirit, is sometimes effective. Belladonna or atropia is an effective remedy. Dyspnœa may be mitigated by ethereal preparations.

Paroxysms or exacerbations of fever are sometimes, in a measure, controlled by full doses of quinia. If the temperature be high this remedy is indicated as an antipyretic. If quinia be not well tolerated or if it prove inefficacious, antipyrine may be substituted. Sponging of the body may be resorted to in order to reduce a high temperature.

A comparison of the measures of management now generally pursued in this disease with the measures in vogue half a century ago shows a complete and radical change. Formerly, general and local bloodletting, cathartics, emetics, mercurials, counter-irritation, low diet, and confinement within doors were sanctioned by teachers and writers, and entered more or less into the treatment usually adopted. At the present time the measures which are most approved are, in all points, the reverse. The latter are based on more correct pathological views and the results of clinical observation. It is not unreasonable to hope that at some future time a remedy or remedies may be discovered capable of exerting a special influence over the disease, but in the mean time reliance is to be placed on an early diagnosis, the employment of remedies which, directly or indirectly, may exert, to a greater or less extent, a salutary influence, and, above all, on the hygienic measures which tend to invigorate and strengthen the system. The hygienic measures just referred to are to be enjoined with a view to the prevention of pulmonary phthisis. These measures are operative by affecting the conditions on which depends the tuberculous predisposition or diathesis. Prophylaxis is, of course, complete if the introduction of the infecting parasite can be prevented. As a measure for prevention a residence is desirable where there are few or no cases of phthisis. This effective precaution, however, is generally impracticable. Exposure to the infection should be restricted as far as practicable. Healthy persons should not occupy the same bed, and, except when required by the dictates of humanity, the same room at night, with tuberculous patients. Free ventilation of rooms occupied by patients should be enjoined.

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[1] *Trans. Amer. Med. Assoc.*, 1880, p. 239.

The sputa should be disinfected by carbolic acid or some other efficient agent. The flesh and milk of tuberculous cows have been shown to be capable of producing the disease; hence danger in this direction is to be avoided.

CHAPTER VIII.

FIBROID PHTHISIS.—ACUTE MILIARY TUBERCULOSIS.—GENERAL TUBERCULOSIS.

Fibroid Phthisis.

THERE is no sharp line of demarcation between fibroid phthisis and other forms of chronic pulmonary phthisis from a pathological point of view; but in its typical manifestations, regarded from a clinical standpoint, it presents peculiarities sufficient to warrant separate consideration. In such typical cases the lung is shrunken in size, with a corresponding retraction of the chest-wall and often a lateral deviation of the spine. The external appearances are like those which follow chronic pleurisy. The pleura is much thickened by the development of firm connective tissue, and the pleural surfaces are adherent. In the substance of the lung there are firm, dense bands and masses or nodules of fibroid tissue, frequently deeply pigmented. Cheesy and calcareous masses are often lodged in the black or grayish indurated tissue. There are, as a rule, abundant and extensive bronchiectases. Ulcerative cavities may also be present. One lung is usually in a more advanced stage of the disease than the other. Usually, tuberculous granula are evident to the naked eye. The microscope shows the presence of tubercles, often of the most exquisite giant-celled type, in the lung. These undergo the fibroid metamorphosis to which reference has already been made. They are frequently situated in the bands of fibrous tissue. Tubercles with cheesy centres are also usually present. Some of the larger fibroid masses may present the tubercular tissue in a diffused form. In addition to the tubercles there is chronic inflammation of the interstitial tissue of the lung. The alveolar septa, the interlobular, and the peribronchial connective tissue become increased in amount. The air-cells and bronchi are compressed, and many of them obliterated. The new connective tissue may be rich in lymphoid cells, or the cells may be few in number and spindle-shaped. According to the amount of caseous or tuberculous pneumonitis present there exist all stages of transition between typical fibroid phthisis and the ordinary form of pulmonary phthisis.

The names chronic interstitial pneumonitis and cirrhosis of the lung (Corrigan) have also been applied to this form of phthisis. The majority of the cases of so-called fibroid phthisis are tuberculous, as has been described; but at least some of the cases of chronic pulmonary inflammation resulting from the inhalation of fine foreign particles, such as bits of steel, of coal, etc. (knife-grinder's, coaler's phthisis, etc.), seem to be examples of pure chronic broncho-pneumonia and interstitial pneumonia.

This variety of phthisis is comparatively rare. In an exceedingly small proportion of cases it is preceded by acute pneumonitis; but in these cases the occurrence of the two diseases is probably due to coincidence. This may also be said of the cases in which it has been observed to follow bronchitis. In the majority of cases the age of patients is under forty years.

The local symptoms are cough with more or less expectoration, and want of breath on exercise. The sputum usually contains tubercle bacilli. The matter expectorated is often notably fetid, and sometimes suggestive of gangrene. Circumscribed gangrene occasionally occurs, but the fetor is generally due to the decomposition of muco-pus, from its detention in the cavities incident to bronchiectasis. Hæmoptysis, either slight or profuse, occurs in a considerable proportion of cases.

The disease may exist for an indefinite period without notable emaciation or debility; but sooner or later hectic paroxysms occur, diarrhœa is not uncommon, the appetite fails, and there is of course a corresponding defect of nutrition. The fingers often become clubbed. General dropsy occurs in a considerable proportion of cases. This is due to dilatation of the right side of the heart, an effect of the obstruction of the pulmonary circulation; that is, assuming the absence of valvular cardiac lesions.

As regards physical signs, aside from the contraction of the chest there is more or less dulness on percussion, rarely flatness, and the resonance elicited is often tympanitic. The tympanitic quality is marked in proportion as bronchiectasis is a concomitant lesion, and of course it is most marked when the dilated bronchial tubes are free from morbid products. Saccular dilatations may occasion cracked-metal and amphoric resonance. Either ordinary cavernous or amphoric respiration may be heard in certain situations. In other situations the respiration may be bronchial or broncho-vesicular. In some situations the characters of cavernous and bronchial respiration may be combined. On the other hand, there may be absence of respiratory sound over more or less of the affected portions of the chest. The vocal signs are those denoting pulmonary solidification and cavities—namely, bronchophony, increased vocal resonance, and sometimes pectoriloquy. Bubbling and gurgling râles are often present. The dilatation of the right side of the heart may give rise to a tricuspid regurgitant murmur and to a jugular pulse. The shrinkage of the lung may cause considerable displacement of the heart. If the left lung be the seat of the affection, the heart is raised and carried to the left; if the right lung be affected, the heart is sometimes removed to the right side of the sternum.

As regards diagnosis, the ordinary form of phthisis is excluded by the extent of the damage, taken in connection with the fact that the affection is especially marked on one side, by the absence of the emaciation which generally characterizes the former, and by the long duration, often with little or no progress. This differential diagnosis is less easy when the affection is bilateral than when it is unilateral. Affections with which it may be confounded are chronic pleurisy and carcinoma. Chronic pleurisy, after the removal of the effused liquid, does not furnish the signs of solidification, and perhaps of cavities, which belong to the clinical history of this disease. Carcinoma of the lung is generally secondary to carcinomatous disease in some other situation; the cancerous cachexia is often marked; there is progressive and perhaps rapid wasting of the body; and the progress of the disease is more rapid. The presence of the characteristic bacilli in the sputum is, of course, proof of the existence of tuberculous disease. In this variety of phthisis they are less abundant than in the ordinary form of the disease, and repeated examinations may be required for their discovery.

Fibroid phthisis involves lesions which do not admit of recovery. Sooner or later the disease ends fatally. But in many instances life is destroyed by some complication or an intercurrent affection. An acute pneumonitis affecting a lobe of the sound lung, or an attack of acute bronchitis, may prove fatal.

The ability to resist any severe disease is, of course, impaired. Alone, the disease ends fatally by slowly-progressive exhaustion; but, existing alone, it may be tolerated for a great number of years.

The TREATMENT has for its most important object maintenance of nutrition and strength. The measures for this object are remedies to promote appetite and digestion, a nutritious and abundant alimenation, together with climatic and hygienic influences which tend to invigoration. Palliative measures, having reference to cough and expectoration, are indicated. Expectorants are useful by preventing the detention of muco-pus in the bronchial tubes, the decomposition of which increases the bronchial inflammation. The inhalation of the vapor of turpentine is useful. Antiseptic inhalations are indicated if the expectorated matter or the breath be fetid.

Acute Miliary Tuberculosis—General Tuberculosis.

Acute miliary tuberculosis is characterized by the presence of miliary tubercles in most of the organs of the body. It is caused by the entrance of tubercle bacilli into the blood-current and their transportation to most parts of the body.

The organs almost constantly affected are the lungs, the liver, the spleen, and the medulla of the bones, and with nearly equal frequency the kidneys, the thyroid gland, the heart, the choroid coat of the eye, the lymph-glands, and the serous membranes. The affected organs are usually studded with small, grayish, firm nodules, which present the tuberculous structure already described in Part First (p. 48) and in the article on Pulmonary Phthisis. Some of the tubercles, particularly those in the liver, are so small that they can be seen only with the microscope. The longer the duration of the case the larger are the tubercles and the more extensive the cheesy degeneration of their centres. The lungs are hyperæmic, and the air-cells contain a small, rarely a large, amount of desquamated epithelium and of inflammatory products, particularly in the neighborhood of the tubercles. Acute inflammation of the bronchial mucous membrane coexists. More or less enlargement of the spleen is usually present. Certain peculiarities in the structure and the distribution of the tubercles in particular organs will be subsequently mentioned in connection with tuberculous disease of these organs. All tubercles, at least at the beginning of their formation, contain bacilli. The bacilli may be very few and difficult to find.

In most but not in all cases it is possible to demonstrate at the autopsy or by subsequent microscopical examination the mode in which the tubercle bacilli gain access to the blood-current. The mode of infection varies in different cases. It is usually to be found in some old tuberculous focus which may or may not have given rise to symptoms during life. In many cases a tuberculous nodule or mass has ruptured into one of the veins, particularly into one of the pulmonary veins. A frequent instance of this is the bursting of a caseous bronchial gland into a pulmonary vein. In some cases the general infection has occurred from tuberculosis of the thoracic duct or from rupture of tuberculous material into the duct. The seat of entrance of the bacilli into the blood may be demonstrable only by microscopical examination;[1] but then the chances of discovering it are much less than in the instances which have been mentioned. It is hardly necessary to say that the idea once entertained that any indifferent cheesy mass in the body might be the source of general tuberculous infection is not true. The primary cheesy mass must be of tuberculous origin in order to cause general tuberculosis.

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[1] As in a case reported by Bergkammer (*Virchow's Archiv*, Bd. 102, p. 397), in which the invasion of blood-vessels in the bronchial lymphatic glands by enormous numbers of bacilli could be demonstrated.

As has already been mentioned, the focus of local tuberculosis—which in most if not in all cases antedates the general tuberculosis—may or may not have given rise to characteristic symptoms. All varieties of local tuberculosis, such as pulmonary tuberculosis, tuberculous disease of the bones and of the joints, genito-urinary tuberculosis, and tuberculosis of the lymphatic glands, may be followed by acute miliary tuberculosis.

In some cases it is impossible to find any tuberculous products which seem to antedate the general tuberculous infection. Whether in these cases the primary tuberculosis is overlooked, or whether in rare instances in some unexplained way tubercle bacilli may directly invade the blood in large numbers from without, is uncertain. Acute miliary tuberculosis frequently develops in persons apparently in perfect health. It does not often develop in the course of pulmonary phthisis.

In cases of acute miliary tuberculosis tubercle bacilli have been found in the blood both during life and at the autopsy. They have been found in blood withdrawn during life from the spleen by a hypodermic syringe.

With reference to the symptomatology and diagnosis, it is to be noted that the name acute miliary tuberculosis is limited to cases in which tubercles involve, not a single organ, such as the lungs, the pia mater, or the genito-urinary tract, but a large number of organs. The lungs may be predominantly affected, but tubercles exist also in other parts.

Cases of this disease are rare; they occur mostly after puberty and before the middle period of life, but no age is exempt from a liability to the disease.

The general symptoms denote a febrile disease. The temperature of the body is more or less raised, but there is much variation in this regard in different cases. There is sometimes hyperpyrexia. Variations in the pyrexia at different periods in the same case are sometimes marked. The pulse is usually rapid, ranging from 120 to 140 per minute. There is complete anorexia and the prostration is great.

The respirations are usually notably frequent, from 30 to 60 per minute. Cough is more or less prominent. The expectoration varies, and is sometimes small or even wanting. Tubercle bacilli have been found in the sputum, but this is exceptional. The expectoration proceeds from secondary bronchitis. It may contain blood in streaks. Hæmoptysis is an occasional event. Among the cases given in my work on *Phthisis* is one in which the disease began with a profuse hæmoptysis. Another instance has more recently come under my observation. In some instances there is much suffering from dyspnœa, but patients do not always complain of a sense of the want of breath even when the respirations are notably hurried. Cyanosis is often a prominent symptom. Double pleurisy with effusion occurs in some instances.

The shortest duration in the cases which I have observed was eleven days. It rarely exceeds two months. Although there is great interference with the respiratory function, asthenia predominates in the mode of dying.

The disease may have no distinctive physical signs. If tubercles be disseminated about equally in both lungs, they do not occasion an appreciable dulness on percussion or the auscultatory signs of solidification. Some dry and moist bronchial râles, due to bronchitis, may be heard at different points, but these are not constant. The fact that physical exploration gives a negative result is to be taken into account in the diagnosis. This result excludes other pulmonary diseases which might give rise to the pulmonary symptoms—namely, lobar pneumonia, the common form of pulmonary phthisis, and capillary bronchitis. The basis of the diagnosis is the exclusion of these affections and the fact that the symptoms point to some grave intrathoracic and general disease.

The difficulty in the diagnosis relates chiefly to the differentiation from typhoid or typho-malarial fever. With these diseases acute miliary tuberculosis is apt to be confounded. This error is likely to be made if, as sometimes happens, cough with expectoration, rapid breathing, dyspnœa, and cyanosis are not as prominent as they are in the majority of cases. Moreover, in some cases of acute miliary tuberculosis fever heat ceases in the course of the disease. Typhoid and typho-malarial fever are to be excluded by the absence of the diagnostic characters of these affections. Positive proof of the existence of tuberculosis is obtained, of course, if examinations of the sputum show the presence of the bacillus tuberculosis. This proof, however, may not be obtained in cases of miliary tuberculosis. There are not as yet sufficient observations to determine how frequently tubercle bacilli may be found in the blood during life in case of acute miliary tuberculosis. The recognition by the ophthalmoscope of tubercles in the choroid coat of the eye may render great assistance in diagnosis.

The TREATMENT of acute miliary tuberculosis may be dismissed with a very few words. The condition of the patient is hopeless, and all that the physician can do is to palliate symptoms and sustain the powers of life. This is one of the diseases in relation to which recovery is proof of an error in diagnosis.

CHAPTER IX.

BRONCHITIS.

Acute Bronchitis affecting the Large Bronchial Tubes: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Subacute Bronchitis.—Acute Bronchitis in Young Children.—Bronchitis affecting the Small Bronchial Tubes.—Epidemic Bronchitis.—Influenza.—Bronchitis with Fibrinous Exudation.—Circumscribed Bronchitis.—Chronic Bronchitis.

THE preceding chapters have embraced inflammatory diseases affecting the pleura and the pulmonary parenchyma. It remains to consider inflammation seated in the lining membranes of the bronchial tubes. Inflammation in this situation constitutes the disease called *bronchitis*. This, in its ordinary form, is the most frequent of the pulmonary inflammations, and is a very common affection in all parts of the globe. A highly important variety of the disease is based upon the size of the bronchial tubes affected. Generally the inflammation is limited to the large bronchial tubes. In an infrequent and much graver form the inflammation affects the small tubes. The latter form is usually, but, as will be seen, incorrectly, called *capillary bronchitis*. Inflammation of the bronchial mucous membrane may be acute, subacute, or chronic, and varieties of the disease are based on these differences as regards the degree and duration of the inflammation. Bronchitis may be a complication of certain diseases, such as pneumonitis and phthisis, or it may not be preceded by any disease of the lungs. As a complication of other pulmonary affections it is more limited than when it is the primary affection, and may be distinguished as *circumscribed bronchitis*. A form of the disease is characterized by the exudation of fibrin, and may be denominated *fibrinous bronchitis*. Finally, bronchitis characterizes an epidemic disease commonly known as *influenza*. These several varieties of the disease will claim separate consideration after having considered the ordinary form—namely, bronchitis affecting the large bronchial tubes. The term *catarrh* has been used to denote inflammation of a mucous structure, giving rise to a mucous or a muco-purulent product. As the term bronchial catarrh expresses neither more nor less than bronchitis, it is superfluous and may be dispensed with. Proceeding to treat first of ordinary acute bronchitis, it will be considered as occurring after infantile life, and afterward certain points relating to the disease as it occurs in young children will be briefly noticed.

Acute Bronchitis affecting the Large Bronchial Tubes.

ANATOMICAL CHARACTERS.—Acute simple bronchitis begins with hyperæmia of the mucous membrane, often accompanied, in severe cases, with little ecchymoses. The mucous membrane becomes swollen and soft. The congestive redness and the swelling apparent after death in the affected tubes may be uniform or more marked in patches or zones. The appearance with respect to redness after death here, as in other situations, is not to be considered as evidence of the degree of hyperæmia present during life, for in parts open to observation—for example, the conjunctiva, mouth, and throat—redness marked during life may diminish or even disappear after death. The inflammatory products consist of mucus, pus-corpuscles, and serum mixed with desquamated epithelial cells. Sometimes, especially in the early stage of the disease, the product is chiefly mucus, when it appears translucent and viscid; at other times it is yellowish, when it contains abundant pus-cells. A coating of muco-pus is found upon the inflamed mucous membrane after death.

In ordinary bronchitis the inflammation, as already stated, is limited to the large bronchial tubes. . The tubes on both sides are equally affected, provided the affection be not incident to an antecedent pulmonary disease. With this exception, bronchitis exemplifies the law of parallelism; it is a bilateral or symmetrical disease. In this respect it differs from pleuritis and lobar pneumonitis.

CLINICAL HISTORY.—Acute bronchitis is generally preceded by inflammation of the mucous membrane of the nasal passages, or coryza. The inflammation begins in the nostrils and travels downward, either affecting or passing by the pharynx and larynx in its passage to the bronchial tubes. The period occupied in the passage varies from a few hours to one, two, or three days. In a certain proportion of cases the bronchial tubes are attacked at once without any affection of the air-passages above.

The symptoms offer marked points of contrast with acute pleuritis and pneumonitis. Pain is not a prominent symptom, but the patient has a sense of constriction and of soreness or rawness. These painful sensations especially accompany acts of coughing. The pain is of an obtuse or contusive character, and is situated beneath the sternum.

The attack is rarely accompanied by a distinct chill, but chilly sensations, followed by flashes of heat, are frequently repeated during the career of the disease. The appetite may be more or less impaired, but is not usually lost. Lassitude is complained of, with a general feeling of malaise.

The pyrexia is moderate. The pulse has not the frequency and strength which it has in pleuritis and pneumonitis. The heat of the surface is not notably raised.

The cough is at first painful, but not sufficiently so to be suppressed. It is at first dry, the secretion of mucus being for a time scanty. Deep inspirations, breathing cold air, and the exercise of the voice excite acts of coughing, which occur in paroxysms and consist of a deep inspiration followed by a series of expiratory efforts. If the paroxysms of coughing be frequent and severe, the traction of the diaphragm occasions pain and soreness, referred to the false ribs and the ensiform cartilage. The patient feels as if a more abundant expectoration would give relief, and desires to have the cough loosened. The expectoration is at first small, glairy, frothy, and viscid, and occasionally streaked with blood. In the progress of the disease, after two, three, or four days the expectoration becomes more abundant, and consists of thick, yellowish, or greenish sputa. The cough is then said to be loose, the acts of expectoration being easier, unattended by pain or soreness, and followed by a sense of comfort. The mucous secretion is rarely sufficient to accumulate in the tubes and occasion embarrassment of respiration. This may happen, however, in young children, in the aged, in feeble persons, and in patients with paralysis affecting either the diaphragm or the costal muscles. The presence of a collection of mucus in the large bronchi or trachea is felt, and the patient is led to make voluntary efforts of coughing for its expulsion. The increased amount of the inflammatory products and the change in character denote diminution or resolution of the inflammation.

Respiration is not sensibly affected.. No portion of the lung is withdrawn from the exercise of its function, as in pleurisy and pneumonitis. Dyspnœa is not produced, save in the exceptional cases in which, from feebleness or the want of voluntary efforts of coughing, the mucous secretions accumulate sufficiently to obstruct the bronchial tubes, provided the bronchitis be not superadded to emphysema or some other pulmonary affection and the muscles of respiration be not paralyzed.

The affection is not accompanied by much debility. Patients are generally not confined to the bed, and they may not confine themselves to the house.

The average duration of acute bronchitis is ten or twelve days. In severe cases after five or six days patients are usually able to be out of doors.

The disease may be divided into two stages. The first stage embraces the period during which the expectoration is scanty, transparent, and viscid. The second stage extends from the time when the expectoration becomes abundant, opaque, and thick, to convalescence.

Pathological Character.—Acute simple bronchitis is an inflammation affecting a mucous membrane, leading to a secretion of mucus and the production of muco-pus in greater or less abundance. Resolution takes place in the bronchial membrane without the occurrence of ulcerations.

Causation.—Primary or idiopathic bronchitis is attributed to the agency of cold. This is implied in the designation by which the affection is popularly known—namely, "a cold." Exposure to cold is supposed to produce this disease by interrupting the eliminative functions of the skin, whereby an increased duty is thrown upon the pulmonary mucous membrane, and by inducing internal congestion. This view of the causation is inconsistent with the fact that great exposure, in a large proportion of cases, is not followed by bronchitis, and also with the fact that in many cases bronchitis is not traceable to any unusual exposure. It is reasonable to conclude that the disease is due to a morbific agent in the atmosphere. This is of course to be inferred when the disease prevails as an epidemic; but it is a matter of common observation that a greater or less number of persons are likely to be simultaneously affected when the number is not enough to constitute an epidemic. That the special morbific agent is a micro-organism—in other words, that the disease is parasitic—is rendered probable by the absence of any appreciable causation in a large proportion of cases, and by the fact that the bronchial inflammation is preceded in most cases by coryza, frequently by laryngitis, and sometimes by pharyngitis, these affections successively occurring after intervals of several hours or days. It seems rational to attribute these successive affections to the migration of the parasites downward, and the formation of colonies in the parts successively affected.[1] The disease is more frequent in cold and temperate latitudes than in the tropics; and in the former it prevails especially in the spring and autumnal months. It presents a contrast to acute lobar pneumonitis, which occurs oftener in warm than in cold climates.

Bronchitis occurs secondarily to, and forms an element of, other diseases. It belongs to the clinical history of rubeola, or measles. In connection with this disease it is frequently acute. It occurs in typhoid fever, but is rarely acute in connection with that disease. Developed in connection with diseases situated elsewhere than in the pulmonary organs, it is symmetrical, as when it occurs primarily; but, developed as a complication of pneumonitis and phthisis, it may be limited to one side—that is, unilateral—and is confined within a circumscribed area.

It may be produced traumatically by irritating gases received with the inspired breath. Inhalation of chlorine gas will produce it. I have known a severe attack produced by inhaling the fumes of sulphuric acid. In some persons, owing to an idiosyncrasy, the bronchial membrane becomes inflamed by constituents of inspired air which are innocuous when inhaled by most persons. The powder of ipecacuanha, emanations from newly-mown hay and from feathers, the pollen of certain plants in the atmosphere during the summer and a portion of the autumnal season, and even fragrant odors, will produce bronchitis in those having a peculiar susceptibility to these causes. Frequently in these cases to the bronchitis is added asthma; and this idiosyncrasy will be noticed in connection with the latter affection. (Vide Chapter X.)

As a rule, the liability to bronchitis is less in proportion as persons are habituated to free exposure in the open air. It is much more liable to attack persons who spend most of their time in warm apartments than those whose occupations keep them out of doors. Soldiers, surveyors, explorers, and hunters, who are accustomed to sleep at night in tents or in the open air, rarely "take cold," but it is a matter of frequent observation that after having been for some time accustomed to camping out of doors bronchitis occurs on resuming in-door quarters. If the parasitic doctrine be admitted, these facts point to the importance of peculiar local conditions favorable to the multiplication of the parasite.

DIAGNOSIS.—Acute bronchitis is to be discriminated from pleuritis and acute lobar pneumonitis, more especially the latter The points relating to symptoms which are involved in this differential diagnosis are as follows: the absence of lancinating pain, the pain which is felt being substernal; the presence of more or less expectoration which is not rusty, but either devoid of blood or containing it in the form of streaks; absence of accelerated breathing and dyspnœa; absence of the circumscribed flush of the cheeks; and the pre-existence of coryza, with, perhaps, soreness of the throat, and subacute laryngitis. These points are chiefly negative, and they are not sufficient for a positive diagnosis in all cases, because the symptoms belonging to pleuritis or pneumonitis, which are wanting in bronchitis, are not uniformly present in cases of the two former diseases. The clinical history, however, and the results of physical exploration render the diagnosis easy and positive.

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[1] Vide "Suggestions as to the Etiology, etc. of Acute Coryza," by the author in *Journal of the American Medical Association,* Nov. 14, 1885.

The physical diagnosis is based chiefly on negative points. Percussion elicits the resonance of health. The vocal resonance is unaffected. The respiratory murmur may be weakened, but is not otherwise altered. Suppression of the murmur over a portion of the chest may occur temporarily from obstruction of a bronchial tube by an accumulation of mucus. These negative points warrant the exclusion of pleuritis and pneumonitis. The dry and moist bronchial râles may or may not be present at the time when the explorations are made. Their presence and diffusion more or less over both sides are indicative of the existence of the disease, but the absence of the disease is not to be inferred because these signs are wanting when the chest is examined. They are oftener wanting than present, the conditions giving rise to the dry râles not existing, and the mucus in the bronchial tubes being too adhesive or thick to produce bubbling sounds.

PROGNOSIS.—Acute ordinary bronchitis, not associated with other affections, is a dangerous disease only when it occurs in the young, the aged, or the feeble. The danger in these classes of patients arises from the accumulation of the products of inflammation within the bronchial tubes, and from the occurrence of collapse of pulmonary lobules or broncho-pneumonia. The danger relates to apnœa. The disease does not lead to great prostration and danger from asthenia. Exclusive of the classes of patients just named, it is generally a disease of little or no gravity. In the great majority of cases it passes through its course and the recovery is complete, but it may eventuate in chronic bronchitis. Associated with certain other affections, it may give rise to much suffering and danger. Occurring, for example, in patients affected with pulmonary emphysema, it occasions more or less dyspnœa, and it may prove fatal if the amount of emphysema be great. So in cases of spinal paralysis affecting the costal respiratory movements the accumulation of the bronchial secretions, from inability to expel them by the action of the diaphragm, alone may occasion death by slow apnœa.

The disease has no tendency to induce either pleuritis or acute lobar pneumonitis, and, contrary to a general impression, it rarely precedes the development of pulmonary phthisis.

TREATMENT.—Acute bronchitis may be prevented in some instances, on the appearance of coryza, by a full opiate. A quarter of a grain of the sulphate of morphia, half a grain or a grain of codeia, a proportionate dose of any of the preparations of opium, or ten grains of either Dover's or Tully's powder, may be given to an adult for this object at bedtime, accompanied by a hot pediluvium and some warm stimulating drink, such as weak punch or toddy or an infusion of some of the aromatic herbs, and followed in the morning by a saline purgative. If this plan of treatment do not succeed, it may mitigate the severity of the disease. A full dose of a salt of quinia (a scruple to an adult) will often act as a prophylactic, or, if given at once when the bronchial tubes become affected, as an abortive remedy. Twenty grains of salicin, repeated once or twice after an interval of two hours, will be likely to prevent or arrest the disease. The Russian or Turkish bath is sometimes effective.

If it be true that the efficient cause is a parasitic organism, topical treatment for its destruction is a rational object of prophylactic treatment. Destruction of the parasite in the nostrils of course prevents the migration and colonization in the parts below. Certain topical applications within the nostrils have been found efficacious. One of these is the German remedy, known as the Brandt-Hagen remedy, consisting of carbolic acid 80 grains, alcohol ½ ounce, liquor ammoniæ 80 minims, and distilled water 2½ drachms. This liquid is poured upon a sponge placed within a paper cone, and the vapor inhaled through the nostrils. Camphor and quinia in equal parts combined, pulverized, and inhaled, is a popular remedy said to be efficacious.

If the disease become established, it does not claim active measures of treatment in view of the almost uniform tendency to recovery with mere attention to hygiene. Bloodletting is not called for except in some rare cases in which the patient is full-blooded and the symptomatic fever unusually intense. Depletion by means of saline laxatives and reduced diet will generally suffice. If the attack be severe, confinement to the bed for a few days may be advisable in order to secure uniform warmth and moisture of the surface. Patients are more comfortable when moderately perspiring. In severe cases an abundance of moisture in the apartment soothes the inflamed membrane and renders the patient more comfortable.

Opium is thought by many to be contraindicated in the first stage. It is supposed to interfere with the free secretion of mucus and to render expectoration difficult. This is an inference from the effect of opium on the secretions in health ; but, so far from these results being produced, opium hastens the second stage. The free secretion of mucus is not the cause, but the consequence, of an abatement of the inflammation, and by contributing to the latter opium virtually acts as an expectorant. Opium, therefore, is indicated in the first stage of bronchitis, as it is in most acute inflammations. In the second stage it is indicated only when the cough is out of proportion to the expectoration ; that is, when the cough is more than is needed to free the bronchial tubes. Opium is contraindicated if, owing to the feebleness of the patient, the efforts of expectoration are inadequate to prevent accumulation in the bronchial tubes. The inhalation of warm vapor or spray affords relief and promotes the secretion of mucus.

During the acute stage, if pain and soreness of the chest be prominent symptoms, sinapisms or stimulating liniments are useful. These symptoms do not demand blisters or other active measures of counter-irritation.

The stimulating so-called expectorants are not indicated in the first stage. In so far as they have the property of exciting the bronchial mucous membrane they tend to increase the inflammation. They are rarely indicated in the second stage. If the affection linger and threaten to become chronic, the sulphate of quinia and other tonic remedies, together with a nutritious diet and an invigorating regimen, are the most efficient measures for recovery. I have been led by experience to regard the iodide of potassium or the hydriodic acid and chlorate of potassa as useful remedies in both the first and second stage of acute as well as of chronic bronchitis.

Subacute Bronchitis.

The difference, as regards symptoms and therapeutic indications, between the acute and subacute forms of bronchitis is such that a passing notice of the latter under a distinct head is called for.

Subacute bronchitis is, in common language, a slight cold, beginning usually with coryza, and running the same course as acute bronchitis, but with less severity, and perhaps not involving the same extent of the bronchial mucous surface. The affection is not of sufficient gravity to keep patients within doors, and in the majority of cases they do not call upon the physician, but either allow it to run its course or resort to some domestic remedies.

This form of bronchitis may frequently be arrested; but if not it claims only a saline laxative, followed by a little anodyne, such as one-eighth of a grain of the sulphate of morphia or a quarter of a grain of codeia every four or six hours in syrup. It is not necessary to enjoin confinement within doors. As it is desirable not to exaggerate, as well as not to depreciate, the importance of diseases, it should be understood by physicians and patients that common colds do not tend to eventuate in inflammation of the lungs or in phthisis. A certain amount of care is advisable and mild remedies are useful, but it would be injudicious to subject patients to active measures of treatment or to needless precautionary restrictions.

Acute Bronchitis in Young Children.

Acute bronchitis in young children offers an important peculiarity in the greater liability to collapse of pulmonary lobules and broncho-pneumonia. (See p. 174.)

Collapse of pulmonary lobules is incidental to bronchitis in young children, in consequence of the accumulation of mucus in the bronchial tubes. The tubes consisting of branching cylinders diminishing in calibre, it is easy to understand that plugs of viscid mucus in certain situations may obstruct the ingress of the inspired air into the air-cells, without obstructing the egress of air from the cells in expiration; hence collapse occurs of the lobules to which the obstructed tubes are distributed. In expiration the plugs are moved by the current of air from the smaller into the larger tubes, but in inspiration the current moves them into a position in which the progress of air is interrupted. It is supposed that collapse may occur when the current of air in expiration as well as in the act of inspiration is obstructed, the air which is thus pent up in the lobules being absorbed. In proportion to the number of collapsed lobules the respiratory function is compromised, and danger from apnœa may attend this accident. It is more liable to occur in young children than in adults, because children do not promote expectoration by voluntary efforts, and also because the respiratory acts are less vigorous. It occurs especially in feeble children. It does not, however, belong exclusively to acute bronchitis in young children; it occurs in aged persons, and may take place in those of any age who are constitutionally weak. It is also incidental to chronic bronchitis in children and adults, and it enters, as will be seen hereafter, into the causation of emphysema of the lungs.

The occurrence of collapse of pulmonary lobules may be strongly suspected in the course of acute bronchitis if the respirations become frequent, with dilatation of the alæ nasi, accompanied perhaps with lividity, and if the symptoms and signs of acute lobar pneumonitis be wanting. Physical exploration does not always furnish definite signs of this accident. Relative dulness on percussion may be found on one side if there be a marked disparity between the two lungs as regards the number of lobules which are collapsed. The respiratory murmur is more feeble over the posterior than over the anterior portion of the chest, and if relative dulness on percussion exist on one side the murmur of respiration will be more feeble on that side. Mucous râles are more or less abundant.

In the TREATMENT of bronchitis in young children the liability to this accident is to be considered. Mild emetics are indicated with a view to promote the expulsion of mucus from the bronchial tubes. They are admissible because they occasion less disturbance than in adults. They are indicated because in young children the acts of coughing are for the most part purely reflex; that is, voluntary efforts to expectorate are not made. They should not, however, be repeated too frequently, and antimonial emetics should never be employed. Apomorphia, administered hypodermically, has been recommended as an emetic and expectorant; but the grave effects which have sometimes followed small doses render the use of this remedy of doubtful expediency, especially in young children. Remedies to maintain nausea are contraindicated on account of their depressing effect; an emetic should be given to produce prompt vomiting, and afterward nauseant remedies withheld until a repetition of the emetic be deemed advisable. In the interval, tonic remedies, a nutritious diet, and perhaps alcohol, are called for to support the general strength. Opium is to be given with great circumspection, since it may favor the accumulation of mucus in the bronchial tubes. The chief objects of treatment, in short, are twofold: *first*, to supply the want of voluntary efforts of expectoration by the judicious employment of emetics; and *second*, by sustaining measures to maintain the force of the respiratory acts. The iodide of potassium, given pretty freely, has seemed to me to have a curative effect. Measures to excite deep inspirations, referred to in connection with broncho-pneumonitis (vide p. 176), are to be mentioned as entering into the treatment.

Acute Bronchitis affecting the Small Bronchial Tubes.

An extension of inflammation from the larger and medium-sized tubes to those of small size gives rise to the variety of bronchitis commonly called capillary bronchitis. This application of the term capillary is not strictly correct. The truly capillary tubes—that is, the ultimate subdivisions of the bronchial tree, or the bronchioles—are not the seat of inflammation in this affection. They are involved in pneumonitis. The bronchial branches of small size, but not the smallest, are affected in so-called capillary bronchitis. The larger tubes are generally first affected, and the small tubes become involved by a more or less rapid extension of the inflammation. The affection has heretofore been known by various titles, such as catarrhus senilis, peripneumonia notha, bastard pleurisy, suffocative catarrh, etc.

This affection differs widely from ordinary acute bronchitis as regards its clinical history and danger. It is an exceedingly grave affection, proving fatal, especially in young children, in a large majority of cases. The danger proceeds from obstruction to the current of air to and from the air-vesicles. The obstruction is due to the small size of the affected tubes; the swelling of the mucous membrane and the presence of muco-purulent liquid, which in the larger tubes do not interrupt the free passage of air, occasion in the small tubes serious interference, and, as the affection is bilateral, fatal apnœa is liable to be produced.

In acute bronchitis of the small bronchial tubes the inflammation is much more intense than in acute simple bronchitis of the large tubes. The small bronchi contain muco-pus, which escapes in small drops when a section of the lungs is made. Upon microscopical examination the walls of the affected portions of the bronchi are found infiltrated with emigrated white blood-corpuscles, and frequently inflammatory products exist in the air-cells immediately adjacent to the inflamed bronchi. Obstruction from plugging of the tubes with muco-pus leads to collapse of pulmonary lobules. These collapsed lobules become secondarily inflamed. It is thus seen that the lesions are not confined to a simple inflammation of the mucous membrane of the small bronchi, but embrace also inflammation of the other coats of the bronchi and of the air-cells in connection with the bronchi. There is, therefore, in most cases, broncho-pneumonitis. The lungs are generally hyperæmic, and are often œdematous. There is usually compensatory emphysema of the superior and anterior parts of the lungs.

The mechanical obstruction and the incidental circumstances just stated afford an explanation of the symptoms which make up the clinical history of this variety of bronchitis; and as regards its clinical history it presents a striking contrast to acute bronchitis limited to the larger tubes. The respirations are frequent in proportion to the bronchial obstruction. In young children they may be increased to 60 or 70 per minute. Dyspnœa exists in a degree corresponding to the acceleration of breathing. In severe cases it amounts to orthopnœa, the patient keeping the sitting posture and suffering extremely for the want of breath. The speech is short and jerking; the alæ nasi dilate; the face is congested and swollen; and the countenance expresses anxiety and distress. Lividity is marked, partly in consequence of imperfect oxygenation of the blood, but more especially of its accumulation in the right cavities of the heart (cyanosis). There is great restlessness. The pulse is notably frequent, but not accelerated in proportion to the respirations. Cough is more or less prominent, and is difficult and ineffectual from the want of breath. The expectorated matter coming from the small tubes does not contain bubbles of air; its specific gravity is greater than that of water, and it has sufficient consistence to preserve the form of the tubes. In its passage along the large tubes it becomes mixed with mucus from the latter, which contains air and consequently floats on water. The characteristic expectoration consists of mucus floating on water, with which are connected filaments sinking below the surface.

The symptoms distinguish sufficiently capillary bronchitis from bronchitis limited to the larger tubes, but they do not always suffice for the discrimination of this from other affections. It should never be confounded with laryngeal affections occasioning obstruction (simple or pseudo-membranous laryngitis), for in these affections the voice is affected, the cough is characteristic, and the respirations are labored, but not notably accelerated. Attention to the points just named will show the affection to be seated within the chest. The diseases with which it is most liable to be confounded are pneumonitis, asthma, and ordinary acute bronchitis with collapsed lobules. It may generally be discriminated from these affections by means of physical signs taken in connection with the symptoms. The resonance on percussion is not diminished in capillary bronchitis, and over the upper lobes it may be greater than in health, owing to an emphysematous condition of the air-vesicles. The presence of muco-purulent liquid in the small tubes gives rise to fine bubbling (subcrepitant) râles. As the affection is bilateral, the râles are heard on both sides, and especially on the posterior aspect of the chest. The respiratory murmur is weakened. The anterior, superior, and middle portions of the chest in a young child may become largely dilated, presenting the characteristic deformity of long-continued and great emphysema, this appearance disappearing after recovery.

Lobar pneumonitis is to be excluded by the absence of the symptoms and signs diagnostic of that disease. Dulness on percussion extending over one or more lobes is wanting. Pneumonitis is generally abruptly developed, and is not preceded or accompanied by bilateral bronchitis. It is rare for pneumonitis affecting but one lobe to occasion so much disturbance of the respiration as takes place in severe cases of capillary bronchitis. The crepitant râle, if it be present, denotes pneumonitis, and if this affection exist and have advanced far enough for bubbling râles to become developed, the latter are confined to one side only, except in the rare instances in which the pneumonitis is double. The temperature of the body is, as a rule, higher than in capillary bronchitis, the thermometer in the latter rarely showing an elevation above 102° F.

Asthma is not accompanied by pyrexia. The respirations are not rapid, but labored, the labor being especially marked in expiration (expiratory dyspnœa). They are accompanied by loud sibilant and sonorous râles. It is a paroxysmal affection, the dyspnœa not persisting as in capillary bronchitis; and unless the case happen to be one in which asthma is experienced for the first time, the liability of the patient to this affection is known.

The discrimination from acute bronchitis of the larger tubes with collapsed lobules or broncho-pneumonitis is more difficult. The differential diagnosis must be based on the greater gravity of the symptoms, the clearness of the resonance on percussion, and the diffusion over both sides of the subcrepitant râles in cases of capillary bronchitis.

Extensive broncho-pneumonitis supervening upon capillary bronchitis of course increases the gravity of the local symptoms and the danger. A rise of temperature to 103° or 104° renders this complication probable, but by no means certain, for, exceptionally, these temperatures are observed in cases of capillary bronchitis without the pneumonic complication. The occurrence of the latter cannot always be ascertained by means of physical signs.

Pleuritis with large effusion may give rise to accelerated breathing, lividity, etc., but this affection is readily excluded by the absence of the signs denoting the accumulation of liquid in the pleural cavity.

Capillary bronchitis occurs especially in young children, and next in frequency in the aged, but it is occasionally met with in adults. It is always a disease of gravity, but particularly so at the two extremes of life. In young children and the aged it proves fatal in a very large proportion of cases. It destroys life in some cases rapidly. I have known it to prove fatal in less than twelve hours after the symptoms denoted gravity of disease. The average duration in fatal cases in children is from three to five days. The duration is longer in adults, and the disease is much less grave after childhood and during middle age. In cases ending in recovery, convalescence becomes established after a duration varying from a week to two weeks. Happily, it is by no means a disease of frequent occurrence even in young children. In the mode of dying apnœa predominates; and the progress toward a fatal termination is denoted by increasing lividity, the pulse becoming more and more frequent and feeble, the skin covered with clammy perspiration, the labor and frequency of the respirations, together with cough and expectoration, diminishing toward the close of life from muscular prostration. Thrombosis, or the formation of a clot in the right side of the heart, is an occasional accident which may prove the immediate cause of death.

Treatment.—Bloodletting may be employed at the onset under those conditions by which the employment of this remedy in other inflammatory affections is to be regulated. It is not admissible in young or feeble subjects, and in employing this or any other debilitating measure the physician should not lose sight of the fact that it is important to economize the strength of the patient in order that the laborious efforts of breathing may be carried on for a length of time sufficient for the resolution of the disease. The danger and distress incident to the disease arise chiefly from the deficiency of fresh supplies of air in the air-cells; and if bloodletting be injudiciously practised, the patient suffers from the want of both blood and air. The importance of economizing strength should also enforce circumspection in the use of saline depletants and sedative remedies. These should not be pushed to the extent of producing prostration. The difficulty consists in mechanical obstruction, which neither these nor any therapeutical measures will immediately remove. The safety of the patient depends on the maintenance of life until the disease passes through its career. The inhalation of oxygen affords marked relief of the dyspnœa. Breathing warm vapor is not only highly useful as a palliative measure, but it does not a little toward promoting resolution of the inflammation. It should form an important part of the treatment; and the most effective method is to keep the atmosphere of the room constantly and abundantly charged with steam during the course of the disease. Water in several vessels should be kept at the point of ebullition by means of heat in fireplaces, stoves, or burning gas. The temperature of the room should be kept at a high point, 85° to 90°. The iodide of potassium should be given freely.

Emetics in young children are advisable for a reason already stated—namely, to supply the want of voluntary efforts of expectoration. Care, however, is to be taken not to repeat them so often as to occasion exhaustion. The use of apomorphia has been already referred to (p. 225). Laxatives are useful by diminishing the contents of the intestines, and thereby giving space for more effective action of the diaphragm in respiration. Revulsive applications to the chest are useful, but blisters are of doubtful utility.

The great object being to carry the patient safely through the disease, supporting measures are early indicated, and they are important in proportion as the symptoms denote failure of the vital powers.

Epidemic Bronchitis—Influenza.

For the last four or five centuries medical observers have noted the occurrence from time to time of an epidemic affection characterized by bronchitis. In most countries it is commonly known by the name *influenza*, after a term introduced by the Italian writers in the seventeenth century. In France it is called *la grippe*. As an epidemic it is remarkable for its extensive and rapid diffusion, sometimes extending within a brief period over many different and widely-separated countries. During its prevalence a vast number of persons of all ages are simultaneously affected. In some epidemics the fatality has been large, but in most fatal cases other affections are developed, especially the so-called capillary bronchitis and broncho-pneumonitis. The fatality is limited chiefly to the aged and to persons with feeble constitutions.

The inflammation of the nostrils which usually precedes the bronchitis often extends into the frontal and maxillary sinuses, to the lachrymal ducts and conjunctiva, and into the Eustachian tube. Frontal headache is a prominent symptom. Epidemic bronchitis is accompanied by more marked general symptoms than the sporadic form—namely, chills, pyrexia, lassitude, debility, anorexia, etc. In the epidemic much oftener than in the sporadic affection the general symptoms seem to be out of proportion to the bronchial affection, and to denote an essential fever rather than a purely local disease. It often ends in free perspiration or with diarrhœa.

A special cause is involved in the production of this as of every other epidemic disease. The nature, source, and mode of action of the special cause are not established. It has been attributed to ozone in the atmosphere by Schœnbein and others, to animalculæ by Holland, and to cryptogamic productions by the late Prof. J. K. Mitchell. The special cause doubtless exists in the atmosphere, and is independent of appreciable atmospheric changes. This fact was exemplified in the Massachusetts General Hospital during the extensive prevalence of this epidemic in 1832. Nearly all the patients in the hospital were affected, although the wards were kept day and night at a uniform temperature. Being an infectious disease, the parasitic doctrine not only offers a rational view of its etiology, but, reasoning from analogy, is a logical inference.

The disease is generally mild, but, as already stated, it is liable to lead to the development of local affections other than bronchitis, and may in this way prove fatal. Gairdner's observations go to show that during the prevalence of influenza other diseases are usually severe, and that the rate of mortality from all diseases is increased. The duration of an attack of influenza is from three to six days.

In the treatment of epidemic bronchitis bloodletting is rarely if ever required. In healthy, vigorous subjects brisk purging has seemed to me useful. This should not be resorted to if the patient be feeble or advanced in years. Aside from purgation, diaphoretic remedies and opium will meet the therapeutic indications. Associated affections claim appropriate treatment, but debilitating or depressing measures are to be employed with greater circumspection than if the affections were primary. In aged and feeble persons tonic and supporting measures may be highly important. The remarks in reference to the disinfectant treatment of sporadic bronchitis (p. 223) are of course applicable to influenza.

Bronchitis with Fibrinous Exudation.

A variety of bronchitis is characterized by a fibrinous exudation on the inflamed mucous surface. This characteristic feature is expressed by the names diphtheritic, pseudo-membranous, plastic, croupous, or croupal, which have been applied to it. It is frequently associated with inflammation of the larynx and trachea, attended with a similar exudation, constituting the affection known commonly as true croup and occurring also in certain cases of diphtheria. But reference is here made to bronchitis with this peculiarity occurring independently of a similar affection of the larynx and trachea. Exclusive of the cases in which it accompanies croup and diphtheria, it is a rare variety of bronchitis. The exudation is similar to that in the bronchitis secondary to croup. It forms a dense membraniform layer, which at first is closely agglutinated to the mucous surface, but sooner or later, loosened and detached by a suppurative process, is expectorated. When expectorated, the false membrane, as it is called, either has the form of patches or strips, or it is thrown off entire from portions of the bronchial tubes, and when carefully displayed may be found to consist of casts representing more or less of the bronchial subdivisions. Aside from the form of casts of the tubes, the fibrinous character of the expectorated matter may be ascertained by microscopical examination and by chemical reagents. It presents the fibrillæ which characterize exuded fibrin in other situations. Placed in a solution of acetic acid, it becomes swollen, whereas mucus contracts and is thereby rendered more dense.

The fibrinous casts are expectorated in a globular mass enveloped in mucus, and without close examination they are liable to be overlooked. When the mucus is carefully removed, the casts, displayed in liquid, sometimes represent a bronchial stem of considerable size with subdividing branches which have extended to the smallest ramifications. These exudative casts are to be distinguished from those produced by the coagulation of blood within the tubes in some cases of bronchorrhagia. The latter do not, like the former, present laminæ or concentric layers, and they contain blood-corpuscles in greater or less quantity. They are of course preceded by hæmoptysis.

The inflammation in cases of bronchitis with fibrinous exudation is either acute, subacute, or chronic. Occurring in young children, it may occasion sufficient obstruction to seriously compromise respiration, and it frequently proves fatal. Affecting adults, however, if not complicated with other grave affections, it rarely destroys life, and in the majority of cases the recovery is complete. I have met with a case in which it was associated with asthma and emphysema. Fibrinous bronchitis has been associated in many cases with phthisis. I have known it to occur in connection with acute lobar pneumonitis.

There are no symptoms or signs by means of which the diagnosis of this variety of bronchitis can be made out prior to the expectoration of false membrane or bronchial casts. When this occurs the character of the affection is of course manifest. This expectoration is preceded and accompanied by violent paroxysms of coughing. It is followed by a muco-purulent expectoration, which may be bloody. The symptoms are relieved by the removal of the fibrinous exudations or casts. The expectoration of the latter may be repeated after intervals of days, weeks, months, or even years. The character of the disease, if it prove fatal before the expectoration of false membrane takes place, may be suspected, but the symptoms and signs show only the existence of bronchitis with obstruction of bronchial tubes and perhaps collapse of pulmonary lobules. This variety of bronchitis sometimes recurs repeatedly in the same person. I have met with cases in which a large number of bronchial casts (forty or fifty) were expectorated at as many different times.

The principles of treatment are essentially the same as in cases of acute or chronic bronchitis without this peculiar feature, and they need not be separately considered. Warm vapor or spray inhaled will aid in the separation of the false membrane.

Circumscribed Bronchitis.

As already stated, bronchitis when circumscribed is incidental to some other pulmonary disease. It is developed in pneumonitis, being limited to the lobe or lobes affected, and is the chief source of the expectoration in this disease if it do not go on to the suppurative stage. It occurs in pulmonary phthisis, and is limited to the tubes in proximity to the seat of the deposit of tubercle. The bronchitis in this disease furnishes the expectoration prior to the softening and discharge of the tuberculous product. As thus developed, bronchitis claims no special attention in a therapeutical point of view.

Chronic Bronchitis—Fetid Bronchitis.

Chronic bronchitis may follow the acute form or it may be subacute from the beginning. It is far less frequent than acute bronchitis. It occurs much oftener in aged persons than during the early and the middle period of life. It is associated with asthma, pulmonary emphysema, and cardiac lesions, especially those involving mitral stenosis or incompetence of the mitral valve.

The hyperæmia is less intense than in acute bronchitis. The color of the mucous membrane may be bluish-red or it may be grayish or slate-colored. The morbid products upon the membrane are similar to those in acute bronchitis, but usually with greater predominance of mucus. More extensive changes, however, may occur in the bronchial wall.. This, on the one hand, may become thickened by the development of new connective tissue, or it may become thinned in consequence of atrophy of the connective tissue and muscular fibres. With the production of new connective tissue little papillary excrescences are often formed upon the mucous membrane. In consequence of loss of elasticity of the bronchial wall from destruction of its elastic fibres, in some cases there results dilatation of the tubes, or bronchiectasis. The pulmonary tissue immediately surrounding a dilated bronchus is usually indurated by increase of the interstitial tissue. Three forms of bronchiectasis may be distinguished—namely, the cylindrical, the fusiform, and the sacculated. Bronchiectasis may accompany simple chronic bronchitis, but it is more likely to be produced when the bronchitis is associated with some affection giving rise to dyspnœa, such as emphysema, old pleuritic thickening with adhesions, fibroid phthisis, etc. Sometimes one or more large cyst-like cavities, with smooth walls lined by ciliated epithelium, are found in the lung. These are bronchiectases, and may be sometimes congenital.

The SYMPTOMS relating directly to the bronchial inflammation are essentially the same as in acute bronchitis, differing only in intensity; but connected with these symptoms frequently are others proceeding from associated affections.

Pain is generally wanting, or the patient complains only of a sense of uneasiness or constriction beneath the sternum. If cough be frequent and violent, soreness may be felt at the base of the chest on both sides or at the epigastrium, due to the traction of the diaphragm on the ribs. Febrile movement is slight or wanting. A slight exacerbation of fever at night is observed in some cases. The appetite may continue good or it may be more or less impaired. The nutrition is frequently not much if at all affected. The loss of weight with the continuance of the disease is not marked, as it usually is in cases of pulmonary phthisis. The patient usually does not present pallor or a notably morbid aspect.

More or less cough always exists. In frequency and severity it varies much in different cases. It is paroxysmal, violent, and difficult in proportion to the small quantity and tenacity of liquid products within the affected tubes and in proportion as the smaller tubes are involved. The expectoration also varies much in different cases. It is sometimes abundant, consisting of mucus with serous transudation. When the latter is copious the affection has been called *bronchorrhœa.* It may consist of large, solid, greenish or ash-colored sputa, and in some cases small pearl-like masses are raised after much coughing. In cases characterized by the latter the disease has been called, after Laennec, *dry catarrh.* The sputa may at times be streaked with blood. In general the expectoration is muco-purulent, the purulent characters not infrequently predominating, and sometimes it appears to consist of pure pus. Formerly it was thought to be highly important to determine whether the expectoration were purulent or not, the presence of pus being supposed to denote something more than bronchitis. This is now deemed a point of comparatively little importance since it is ascertained that pus is formed on a mucous surface. A very copious expectoration of pure pus, however, does point to some other source than bronchitis, such as the discharge of a tuberculous abscess, the third stage of pneumonitis, abscess of the liver, evacuating through the lung, empyema with perforation, etc. The diversified characters which belong to the expectoration in different forms of the disease are of less value in a diagnostic view than was formerly supposed, since the knowledge which has been acquired of physical signs furnishes a far more reliable source of information respecting intrathoracic conditions. The obvious differential characters by which mucus and pus may be distinguished from each other are as follows: Mucus is transparent or semi-transparent; it is more or less viscid; it is stringy and not miscible with water; it retains bubbles of air, and consequently floats on water. Pus is opaque, less viscid than mucus, miscible in water, and in water it sinks to the bottom. These differential characters are combined in varying proportions when the matter of expectoration is muco-purulent.

The matter of expectoration in some cases of chronic, as also of acute bronchitis, is notably fetid, having the characteristic odor of decomposed animal substances. The affection is then said to be *fetid bronchitis.* Generally, this characteristic fetor denotes either gangrene of lung, bronchitis with dilated bronchial tubes, or decomposition of matter within phthisical cavities; but it may occur independently of any of these conditions. Gangrene may be excluded by the absence, on microscopical examination, of any appearance of expectorated pulmonary tissue, and dilated tubes by the absence of the signs of that physical condition. The diagnosis is to be made by excluding these diseases, together with pyothorax and perforation of lung, the expectoration in the latter sometimes having a notable degree of fetor. The explanation of fetid bronchitis is that circumstances pertaining to morbid products within the tubes are favorable for the action of the putrescence-producing bacteria derived from the atmosphere. These bacteria, together with acicular crystals of the fatty acids, are found in the sputum, particularly in little whitish particles present therein.

Chronic bronchitis of course involves the same causes as the acute in the cases in which it is preceded by the latter. It may be produced and maintained by the inhalation of irritating particles of stone or metal in the exercise of certain occupations. Its continuance in some cases is referable to organic affections of the heart, consisting of obstructive or regurgitant mitral lesions. These act by inducing pulmonary congestion. It is an affection incident to old age, occurring without any obvious causative agencies. It occurs in youth and middle age irrespective of apparent causes, and is often associated with asthma and emphysema. To these affections it stands in the relation of a cause rather than an effect. In cold latitudes it is sometimes manifestly dependent on climatic causes, recurring with each successive winter, and disappearing during the summer season or on removal to a warm climate ("winter cough").

It is not an affection which tends directly to destroy life. In the aged it may sometimes predispose to the development of capillary bronchitis, and thus indirectly prove serious. In connection with the feebleness of advanced years it may lead to collapse of pulmonary lobules, and in this way shorten life. Its existence in a patient prostrated with any other disease involves danger from the accumulation of morbid products in the bronchial tubes. It may lead to the development of asthma and pulmonary emphysema—affections which, although not immediately dangerous, diminish the duration of life. Aside from these contingencies, the evil to be apprehended is the permanency of the affection. It is liable to become established, persisting for years, and in aged persons for the remainder of life. The existence of chronic bronchitis does not involve an increased liability to the development of pulmonary phthisis.

The DIAGNOSIS of chronic bronchitis involves discrimination from pulmonary phthisis. This would be difficult, and sometimes impossible, were the investigation to be limited to the symptoms; hence before physical signs were studied these two affections were of necessity confounded. The importance of the discrimination is obvious in view of the great difference between the affections as regards prognosis. The differential diagnosis is to be based on the absence of the symptoms and signs which are diagnostic of phthisis. In other words, it is concluded that the latter does not exist, from negative evidence or reasoning by way of exclusion. The absence of symptoms which belong to the clinical history of phthisis is also to be considered, such as progressive marked emaciation, hæmoptysis, and accelerated breathing. These symptoms are wanting in chronic bronchitis. The physical signs in this variety of bronchitis are the same as in the acute form—namely, the presence of dry and moist bronchial râles, the resonance on percussion and the vocal resonance unaffected, and the respiratory murmur weakened and sometimes suppressed over a portion of the chest from temporary obstruction of bronchial tubes, but its characters, aside from weakness, unchanged. The parasitic doctrine of phthisis has rendered the microscopical examination of the sputum important in the differentiation of this affection from chronic bronchitis. The absence of the bacillus tuberculosis in a single specimen, however, is not sufficient to exclude phthisis, but the negative result in repeated examinations goes far toward the exclusion of that disease.

The coexistence of pulmonary emphysema is to be determined by symptoms and signs diagnostic of this affection, which will be considered in the next chapter.

In the TREATMENT of chronic bronchitis bloodletting or other depletory measures are never indicated. The affection seldom exists under circumstances which render it desirable to lower the powers of life. Counter-irritation by means of croton oil or stimulating liniments is sometimes useful. The diet should be nutritious, but not stimulating, consisting of milk, eggs, fowl, fish, and farinaceous articles. Meat, however, is not to be interdicted if the system be enfeebled. Wine and alcoholics are to be avoided, except when measures to support or strengthen the system are indicated. Attention to the surface of the body is important. In a cold or variable climate woollen or silk garments should be worn next the skin. An undergarment of buckskin or chamois leather, worn over light woollen or silk, affords during cold weather great protection and saves the inconvenience of an undue quantity of clothing. The object is to secure uniformity of the temperature of the surface and to maintain the functions of the skin.

Medicines may be employed with a view to palliation and cure. If cough be troublesome, exceeding that required for expectoration, soothing remedies are called for. Opium, however, is to be prescribed with circumspection; in the first place, lest the habit of using it be formed—a consideration to be taken into account in all chronic affections; and in the second place, in feeble subjects and under circumstances in which there may be danger from an accumulation in the bronchial tubes, serious consequences may follow blunting that sense of the presence of morbid products which induces acts of expectoration. Moreover, the use of opium tends to impair the digestive powers. For these reasons other anodynes, such as hyoscyamus, conium, belladonna, and hydrocyanic acid, are generally to be preferred.

As a rule, the remedies which are called expectorants are not indicated. The nauseant expectorants do harm by their depressing effect and by disturbing the appetite and digestion. The stimulant expectorants, such as squills, senega, etc., are of doubtful efficacy, and if not useful are more or less hurtful.

Certain remedies, however, sometimes exert a curative effect. The iodide of potassium (or the iodide of ammonium) is one of these, and perhaps the one most likely to be successful. Five or ten grains three or four times daily may be given at first, and before abandoning the remedy as useless the dose should be increased to the limit of tolerance. This remedy is not always tolerated even in moderate or small doses, and it is by no means always successful, even when large doses occasion no inconvenience. The muriate of ammonia is in some cases a curative remedy. I have known it to effect a prompt cure, the disease having existed for ten years. The chlorate of potassa, half an ounce of a saturated solution given twice or three times daily, in some instances proves highly useful and may effect a cure. The balsam of copaiba is a remedy the value of which in this disease is not generally appreciated. It has no value in certain cases, but its curative effect is sometimes so marked that it would seem to be almost a specific remedy. The spirits of turpentine occasionally prove equally efficacious. A good method of giving the latter is in what the late Prof. Clark of Boston called the continuous dose—namely, a drop in an emulsion every one or two hours throughout the day. Other balsamic remedies, the balsams of Tolu, Peru, and sandalwood, are among the curative remedies, to which are to be added tar and the compound tincture of benzoin. The bromine salts are to be mentioned as useful remedies. This list might be extended. As an illustration of the great number of remedies which have been found useful in bronchitis, the National Dispensatory enumerates under this head one hundred and twenty-two articles.

Frequently, marked benefit as regards the bronchitis is derived from tonic remedies in conjunction with measures to invigorate the system. The salts of quinia and the preparations of iron are often highly useful. These and other tonic remedies are indicated if the powers of the system be reduced or the patient be anæmic. A generous diet is to be conjoined, and wine or alcoholics may be taken moderately with advantage. Out-of-door life in such cases should be added. This plan of treatment is applicable to a pretty large proportion of the cases of chronic bronchitis; and to improve the general health and restore vigor are important objects in the treatment of this as of any other chronic local affection.

It is an object of treatment in some cases to diminish the amount of expectoration when this is excessive. For this end astringent remedies may be prescribed, such as tannic acid, the persulphate or pernitrate of iron, and the preparations of zinc. After a bronchitis, however, has become habitual, especially in the aged, it sometimes appears to be, as it were, an element of health. At all events, patients complain of the discomfort arising from the suspension or diminution of the expectoration, and for this reason opiates do not afford relief. Under these circumstances expectorants are useful as palliatives.

The inhalation of the spray from medicated liquids, produced by an atomizer, may be resorted to for several objects. A solution of the astringent remedies just named may sometimes be useful as a topical application in the form of a spray, with a view to diminish profuse expectoration. Oftener it is an object to facilitate expectoration. Spray from a solution of the muriate of ammonia—gr. j to gr. xv to the ounce—is useful in this way. Opium and other narcotics brought into direct contact with the affected membrane may be of service by allaying the irritation which occasions troublesome superfluous cough. The vapor of tar is a soothing application. In fetid bronchitis antiseptic inhalations are indicated. For this object the vapor of the oil of turpentine—a solution of carbolic acid from gr. j to grs. iv to the ounce, in the form of spray—may be inhaled, or a similar strength of a solution of permanganate of potassa. The bisulphite of soda in scruple doses, given every two hours by the mouth, is an efficient antiseptic. Medicated spray is to be employed with care as regards the strength of the solutions atomized and the frequency or continuance of the inhalations, bearing in mind that remedies held in solution pass rapidly into the blood and may produce toxic effects.

Change of climate is sometimes advisable. A removal from a cold and variable climate to a situation in which the temperature is mild and equable may be attended with marked relief, and perhaps lead to recovery. Benefit may be looked for especially in the cases in which there is either immunity from the disease or marked amelioration during the summer season.

CHAPTER X.

EMPHYSEMA OF THE LUNGS.—ASTHMA.—PERTUSSIS.

Emphysema of the Lungs: Varieties; Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Asthma: Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Pertussis, or Whooping Cough.

Emphysema.

EMPHYSEMA in general signifies the presence of air in the interstitial connective tissue. In the lungs, however, it is necessary to distinguish two kinds of emphysema. In one form there is an escape of air into the interlobular and subpleural connective tissue. This form is analogous to emphysema in other situations, and is called *interlobular* or *interstitial emphysema*. The other and the more common variety is called *vesicular emphysema*, and consists in an abnormal accumulation of air within the air-cells, whereby they become distended and their walls often atrophied.

Interlobular emphysema arises from rupture of the air-cells, causing a communication between them and the interlobular connective tissue. This sometimes follows perforating wounds of the chest or injury of the lung by the extremity of a fractured rib. It may also result from violent paroxysms of coughing, as in whooping cough, especially when the lung has been previously affected with vesicular emphysema. Its favorite situation is the anterior border of the upper lobe. Just beneath the pleura little air-bubbles, often arranged like rows of beads, can be seen in the course of the septa between the lobules. The air sometimes detaches the pleura so as to form air-blebs of considerable size. A subpleural air-tumor may be as large as an English walnut or even larger. Bouillaud has reported a case in which an air-tumor existed as large as the stomach. It is important not to mistake for interlobular emphysema a post-mortem generation of gas in the interstitial tissue of the lungs as a result of decomposition—a condition not infrequently found after death. In rare cases the subpleural air-blebs rupture during life, and pneumothorax, with or without pleuritis, results. Another very rare occurrence is the passage of the air from beneath the pleura along the root of the lung into the connective tissue of the mediastinum, and thence into the subcutaneous tissue of the neck. In this way the emphysema may become diffused over more or less of the body.

Vesicular emphysema—and this is usually meant when the term pulmonary emphysema is used—may occur as an independent affection preceded or accompanied by chronic bronchitis. This is known as *substantive* or *idiopathic emphysema*, in distinction from *vicarious*, *compensatory*, or *symptomatic emphysema*, the latter terms being applied to an abnormal accumulation of air in the air-cells of certain portions of the lung when the penetration of air into other portions has been obstructed; as, for instance, by pneumonitis, tuberculous deposits, tumors, effusions in the pleural cavity, pleuritic adhesions, occlusion of the bronchi, etc. Vicarious emphysema does not necessarily appear in the immediate neighborhood of the obstructed alveoli. Like all forms of emphysema, it affects most frequently the anterior borders and the upper lobes of the lungs. When one lung is compressed or hepatized the other is often emphysematous.

The names *acute emphysema* and *insufflation* are given to a dilatation of the air-cells frequently met with in the lungs of those who have suffered from severe dyspnœa during the last days or hours of life. It is usually observed in the apices and the borders of the lungs. The air-cells may be dilated to twice their normal volume. The elasticity of the alveolar walls is impaired, but in distinction from chronic emphysema they present no evident structural changes. Acute emphysema or insufflation of the lungs accompanies chiefly acute diseases of the respiratory organs or other diseases terminating rapidly with dyspnœic symptoms. It is due to repeated forced inspiratory efforts.

In a well-marked case of chronic vesicular emphysema the lungs are enlarged. Upon opening the chest they do not retract like healthy lungs, but remain in contact along their anterior borders, so that the pericardium may be covered. Their contractility is impaired. The diaphragm may be pushed down to the seventh or eighth rib, and the adjacent viscera correspondingly displaced. The heart is pushed downward and backward, and in consequence of hypertrophy and dilatation of the right ventricle, which are usually present, extends farther to the right than normal. The borders of the lungs are rounded and puffy; the emphysematous portions do not crepitate upon pressure or incision, as do normal lungs, but have a soft, cushiony feel. The imprints of the fingers remain a long time. The lungs appear in general pale, anæmic, and dry, but often present patches and striæ of black pigment. The dilated air-cells can be distinguished with the naked eye. The air-spaces, much larger than a pin's head, are the result of rupture of the interalveolar septa and coalescence of the air-cells. Such air-spaces or blebs frequently attain the size of a pea, and they may be considerably larger. Large air-blebs, from the size of a pea to that of a pigeon's egg, sometimes project above the lung-surface, and may become so detached as to be pedunculated. Upon microscopical examination it is seen that the dilatation begins in the alveolar passages and infundibula. They become expanded and the alveolar septa are less prominent. The alveolar walls are thinned, and finally become perforated, at first usually in their centres. The holes between the alveoli enlarge, and the air-cells coalesce, so that by continuation of the process larger and smaller air-sacs are formed. The process appears to be a gradual atrophy of all of the elements composing the alveolar walls. The finer elastic fibres early disappear, the larger ones diminish in size and frequently rupture, and the capillary network becomes coarser by obliteration of a certain number of capillary vessels. This destruction of pulmonary capillaries is not attended with hemorrhage, as the vessels become impermeable before they disappear. The most important symptoms of emphysema are referable to this loss of pulmonary blood-vessels. A partial compensation for this disturbance of the pulmonary circulation exists in the anastomoses between the pulmonary and the bronchial and subpleural vessels. Fatty particles are often found in the alveolar walls, probably from fatty degeneration of the alveolar epithelium and of the capillary endothelium. An apparent thickening of the alveolar walls may result from their being compressed and crowded together by dilated air-vesicles. Rindfleisch, however, claims to have seen an actual hypertrophy of the smooth muscular elements which, according to him, exist in the alveolar walls. There is nothing in the pathological appearances of chronic emphysema which supports the view, otherwise not improbable, that a primary vice of nutrition in the alveolar walls antedates the development of the emphysematous condition in many cases. Substantive emphysema is most developed in the upper lobes and along the anterior borders of the lung, also along the lateral borders of the base of the lung; but it may be diffused over the whole lung-surface and in its substance. It is accompanied by chronic bronchitis, which may either precede or follow the emphysematous condition. The bronchial mucous membrane is coated with muco-pus, the bronchial wall may be thickened, and sometimes dilatations of the bronchi are present. In consequence of the obliteration of pulmonary capillaries there is obstruction to the flow of blood through the lungs, leading to hypertrophy and dilatation of the right ventricle, and frequently to venous congestion in the various viscera. The enlargement of the heart is compensatory so long as hypertrophy predominates over dilatation; but it becomes a source of grave consequences when the latter predominates, giving rise to chronic congestions of the liver, spleen, kidneys, stomach, and intestines. These conditions are similar to those met with in valvular lesions of the heart, and will be more fully described in another connection.

The emphysematous condition, especially in vicarious emphysema, may be limited to a few vesicles. Its practical importance of course depends upon the extent of lung involved.

A pathological condition of the lungs which bears little resemblance to true emphysema has received the name of *senile emphysema.* It is also called senile atrophy of the lungs. Here, instead of an abnormal accumulation of air in the lungs, these organs are smaller and contain less air than normal. In consequence of atrophy of the alveolar walls the air-cells coalesce and form larger air-spaces. These, however, do not result from a dilatation of the alveoli, but from a gradual shrinkage and disappearance of the lung-tissue. These atrophic lungs are usually deeply pigmented, and take up less space than normal in the thoracic cavity. Senile atrophy of the lungs does not lead to hypertrophy of the right ventricle.

Emphysema developed in a person free from tubercle undoubtedly diminishes the liability to tuberculous disease. The two affections are rarely associated, exclusive of the cases in which pulmonary lobules in the vicinity of tuberculous deposits become secondarily emphysematous. In these cases the emphysema is vicarious. A reason for the protection against tubercle afforded by emphysema may perhaps be found in the diminished quantity of blood within the emphysematous portions of lung, and in the fact that the portions affected with emphysema—namely, the upper lobes—are the primary seat of tubercle in the immense majority of cases. The condition of emphysema is also, to some extent, incompatible with hæmoptysis. Pneumonitis, as a rule, is not developed in an emphysematous patient. I have met, however, several exceptions to this rule.

CLINICAL HISTORY.—Emphysema, existing to an extent to compromise considerably the respiratory function, is characterized by habitually labored breathing and inability to take active exercise without suffering from want of breath. The labored breathing is manifested especially in expiration. The rhythm of the respiratory acts is altered; the inspiration is shortened and the expiration prolonged. As chronic bronchitis generally coexists, cough and expectoration are habitually more or less prominent as symptoms. The cough is usually violent, paroxysmal, and characterized by a series of prolonged, spasmodic expiratory efforts, as in whooping cough. The expectoration varies much in different cases as regards quantity and character. The acts of expectoration are difficult, and the sputa are frequently accompanied by an abundant, frothy, serous liquid resembling soapsuds. Not infrequently sputa streaked with blood are expectorated.

The affection is chronic and unaccompanied by fever. The pulse is feeble and the body cool. Owing to an accumulation of blood within the right cavities of the heart, the surface of the body presents venous congestion, and this, together with deficient oxygenation of the blood, may give rise to a cyanotic hue. In extreme cases lividity is marked. The paroxysms of coughing are accompanied with great congestion of the face and turgescence of the cervical veins. Symptomatic phenomena, aside from those referable to respiration and circulation, are accidental. The appetite and digestion may not be notably impaired; the body for a considerable period may be well nourished, but after a time slow, progressive emaciation takes place. The countenance has an expression of distress which, conjoined with tumidity and a dingy or livid hue, renders the physiognomy somewhat characteristic. At an advanced period anasarca may occur as a consequence of dilatation of the right side of the heart. Albumen in small quantity may be found in the urine, this being an effect of congestion of the kidney; and congestion of the liver and spleen may render these organs appreciably larger than in health.

These symptoms accompany the affection when it exists to an extent to considerably compromise respiration. Existing in a moderate or slight degree, there is no habitual want of breath or labor of breathing. But the respiration is disturbed by active exercise; the patient is short-winded on attempting to run or walk fast; and this difficulty is apparent also in speaking, singing, and especially in paroxysms of laughter. Cough and expectoration are usually present, having the same characters as when the affection exists to a greater extent, but less marked. Congestion, tumidity, and lividity are present—not habitually, but during paroxysms of coughing or any violent physical exertion. The functions of the body generally may present little or no disturbance. Different cases exemplify all gradations between the two extremes as regards the symptoms belonging to the affection.

Many persons affected with emphysema are subject to paroxysms of labored respiration and dyspnœa which are due to another affection—namely, asthma. Acute ordinary bronchitis occurring in a person affected with emphysema occasions symptoms of embarrassed respiration, which do not belong to the clinical history of bronchitis occurring in a healthy person. The reason of this is to be found in the anatomical changes which characterize emphysema.

The development of emphysema is generally slow. In the majority of cases the previous history shows the origin of the affection, as indicated by deficiency of breath on exercise, to be dated back many years, frequently extending to childhood, and not infrequently beyond the recollection of the patient. Exceptionally, it may be developed with rapidity. Notable temporary emphysema occurs in paroxysms of asthma and in cases of capillary bronchitis. The affection has no fixed duration, usually continuing and progressively increasing during the patient's lifetime.

PATHOLOGICAL CHARACTER.—Exclusive of the senile or atrophic variety, emphysema involves permanent dilatation of the air-cells, with, frequently, more or less destruction of the cell-walls and coalescence of the cells. In so far as destruction of air-cells takes place, it is obvious that the area of the respiratory surface is diminished, and the function of respiration in this way compromised. The dilatation of the cells involves a loss of the elasticity of the lungs, so that the accumulation of air within the cells offers an obstacle to the contraction of the chest in the act of expiration. Consequently, the air in the cells is not sufficiently renewed for the purpose of respiration, and the air stagnates in the cells. In these ways the affection compromises the respiratory function. The distension of the cells involves pressure upon the terminal branches of the pulmonary artery, and in this way the pulmonary circulation is obstructed. This obstruction is further increased by diminished respiratory function. The obstruction to the pulmonary circulation occasions an over-accumulation of blood in the right cavities of the heart; hence the venous congestion of the face and neck, and, to a greater or less extent, of the surface generally, with, in some cases, cyanosis. Hence, too, sooner or later supervenes dilatation of the right side of the heart.

The pathological character of emphysema furnishes an explanation of the symptoms which make up the clinical history of the affection. When the respiratory function is so far compromised that the functional capacity of the lungs is insufficient, while the respiratory acts are performed without effort, want of breath is felt and the respiration becomes labored. But if the emphysematous condition be slight or moderate, no difficulty is experienced habitually, because there is still functional capacity enough for ordinary respiration. The quantity of lung in health exceeds the amount required, there being a reserve provided for any unusual demand on the respiratory function, as in muscular exercise. If, therefore, the extent to which the respiratory function be compromised do not exceed the amount of lung held in reserve, no difficulty is experienced save when an extra demand is made on the function. Under these circumstances exercise, which involves an extra demand, cannot be taken without the want of breath being felt. The patient, who is habitually using the lungs to their fullest functional capacity, is disturbed by any interference with respiration. Ordinary bronchitis, which in a healthy person requires only a little of the reserve lung to be brought into play (a fact of which the patient is not conscious), occasions, if there be no lung in reserve, difficulty arising from the slight diminution of the calibre of the larger bronchial tubes which is incident to this disease; hence, patients with emphysema existing to much extent suffer from dyspnœa and labored breathing in ordinary bronchitis. The cough in connection with emphysema arises from the coexisting bronchitis, and the difficulty of expectoration is in consequence of the impaired elasticity of the lungs, the expiratory efforts in coughing not being brought to bear readily on the contents of the bronchial tubes.

The venous congestion of the face and of the surface generally is due to obstruction caused by over-accumulation of blood in the right cavities of the heart. The lividity is in a great measure due to the same condition, but in part perhaps to deficient oxygenation of the blood. The smallness and weakness of the pulse also proceed from an obstruction to the free passage of the blood through the pulmonary circuit.

CAUSATION.—The mode in which dilatation of the air-cells is produced has given rise to much discussion. Laennec, who was the first to describe distinctly this lesion, attributed it to obstruction of the bronchial tubes from the products of bronchitis. Regarding the inspiratory act as more powerful than the expiratory, he supposed air to be forced through imperfectly obstructed tubes in inspiration, and to remain imprisoned in the cells in consequence of the want of sufficient expulsive force in expiration. The dilatation, according to this theory, is produced by the rarefaction of the imprisoned air by heat, and by the reaction of the force of expiration in coughing, etc. upon the cells already filled with air. This theory is certainly not tenable in view of the fact that of the two acts of respiration the expiratory is a third more forcible, and still more when voluntary and spasmodic efforts are brought to bear upon it. The more powerful the efforts of expiration in coughing, etc., the less ought to be the liability of accumulation of air in the cells communicating with obstructed tubes. An explanation of the mode of production has been advocated by Gairdner of Glasgow which differs essentially from the theory of Laennec. Gairdner attributes the affection to obstruction of bronchial tubes incident to bronchitis; but he supposes that the obstruction from collections of plugs of mucus leads to collapse, instead of dilatation, of the cells which communicate with the obstructed tubes, in the manner already adverted to in the chapter on Broncho-pneumonitis (p. 174). The current of air in the act of expiration may remove the mucus from its situation in tubes of small size into larger tubes, where the obstruction is less; but the effect of the inspired current will be to carry it back from the larger to the smaller tubes, the mucous plugs thus acting like a ball-valve upon the orifice of a syringe, obstructing the entrance of air to the cells, but permitting the escape of air from the cells. Moreover, owing to the greater force of the expiration, the expired air may be driven past obstructions which arrest the current of air in inspiration. Collapse of more or less of the pulmonary lobules being thus produced, a larger amount of air enters the cells communicating with unobstructed tubes, and these cells become consequently dilated. The dilatation of cells, in other words, is supplementary to the diminished volume of the lungs due to collapsed lobules, and is produced under these circumstances in the act of inspiration. In support of this explanation, Gairdner states that observation shows the tubes connected with emphysematous lobules to be unobstructed, and that close examination of lungs affected with emphysema will show the traces of collapsed lobules. Moreover, experiments of introducing into the air-tubes in inferior animals foreign bodies, such as shot and wads of paper, have been found to lead to collapse of lobules connected with the tubes in which these bodies become lodged, and dilatation of lobules connected with other tubes. This explanation will account for the production of emphysematous lobules so frequently found in connection with tuberculous deposits, the latter inducing solidification and diminished volume of the portion of the lungs in which they are situated. The term *vicarious emphysema* expresses the fact that it is supplementary to diminished volume of lung.

There is reason to believe that by prolonged, forcible efforts of expiration, as in long-continued exertions at lifting or pulling, playing on wind instruments, glass-blowing, or in voluntarily blowing through a partially obstructed tube in the manner which has been to some extent practised as a means of preventing and curing consumption, the elasticity of the lungs may be diminished and permanent dilatation produced. It is well known that the chest may be in this way considerably enlarged, and deficiency of breath on exercise is sometimes a result. Expiratory efforts thus may sometimes co-operate with collapse of pulmonary lobules in the causation of emphysema. The paroxysms of whooping cough and the labored efforts of breathing in attacks of asthma may be more or less involved in the causation.

The mechanism of the causation of emphysema by expiration is as follows: During violent acts of coughing the glottis is contracted and offers an obstacle to the free escape of air, and the muscles involved in the acts of coughing exert their effect especially upon the inferior portion of the chest. Under these circumstances the air, not escaping through the glottis, distends the upper lobes of the lungs, and this distension, often repeated, leads to dilatation of the air-cells in these lobes. This explanation accounts for the fact of the upper lobes being especially the seat of emphysema, and that the anterior portions of the chest in some cases project beyond the limits of a forced inspiration in health. It is obvious that this explanation applies also to the production of emphysema by forcible acts of expiration produced otherwise than in coughing. Accepting this explanation, the fact that physical signs show emphysema, as a rule, to be more marked in the left than in the right upper lobe may be thus accounted for. In coughing and all forcible expiratory efforts the lower portion of the chest is more contracted on the left than on the right side, owing to the presence of the liver on the latter side.

The force with which the distension of the upper lobe takes place in violent coughing is appreciable by palpation over the clavicles and between the ribs in thin persons. It was strikingly exemplified in the case of Groux, in whom there was a congenital absence of a portion of the sternum. Forcible bulging within this wall was produced by coughing and by voluntary strong expiratory efforts.

The inspiration theory, as set forth by Gairdner, suffices to explain vicarious emphysema, but the expiration theory seems necessary for the explanation of substantive or idiopathic emphysema. Something, however, in addition to violent expiratory efforts is requisite, in view of the fact that a comparatively trifling expiratory pressure will produce the affection in one person, while in many persons severe paroxysms of coughing, etc., continued for a long time, do not occasion it. This view is supported by another fact—namely, that heredity is involved in the causation. Giving proper weight to these facts, it may be assumed that in many if not most cases a causative condition is some error of nutrition which impairs the elasticity of the alveolar walls.

The diminution of the elasticity of the ribs and costal cartilages incident to advancing age favors the production of emphysema. This change involves diminished extent and force of expiration, with undiminished, if not increased, power of inspiration. The result is, when the chest is expanded in inspiration, the lungs already contain a surplus of air. The lessened elasticity of the lungs in old persons has the same effect, keeping the organs permanently expanded.

The fact that emphysema belongs among the hereditary affections was first established by James Jackson, Jr., of Boston, who found that in 18 out of 28 cases one or both parents had been similarly affected. The affection may be developed at any period of life. In a large proportion of cases it begins in infancy or childhood. Its slowly progressive increase explains the fact that patients frequently attain middle life before the affection has advanced sufficiently to occasion great inconvenience.

DIAGNOSIS.—The symptoms described under the head of the Clinical History render the affection probable when it compromises considerably the respiratory function. The probability is increased by the coexistence of asthma in a large proportion of cases. The physical signs, however, render the diagnosis positive, and when the affection is moderate or slight the signs are indispensable to the diagnosis. If asthma be not associated, patients are often supposed to have phthisis by those who rely on the symptoms in making the diagnosis.

The enlarged volume of the upper lobes of the lungs in certain cases of emphysema gives rise to distinctive signs obtained by inspection. In the cases in which the characters of the "emphysematous chest" are strongly marked the diagnosis may be at once made with the eye. The upper and middle thirds of the chest on its anterior aspect are expanded, and present a globular or barrel-shaped form. This projection extends from the summit to the sixth rib. The obliquity of the ribs is diminished, and the angle formed by the junction of the ribs with the costal cartilages is more obtuse than in health. The inferior portion of the chest is relatively and actually contracted. The dorsal spine becomes curved anteriorly. In extreme cases these changes amount to a deformity which is highly characteristic. The superior costal respiratory movements are lessened, and in labored breathing the ribs and sternum are raised together, as if they formed a solid bony case. The lower anterior portion of the chest is contracted, and the soft parts above the clavicles and sternal notch are depressed in inspiration. The chest is drawn permanently upward by the sterno-cleido-mastoid muscles and the scaleni, so that the neck appears to be shortened. The upper part of the chest during the act of expiration is in the same condition as at the end of a forced inspiration. The intercostal depressions are usually strongly marked. The heart's impulse is frequently seen and felt in the epigastrium.

These visible changes are present in cases in which the emphysema exists to an extent to compromise considerably the function of respiration, and in which the volume of the lungs is notably increased. The signs are more marked on the side in which the emphysema is greatest, this side being, in my experience, generally the left. If the emphysema be comparatively slight or moderate, the size, form, and movements of the chest will not be sensibly altered. An abnormal relative fulness, however, may be observed on one side below, and perhaps above, the clavicle, with deficient superior costal movement. This disparity of the summit between the two sides as regards size and mobility is to be distinguished from a similar disparity due to contraction of one side from phthisis.

Percussion elicits an abnormally intense resonance, the character of the resonance being altered. The vesicular and the tympanitic quality are combined; that is, the resonance is vesiculo-tympanitic. This vesiculo-tympanitic resonance is more marked on the side corresponding to the lung which is the more emphysematous; that is, generally the left side. The exaggerated resonance being greater on one side, care must be taken not to consider the resonance as diminished on the side in which it is less exaggerated. The pitch of the resonance is always higher on the side which yields the greater resonance, whereas if the disparity be due to diminished resonance or dulness on one side, the pitch is higher on that side. That the resonance is vesiculo-tympanitic over the upper lobe on the side on which it is least marked—that is, usually the right side—may be shown by comparing this resonance with that over the lower lobe on the same side.

The murmur of respiration is weakened in proportion to the degree of the emphysema. If the affection exist to such an extent that the respiration is habitually labored, the inspiratory sound may be shortened (deferred) and the expiratory sound prolonged. The expiratory sound, however prolonged, is lower in pitch than the inspiratory, as it is in health. The changes in intensity and rhythm are the only modifications proper to emphysema. Not infrequently over portions of the chest corresponding to parts of the lungs which are not highly emphysematous the labored efforts of breathing evolve an exaggerated vesicular murmur. The weakness and alteration of rhythm are likely to be more marked on the side corresponding to the greater degree of the emphysema. Sibilant and sonorous râles, due to coexisting bronchitis or asthma, are often present in cases of emphysema.

The normal relation between the two sides, as regards vocal resonance and fremitus, continues. In some cases, owing to the displacement of the heart, the resonance on percussion within the præcordial region is abnormally clear.

In arriving at the diagnosis by means of the foregoing signs, it is to be considered that other affections, such as phthisis, pleuritis, and pneumo-hydrothorax, are excluded by the absence of signs which should be found if these affections existed. The signs are also to be taken in connection with diagnostic points embraced in the clinical history—namely, the character of the cough and expectoration, the absence of marked emaciation notwithstanding the duration of the pulmonary symptoms, and in many cases the coexistence of asthma.

In the foregoing account reference has been had to the ordinary form of emphysema—namely, the form characterized by dilatation of the air-cells. The atrophic form, or senile emphysema, offers certain marked points of difference, especially as regards the physical signs on inspection. The size of the chest, instead of being increased, may be notably diminished. The obliquity of the lower ribs may be much greater than in health, leaving sometimes but little space between the base of the chest and the pelvis. The resonance on percussion may be diminished.

PROGNOSIS.—Emphysema, even in the cases in which it is most marked, rarely if ever involves in itself immediate danger. It is not a fatal affection *per se*, but it diminishes the power of the system to resist intercurrent or superadded diseases. It leads to dilatation of the heart, which further impairs the ability to overcome other diseases. But, aside from danger to life, it is an affection which, if it exist to an extent to compromise respiration, incapacitates for physical exertion and renders life uncomfortable. Existing in a less degree, it is a source of much inconvenience and annoyance from the deficiency of breath on exercise.

In so far as it involves destruction of the alveolar walls and the coalescence of air-cells, it is an irremediable lesion. And in the majority of cases the dilatation of the air-cells which takes place is permanent; the tendency, in fact, is oftener to increase rather than to decrease. I have known, however, marked improvement to take place, as shown by the physical signs. In a case in which the enlarged volume of the upper lobes was sufficient to give rise to the characteristic deformity in a marked degree, the improvement was so great that the chest resumed nearly its normal form. In so far, therefore, as the lesion consists in dilatation of the cells, it is not necessarily irremediable.

TREATMENT.—The treatment of emphysema has reference, first and chiefly, to the bronchitis with which it is generally associated. The object is to relieve, and if possible remove, the bronchial inflammation. In proportion as the bronchitis is relieved the condition of the patient is rendered more comfortable, notwithstanding the emphysema continues undiminished; the emphysema is less likely to increase, and it may undergo diminution.

With reference to this object the measures to be pursued are those indicated in chronic bronchitis not connected with emphysema. The treatment, in fact, so far as this object is concerned, resolves itself into the treatment of chronic bronchitis, which has been considered in the preceding chapter. Trial should always be made of the iodide of potassium or of ammonium or of sodium. The effect is in some cases truly marvellous, but in other cases these remedies produce little or no effect. It may not be amiss to remind the inexperienced practitioner that some persons cannot continue the use of the iodide of potassium in consequence of great irritation of the fauces; and I have met with an instance in which it always produced violent vomiting and general disturbance. The eruptions on the face to which it frequently gives rise are an annoyance which may be submitted to if it exert a curative effect as regards bronchitis. When the remedy is well borne it may be continued, in moderate doses, for a long period without any unpleasant consequences. If moderate doses do not prove efficacious, they may be increased up to the limit of comfortable tolerance. The syrup of hydriodic acid is sometimes better tolerated than the iodides. The chlorate of potassa, the bromide of potassium, and the muriate of ammonia should also be tried if the iodide of potassium prove ineffectual. If these be unsuccessful, the various other remedies which experience has shown to be useful in cases of chronic bronchitis should be tried.

Tonic remedies and the regulation of diet and regimen with a view to invigorate the system form an important part of the treatment. If these measures fail, change of climate, if practicable, should be advised.

As there is reason to believe that violent paroxysms of cough contribute to perpetuate and increase the dilatation of the cells, palliative remedies for this symptom are called for, provided the bronchitis on which it depends cannot be removed. The effects of labored breathing in paroxysms of asthma probably have the same effect; and if this affection coexist, it is desirable, with reference to the emphysema, to relieve the paroxysms as speedily as practicable. It is important for the patient to avoid, as far as possible, attacks of acute bronchitis, and when they occur they should be cured as quickly as possible. Flatulence of the stomach or bowels and constipation contribute to dyspnœa by mechanically interfering with the play of the diaphragm. They are to be avoided by a regulated diet and appropriate remedies.

Exercise, as far as it can be taken without inconvenience, is not to be interdicted, but exertions which induce labored breathing are to be abstained from. Prolonged efforts of expiration, as in public speaking, singing, and straining at stool, are to be avoided.

The treatment by inhaling compressed air and breathing into rarefied air is recommended by German authors as often beneficial and sometimes effecting a cure. It has thus far found little favor in this country. Its employment requires caution lest it do harm. There is abundant testimony by competent observers of its value in cases of emphysema.[1]

The affection distinguished as atrophic or senile emphysema admits of no permanent relief. Ethereal preparations and remedies to palliate dyspnœa, together with measures to support the system, comprise the treatment.

Asthma.

The term asthma, used in a properly restricted sense, is applied to a disease which has well-defined symptomatic features, and which, in the opinion of most authors at the present time, has an essential pathological condition—obstruction of the smaller bronchi, caused by spasm of their muscular fibres. The term has heretofore been applied in a loose way to dyspnœa dependent on different pathological conditions. For example, it has been customary to call the dyspnœa dependent on diseases of the heart, cardiac asthma. The true pathology of asthma—that is, of the individual or substantive disease to which the term should be restricted—seems to me clearly that just stated, namely, bronchial spasm. All, however, do not so consider it, and other opinions will be noticed in connection with the pathological character of the disease. Asthma has no anatomical characters. It is true that in the great majority of cases pulmonary lesions are found after death, particularly those which belong to chronic bronchitis and emphysema; but this fact shows only the frequent coexistence of these affections with asthma.

Clinical History.—Asthma is essentially a paroxysmal disease. The clinical history will therefore consist of an account of the characters which distinguish asthmatic paroxysms, together with the laws of their recurrence and the condition of health in the intervals.

A paroxysm of asthma may begin without warning or it may be preceded by certain premonitions. The premonitions vary in different cases. Patients are often able to predict an attack for some hours before its occurrence, sometimes by a sense of drowsiness or heaviness; sometimes, on the other hand, by an unusual excitation of the mind; and, again, by sensations the significance of which they have learned by experience, but which are not easily described. The paroxysm may be slowly developed—that is, an hour or two, and sometimes a longer period, may be required for its full development—or the attack may be sudden and the intensity quickly reached. It takes place, in the great majority of cases, during the sleeping hours, and generally in the latter part of the night or very early in the morning.

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[1] Vide article by Hertz in *Ziemssen's Cyclopædia*, Am. ed., vol. v.

The paroxysm is characterized by laborious efforts of breathing, prompted by a painful sense of the want of air, or dyspnœa. The patient is unable to lie down, but sits with the elbows on the knees or resting on some solid support, the mouth open and gasping with each inspiration. The respirations are not increased, but may be less in number than in health. The inspiratory act is performed with a spasmodic effort, and the expiratory act is prolonged and accompanied with a wheezing sound. Speech is difficult from the want of breath, and words are uttered with interrupted efforts. Movements of the body are dreaded, and changes of position are made slowly. Cold air, from its density, affords relief, and the patient desires the windows to be opened without regard to chilliness of the body. The face is pallid, and sometimes presents with the pallor a cyanotic hue, giving to the face a death-like appearance. Frequently perspiration is profuse. Limpid urine in great abundance is passed frequently, especially at the beginning of the paroxysm. The pulse is small and feeble, on account of an accumulation of blood in the right cavities of the heart due to obstruction to the pulmonary circulation. The surface of the body is cool or cold. The temperature, as shown by the thermometer, is either normal or subnormal. The nostrils are widely dilated. The countenance denotes extreme anxiety and distress. A frequent symptom is an itching sensation under the chin, which the patient endeavors ineffectually to relieve by rubbing, and the same sensation is frequently felt on the sternum and between the shoulders.

Paroxysms differ in severity. A patient suffering from a severe paroxysm presents a spectacle more distressing than can be well imagined, and to one not familar with the disease he appears to be on the point of death. The suffering is considerable, even when the paroxysms are so mild as not to present to the spectator a picture of great distress. Their duration differs. They may continue for a few minutes only or for several days. Usually, after continuing for a few hours, either partial or complete relief occurs. If the relief be complete or nearly so, tranquil sleep follows, which is peculiarly sweet and refreshing. With the diminution of suffering there are usually cough and more or less expectoration.

Cough and expectoration may be absent during an asthmatic paroxysm. Usually, however, there is an expectoration of a small amount of viscid, transparent, frothy mucus. In this mucus are frequently found grayish particles, which, upon close inspection, are found to contain peculiar mucous bands of varying length and diameter twisted in a spiral manner. These spiral bands often contain a central glistening thread. They are sometimes called "Curschmann's spirals," from the author who has described them most fully.[1] These spiral cylinders or bands are doubtless moulds of the small bronchial tubes, and are probably composed of dense mucus. They are considered by Curschmann as evidence of a peculiar form of bronchitis which he calls *bronchiolitis exudativa*, and which he believes to stand in intimate causative relation to asthma. The sputum also often contains pointed octahedral crystals of some organic substance. These are Leyden's crystals, and are believed by him to be, by their irritation, the cause in many cases of the asthmatic paroxysms. Both Curschmann's spirals and Leyden's crystals have been found in the sputum of persons not affected with asthma, and asthma may occur without the presence of either of these structures in the sputum. The expectoration is sometimes streaked with blood, and occasionally hæmoptysis takes place.

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[1] *Deutsches Archiv f. klin. Med.*, Bd. 32, p. 1.

The paroxysms recur after intervals varying greatly in duration in different cases. They recur in some cases with great regularity after a stated period, and in other cases the recurrences are extremely irregular. Some patients suffer from a diurnal recurrence, and it is remarked by Salter that in these cases the affection is almost always associated with bronchitis or disease of the heart. Recurrence regularly after the interval of a week or a fortnight is not uncommon. Women sometimes have attacks only at the menstrual periods. Some persons experience an attack annually, and it may occur either in the winter or summer season. Examples of the latter are cases of so-called hay asthma, which will be noticed presently under another head. When the paroxysms recur irregularly they are sometimes attributable to a special exciting cause, but in other cases the reason of their recurrence is not apparent.

In the intervals the condition varies, the variations depending on the existence or otherwise of associated affections. Asthma is not infrequently associated with persisting chronic bronchitis, and under these circumstances more or less cough and expectoration are habitual. Emphysema is also a frequent concomitant, and deficiency of breath on exercise, or constant dyspnœa in proportion to the amount of emphysema, characterizes these cases. The habitual dyspnœa in these cases is due to the emphysema, and is not properly asthmatic. Dilatation of the heart is another affection not infrequently coexisting which may give rise to more or less dyspnœa.

PATHOLOGICAL CHARACTER.—The pathological character of asthma has been already stated in defining the term as applied to an individual disease. It is a neuropathic affection, tonic spasm of the bronchial muscular fibres being induced by a morbid excitation through the nervous system. The exciting causes of the paroxysms doubtless exert their effect through the excito-motor or reflex nerves. It is true that bronchitis generally exists at the time of the occurrence of the paroxysms, but bronchitis exists often enough without asthma, and cases occur in which the spasm takes place independently of bronchitis. In the latter cases the affection has been distinguished as nervous asthma; but, with the pathological view just presented, asthma is always nervous, and these cases differ from the majority only in the absence of coexisting bronchial inflammation. It is probable that inflammation renders the muscular fibres more prone to spasmodic action, and in this way it is involved in the production of the affection, not constituting, however, an essential part of it.

This view of the pathological character of the disease is believed to be substantially correct, but it is proper to state that opposing theories have been advanced by distinguished authors. Wintrich, Bamberger, and others contend that bronchial spasm does not exist in asthmatic paroxysms, but that the disturbance of respiration is to be attributed mainly to tonic spasm of the diaphragm. This theory is based on the evidence of the diaphragm remaining depressed and immovable during an asthmatic attack, and on the fact that there is an abnormal expansion of the lungs, the latter being, as is claimed, inconsistent with bronchial obstruction. The increased volume of the lung will perhaps account for the fixedness of the diaphragm, assuming this to be a clinical fact. That a spasmodic obstruction of the bronchi should be overcome by expiration rather than by inspiration may be, *a priori*, reasonable, but this idea should give way before clinical facts. The dry bronchial râles which are undoubtedly present, forming a marked feature of the disease, demonstrate bronchial obstruction; and the complete disappearance of these râles, together with that of the symptoms of the obstruction in some cases, as an effect of certain remedial agents, such as the inhalation of choloroform, is proof that the obstruction proceeds from spasm.[1]

A later theory, by Weber (1872), attributes the obstruction in great part to a swelling of the mucous membrane in the smaller bronchi from dilatation of the blood-vessels caused by a vaso-motor nervous influence. This theory was advanced many years ago by Beau in France. It does not do away with spasm, but attributes to the swollen condition of the membrane a certain measure of obstruction. This theory is not devoid of substantial support from clinical facts. It is consistent with the frequent occurrence of more or less expectoration coincidently with relief of the asthmatic condition, and it serves to explain the not infrequent existence of obstruction notwithstanding the employment of treatment for the relief of spasm. That it is a pathological element of the disease is highly probable, but that it is an effect of the bronchial spasm, as held by Biermer and Trousseau, is perhaps the most rational explanation of its occurrence.

According to Curschmann, many cases of asthma are due to a peculiar chronic affection of the small bronchi, which he designates as *bronchiolitis exsudativa*, and which is characterized by the appearance in the sputum of the spiral structures already described. This affection is often hereditary. The immediate cause of the asthmatic paroxysm Curschmann believes to be bronchial spasm induced by the bronchiolitis.

CAUSATION.—The occurrence of asthma involves a peculiar susceptibility in the bronchial muscular fibres to take on spasmodic action. This susceptibility does not exist in all persons. It constitutes a predisposition to the affection which characterizes certain constitutions. The majority of mankind, no matter to what influences they may be exposed, never have this affection. The predisposition may be transmitted by inheritance. Of those who become asthmatics so large a proportion have had parents or progenitors who manifested the same peculiarity of constitution that there must be admitted to be, in some cases, an innate and inherited predisposition. The fact of its being congenital is also shown by the occurrence of the affection not infrequently in early childhood and infancy, independent of any appreciable external causes. Whether the peculiar susceptibility be always congenital, or whether it be acquired in a certain proportion of cases, it is impossible to say. It is not proof against an innate idiosyncrasy that the affection frequently does not occur until after youth, and is sometimes deferred until old age, and that it follows some other pulmonary affection, such as whooping cough, bronchitis, or measles.

The etiological connection of asthma with nasal polypi and other morbid conditions seated in the Schneiderian membrane, especially hyperplasia of the erectile tissue covering the inferior turbinated bones, and also with affections of the throat, has recently given rise to discussion. Making allowance for the error of considering the embarrassment of respiration from obstruction of the nasal passages as constituting asthmatic dyspnœa, clinical facts have been reported which show that in some cases true asthma may be produced by a reflex influence originating in the membrane of the nose and throat. This is true of local affections elsewhere, always assuming the existence of an asthmatic predisposition as an essential element in the etiology. The term "nasal asthma" used by some late writers denotes only that in a person whose bronchial muscular fibres are liable to spasm, as an individual idiosyncrasy, certain local conditions within the nostrils may excite an asthmatic paroxysm.[1]

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[1] For a fuller exposition of the doctrine of Wintrich the reader is referred to the article by Riegel in *Ziemssen's Cyclopædia*, Am. ed., vol. iv. For its refutation *in extenso*, vide paper by Biermer, "Ueber Bronchial-asthma," in *Volkmann's Sammlung klinischer Vorträge*, 1870; see also the classical work by Salter, *On Asthma: its Pathology and Treatment*, Philadelphia, 1864.

For the development of the affection, certainly in most cases, in addition to the predisposition, exciting causes are requisite. The exciting causes are various, and the facts with regard to these show in some persons a remarkable susceptibility to particular agencies which upon most persons produce no morbid effect. A striking illustration of an idiosyncrasy in this regard is the variety of the affection commonly called *hay asthma*. The emanations from newly-mown hay produce in some persons coryza, bronchitis, and asthma, and the latter affection may never occur save when produced by this particular cause. The term hay asthma, however, is applied to cases occurring exclusively during the summer season, although the exciting cause may not proceed from hay. We meet with persons who suffer for a certain period during the summer months with asthma, associated with coryza and bronchitis, and during the remainder of the year they are entirely free from the affection. Sometimes the annual visitations in successive years occur precisely on the same date, and the duration is always about the same. For example, a medical friend of the author is attacked early in September of each year with coryza and bronchitis, and subsequently paroxysms of asthma occur during the night. He continues to suffer from the former affections and the nightly recurrence of the asthma for six weeks. The affections then disappear, and except during the period just stated his health is excellent. Relief and exemption are obtained by removing at the period when the affections occur to a situation devoid of, and distant from, vegetation. On going to sea the affections are sure to disappear after sailing a certain distance from land.

The affection known as "hay asthma" or "hay fever," first described by Dr. John Bostock in 1819 under the name *catarrhus æstivus*, has been studied, as it occurs in the northern part of this country, by Dr. Morrill Wyman,[2] and more recently by Dr. George M. Beard. The latter gives the results of an analysis of 200 cases.[3] According to Dr. Wyman, there are two forms of annually-recurring bronchial inflammation (catarrh) in persons with a peculiar idiosyncrasy. The first is often called the "rose cold" or "June cold," beginning in the latter part of May or early in June and continuing until early in July. This corresponds with the affection known in England as "hay asthma" or "hay fever." The other form is called by Dr. Wyman *catarrhus autumnalis*, or autumnal catarrh. In this form the affection begins generally in the third or fourth week of August, and ends in the latter part of September or in October. It rarely if ever continues after the occurrence of black frost. Asthma occurs in a certain proportion of cases only. The local symptoms denote irritation and subacute inflammation of the nasal and bronchial mucous membranes, together with some febrile movement and more or less constitutional disturbance. The affection appears not to exist in the Southern States or in the colder regions of Canada. Dr. Wyman has collected a considerable number of cases showing that relief may be obtained by going to certain parts of the White Mountain region, Mount Mansfield in Vermont (one of the Green Mountains), and the Adirondack Mountains in the State of New York.

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[1] For a full discussion of the several theories of the causation of asthma by Curschmann, Riegel, Donders, A. Fränkel, B. Fränkel, Hecker, Schmidt, Lazarus, Liebig, and Ungar, see *Verhandlungen des Congresses für innere Medicin*, Vierter Congress, 1885. In this country Dr. John N. Mackenzie, Dr. William H. Daly, Dr. John O. Roe, and others have contributed papers on this subject. (Vide *System of Medicine by American Authors*, article by Dr. W. H. Geddings, vol. iii. 1885.)

[2] *Autumnal Catarrh* (*Hay Fever*), New York, 1872.

[3] *Hay Fever, or Summer Catarrh*, New York, 1876.

Dr. Beard's cases show that the affection occurs in July as well as in May, June, and August, but less often than in the latter months; also, that the Southern States and Canada are not exempt from the disease, but that its occurrence is rare. It is a rare disease west of the Mississippi, either north or south. It is undoubtedly caused by the inhalation of some agent connected with vegetation, and probably this agent is the pollen from grass, flowers, and trees. The same cause evidently is not alike involved in all cases. The idiosyncrasy in some persons relates to emanations only in May or June, and in others only to those in August; and of those who have the predisposition some are affected at a particular place at a particular time, and others at the same place and time are not affected. The vegetable emanations, therefore, must be derived from different sources. The discovery of the sources of the causation in a certain number of cases will not apply to other cases. Assuming the morbific agent to be the pollen from plants, the idiosyncrasy in different persons has relation to different plants. The fact that removal during the summer and autumnal months to certain situations secures exemption from the disease to some and not to others is thus intelligible. Dr. Beard's observations give evidence of heredity as involved in the peculiar idiosyncrasy.

A curious idiosyncrasy is exemplified in the author's personal experience. This consists in the production of coryza, bronchitis, and asthma by an emanation from feather beds or pillows. Repeated attacks on sleeping away from home, especially in inns and steamboats, had occurred before the source was discovered. All feather pillows, and even feather beds, do not furnish the special emanation, and the circumstances on which the latter depends I have not ascertained. The poisonous principle is not connected with the odor which is sometimes perceived. I cannot determine beforehand whether a strange bed will excite an attack or not. If the feathers be of the (to me) poisonous kind, shortly after retiring labored respiration, cough, and wheezing begin, and progressively increase until I am obliged to get up, when the difficulty passes off in a few hours, leaving a little bronchitis, which continues for a day or two. I have never experienced the least degree of asthma except as a result of an emanation from feathers.

Dr. Richard M. Crane of Hagerstown, Md., has communicated to me a case under his observation in which attacks of asthma were distinctly traced to sleeping beneath a feather bed made of duck feathers. The cause was not ascertained until after four years of suffering from the affection. It had not existed previously, the patient being fifty years of age, and there was no return of the affection after an ordinary feather bed, made of geese feathers, was substituted for the bed which furnished the causative emanation.

Inhalation of the powder of ipecacuanha in some persons produces similar effects. Several examples of this idiosyncrasy have been reported. On relating my own experience at a meeting of a medical society on one occasion, a member of the society gave an account of a case in which the same effects were produced by an emanation from the body of the horse, so that the person was debarred from riding or driving on that account. I have recently heard of another case of this kind. Beard cites other instances. Salter gives several examples in his work on asthma. Asthmatic attacks are sometimes brought on by emanations from the bodies of other animals, as the cat, rabbit, etc.

In persons subject to asthma the paroxysms are liable to be induced by a variety of exciting causes, but individual cases differ as regards a particular susceptibility to certain of these causes. In some cases the inhalation of dust is particularly likely to bring on an attack. I have known a patient specially susceptible to the dust of grain and that arising when carpeted rooms are swept. The fumes of burning sulphur produce asthmatic breathing in those subject to the affection. Indigestion in some cases proves an exciting cause oftener than anything else. Some can refer paroxysms frequently to constipation. Salter relates a case in which the application of cold to the instep was sure to provoke an attack. Mental emotions sometimes act as an exciting cause. Occasionally the paroxysms are habitually connected with the menstrual function.

In the great majority of the cases of asthma, bronchitis coexists. In a certain proportion of cases the bronchitis is constant or habitual, and the frequency of the paroxysms depends in a measure on the bronchitis; for if the latter affection be relieved or removed the asthmatic attacks become less frequent. The susceptibility to the exciting causes of spasm is increased by the existence of bronchial inflammation. In other cases the attacks of asthma occur whenever bronchitis or a common cold occurs. Finally, climatic influences are involved in the causation, for it is a matter of frequent observation that the same persons are much more liable to recurrences in some situations than in others. It is also a matter of common observation with some asthmatics residing in a locality in which they are subject to frequent recurrences that atmospherical changes are likely to induce an attack.

Asthma occurs more frequently in males than in females, the proportion being as two to one. In the majority of cases it is developed prior to twenty years of age. It occurs not very infrequently in infancy.

DIAGNOSIS.—The diagnosis in well-marked cases is not difficult. The features of the asthmatic paroxysms are quite distinctive. First paroxysms occurring in young children may occasion some perplexity, but with proper knowledge and care the disease should not be confounded with other affections. Capillary bronchitis is distinguished by the frequency of the respirations, the acceleration of the pulse with more or less fever, and the diffusion over the chest of the subcrepitant râle. In asthma the respirations are not accelerated, but labored, the pulse is not notably frequent, and the dry bronchial râles are abundant. The dyspnœa incident to diseases of the heart often goes by the name of asthma, but the breathing in this form of dyspnœa is not labored, especially the expiratory acts, and accompanied by wheezing, as it is in asthma, and the difficulty in breathing is more or less habitual. Moreover, heart lesions adequate to the production of dyspnœa are determinable by physical signs. Asthma and cardiac lesions, however, may be associated. If asthma have existed for some time, it is usually associated with emphysema, and this association is diagnostic; yet it is to be borne in mind that emphysema sufficient to occasion dyspnœa, which may become greatly increased with a fresh occurrence of bronchitis, may exist without asthma; in other words, the dyspnœa due to emphysema and coexisting bronchitis is not properly called asthma. In addition to the abundance and diffusion of the dry bronchial rales, the physical signs during a paroxysm are enlargement of the chest in its upper and anterior regions, with diminished respiratory movements, vesiculo-tympanitic resonance on percussion, and suppression of the respiratory murmur. I have met with instances repeatedly of laryngeal spasm that have been considered as asthma. The differential points are, however, distinctive. In laryngeal spasm the inspiration is slow and labored. In asthma the labor and slowness are in the expiration. The former has been well termed inspiratory, and the latter expiratory, dyspnœa. Laryngeal spasm is accompanied by stridor referable to the larynx, and the latter moves up and down with inspiration and expiration. The dry bronchial râles and the other physical signs referable to the chest are wanting if there be only laryngeal spasm.

Prognosis.—A paroxysm of asthma, however severe or protracted, involves no immediate danger to life. Formidable as may appear an attack of great severity, and intense as is the suffering, it is doubtful if a fatal termination ever took place. In view, however, of the suffering, the tendency of the disease to become confirmed, with an increasing liability to recurrences, its existence must be regarded as a great calamity. If the paroxysms be produced by a particular exciting cause, which, being known, may be avoided, the disease imposes simply more or less inconvenience; but when they are liable to be produced by various causes which cannot be guarded against, it is truly a grievous affliction; and it is calamitous, of course, in proportion to the frequency with which the paroxysms recur and their severity.

Asthmatics are not infrequently long-lived, yet that it may shorten the duration of life cannot be doubted. The labored efforts of breathing in the paroxysms contribute to the development and increase of emphysema. During the paroxysms also, the circulation through the lungs being impeded, the right ventricle and auricle are unduly distended with blood returned by the systemic veins, and hence the disease occasions dilatation of the right side of the heart. The chances of long life are lessened by these lesions. Moreover, if the paroxysms be frequent and severe they can hardly fail to impair the vital powers and diminish the ability to resist other diseases. If asthma secure, to a certain extent, exemption from pulmonary phthisis and pneumonitis, this advantage is perhaps overbalanced by the fact that pneumonitis, when it does occur, is likely to prove fatal, and that the supervention of a severe attack of bronchitis may destroy life.

Treatment.—The management of asthma embraces—*first*, the treatment of the paroxysms; and *second*, the treatment in the intervals.

During a paroxysm the objects of treatment are to lessen the suffering and bring the paroxysm to an end as speedily as possible. The measures for these objects have reference to spasm as the essential pathological condition. The measures to relieve spasm are various, each of which proves efficacious in some cases and not in others. Frequently the past experience of the patient is the best guide as to the particular measure which will be most likely to afford relief. In cases of asthma unaccompanied by bronchitis a full opiate is sometimes quickly and completely successful; but in the larger proportion of cases it will not succeed in cutting short the paroxysm or in affording marked relief. Of other narcotics, stramonium is one which should be tried. The usual mode of administration is to smoke the dried leaves or the fibres of the root either in a pipe or prepared as a cigarette. This measure in some persons acts like a charm, and may be confidently relied upon as a prompt and effectual mode of obtaining relief; but in the great majority of cases it either produces no effect or merely mitigates the severity of the paroxysm. Asafœtida, dracontium or skunk-cabbage, and the Indian hemp are sometimes efficacious. The ethers, given internally, are to some extent useful as palliatives, and occasionally they produce complete relief. Belladonna is sometimes signally efficacious, given as recommended by Salter and Ringer—namely, 10 minims of the tincture repeated every two or three hours unless disturbance of vision be produced. The bromides in full doses sometimes afford marked relief. A paroxysm is sometimes made to abort by a full dose of quinia—from 20 to 30 grs. to an adult.

Marked relief is frequently obtained and the paroxysm is sometimes cut short by nauseant remedies—namely, antimony, ipecacuanha, lobelia inflata, and common tobacco. The two remedies last named are especially efficacious in a certain proportion of cases. The common tobacco will be more likely to be successful if the patient be not accustomed to its use. With a view to the relief of the spasm these remedies need not be carried to the extent of producing vomiting; if not effectual when nausea is induced, it will be useless, if not injurious, to push them farther. I have known a paroxysm to be arrested at once by bloodletting, but this is a measure too potent to be employed, except occasionally in persons who are robust and full-blooded.

The inhalation of the vapor of ether is often an effective measure of treatment. Not infrequently the paroxysms are completely controlled by it, the patient passing in a few moments from a condition of great sufffering into one of ease and comfort. These cases are among those which afford the most striking examples of the resources of practical medicine. The dry bronchial râles, which before the inhalation were loud and universally diffused over the chest, sometimes disappear as soon as complete relief is procured by the inhalation—a fact proving conclusively the existence and the cessation of spasm. Unhappily, this measure, like the others, is efficacious only in a certain proportion of cases. It should be tried always, provided there be no circumstances to contraindicate it. The hydrate of chloral succeeds in a certain proportion of cases, but in view of the possibility of alarming effects it should rarely be resorted to. The inhalation of chloroform is often successful, but in view of danger ether is always to be preferred. The inhalation of the nitrite of amyl affords temporary relief, and in some instances arrests a paroxysm. Four or five drops may be inhaled from a handkerchief. Iodic ether or iodide of ethyl is recommended by Thorowgood as highly useful in some cases. Ten drops may be inhaled three or four times daily. This remedy may be conveniently inhaled by dropping it on a piece of patent lint held in the palm of the hand.[1]

The vapor of arsenious acid is to be included among the remedies which are useful administered by inhalation. Arsenical cigarettes have been employed with success, each cigarette containing from a half a grain to a grain of the acid.[2]

The inhalation of compressed air is recommended by German authors. Trousseau found the inhalation of the vapor of ammonia useful in some cases. Duclos succeeded in sometimes arresting a paroxysm by the application of aqua ammoniæ to the fauces by means of a probang. Others who have made trial of this measure have found it in some cases to succeed, but in other cases to increase the dyspnœa.[3] Dr. Monell states that he has succeeded in obtaining relief in an attack of asthma by prolonging as far as practicable the expiratory act, then waiting as long as possible before inspiring, and retaining the air at the end of the inspiration as long as possible, this plan being continued for fifteen minutes. He states that he has repeatedly obtained immediate relief in this way, and that he knows of several cases in which the plan has proved successful.[4]

It is hardly necessary to say that if there be ground to suppose the attack to have been brought on by overloading the stomach or by constipation, an emetic or a cathartic is indicated; and of course the patient is to be removed from the action of any known exciting cause, such as the emanations from hay, feathers, etc. The apartment should be large, high, and airy. Warm and stimulating pediluvia afford some relief. Strong coffee, taken hot, is generally useful as a palliative. It may be rendered more efficient by adding two or three grains of the citrate of caffeine. Another palliative measure which sometimes proves to be curative is to be added. This consists in diffusing throughout the apartment the fumes of burning nitre-paper—that is, bibulous paper dipped in a saturated solution of the nitrate of potassa and dried—or by burning pastilles prepared for that purpose. Some patients find great relief from this measure, and occasionally the paroxysms are arrested by it.

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[1] Vide *Lettsomian Lectures on Bronchial Asthma*, 1885.

[2] *Ibid.*

[3] The tincture of quebracho is recommended by Penzoldt and others in asthma and other diseases attended by dyspnœa.

[4] *N. Y. Med. Record*, Aug. 15, 1866.

The treatment in the intervals has for its object prevention of the paroxysms. The removal of the predisposition would be the most effectual mode of accomplishing this object. This, however, is not to be expected; and the object is to be attained by measures having reference to circumstances which increase or act upon that susceptibility of the bronchial muscular fibres constituting the predisposition to the affection.

When, owing to a peculiar idiosyncrasy, paroxysms are excited by a particular cause, such as emanations from hay, feathers, etc., avoidance of exposure to the cause, if possible, is obviously indicated. It is doubtful if any remedy will produce insusceptibility to the action of the particular cause or secure exemption so long as the exposure to the cause continues. In the cases of the so-called hay asthma which have come under my observation a great variety of remedies have been tried, but all without much avail. The patient must either endure the continuance of the affection for several weeks, or seek a situation where he is not exposed to the exciting cause. Inasmuch as the cause of this variety of asthma enters the air-passages by the nostrils, and occasions coryza before it reaches the bronchial tubes, the attempt has been made to neutralize it by topical applications within the nostrils. Liquid injections or atomized fluids have been employed for this purpose. Helmholtz found in his own person that a solution of quinia injected into the nostrils was preventive of effects other than the coryza; and this result has been obtained in some other cases. This treatment has, however, much oftener failed than succeeded. Experiments with other remedies have not furnished striking results; but this plan of prophylactic treatment merits more attention than it appears as yet to have received.

Recent observations having shown that paroxysms may be excited by polypi and other affections of the nose, and by affections of the pharynx, appropriate local measures of treatment are to be directed to these situations, and in general terms any existing local affection which may be suspected of having a causative relation to the asthmatic paroxysms should, if practicable, be removed.

In a large proportion of the cases of asthma the liability to paroxysms is more or less increased by the existence of habitual or chronic bronchitis. The relief or cure of the latter affection in these cases is the immediate object of treatment. The iodide of potassium or of ammonium should always be tried. I have known patients who were rendered comparatively comfortable as regards the recurrences of asthma by the use of this remedy. In small or moderate doses it may be continued indefinitely without any apparent injury. I have known from 5 to 10 grs. to be taken most of the time during four or five years, the patient finding that thereby he was protected against a recurrence of the paroxysms. The remedy will be useful in proportion to its curative effect on the bronchitis; it is, however, useful in some cases in which the coexisting bronchitis is slight and when no apparent effect upon the latter is produced. The hydriodic acid has proved in some cases as effectual as the iodides. In the treatment of asthma, as of chronic bronchitis without asthma, the doses of the iodides may be gradually increased so as to amount to 50 or 60 grains per diem. The bromides are serviceable in certain cases, and they are well borne by some who cannot take the iodide of potassium without unpleasant effects. The chlorate of potassa is sometimes useful, and the other measures indicated in cases of chronic bronchitis may be employed. The arsenical medication—that is, arsenic in small doses continued for a considerable period—has been found in some cases highly useful. The same is to be said of sulphur.

If measures addressed to the bronchitis prove ineffectual, or if the paroxysms recur when bronchitis does not coexist, and they be not referable to particular causes which may be avoided, nothing is to be depended on but change of residence. And it is probable that most asthmatics may find some place in which they will be comparatively if not entirely free from the affection. Facts showing the immunity obtained by change of residence are remarkable as illustrative of an apparent capriciousness of this affection. Persons residing in the country often find relief by living in cities or large towns. A change from the most salubrious part of a city to a part where the atmosphere is smoky and insalubrious as regards the general health sometimes secures exemption from the affection. A change of apartments from one story to another or to a different exposure has been known to prove effectual. I have known a patient to suffer nightly from a severe attack directly on the seashore, whereas the nights were passed comfortably a mile inland. There are no fixed laws with respect to the best climate or situation for asthmatics; and each case has its own law, which is to be ascertained only by experience. In beginning trials of change of residence, a place should be selected in which the climatic influences are the opposite of those belonging to that in which the patient resides; that is, if the residence be in the country, the city may be tried, and *vice versâ;* if on the seashore, an inland situation, and *vice versâ;* and so with regard to temperature, moisture, elevation, etc. The principle to be acted on is, that there is a place in which each martyr to this complaint will suffer less, and perhaps be entirely free from it, and the plan should be to make repeated trials until the desired spot is found. I could cite from the cases which have come under my observation several in which this plan has proved successful. It is to be enjoined, whenever practicable, as soon as it is evident that other measures will not secure the patient against the suffering incident to the frequent recurrence of the affection.

At the present time, in this country, the climate of Colorado is regarded as the most favorable for asthmatics. That residents in that climate are rarely affected with asthma, and that persons who suffer from the disease generally find relief there, cannot be doubted in view of ample testimony; yet it has happened to me to be consulted in the case of a boy about twelve years of age who had suffered from the disease since early childhood, and who has recently come from Denver, where he had lived from birth. A brief stay, however, in Colorado or elsewhere secures exemption only during the time of sojourn; persistent relief requires a permanent change of residence. Hence it is generally an object with patients to find some place where, aside from exemption from this disease, it is for their interest and happiness to dwell.

Pertussis, or Whooping Cough.

This affection belongs among the diseases of children, but it occasionally occurs after childhood and at all ages. In the cases occurring after adolescence it is important that the physician be prepared to recognize it. The affection has no anatomical characters except those of ordinary bronchitis. Other morbid appearances found after death are due to complications which will be noticed in connection with the clinical history.

CLINICAL HISTORY.—The names of this affection derive their significance from certain characteristics pertaining to the cough. The first or the forming stage embraces a period prior to the appearance of these characteristics. The primary symptoms are those of simple coryza and bronchitis. Frequently during this period there is nothing which denotes the affection to be other than a common cold. But in the majority of cases the cough is more violent, the acts of coughing persist for a longer time, and they progressively increase in duration and violence. There is also more or less febrile movement, which is more marked and continues longer than in ordinary bronchitis. At length the cough becomes distinctly and in a marked degree paroxysmal, and the distinctive features of the affection relate especially to the paroxysms. The duration of this first stage varies from one to two weeks. After the affection has advanced to the second stage, the patient is generally aware for a few moments previous to a paroxysm that it is impending. A child engaged in play suddenly is quiet, and the countenance expresses apprehension and distress. The morbid sensations are those of constriction and an irritation in the larynx and trachea. These premonitions are sufficient to arouse the patient when asleep. The paroxysm is denoted by cough, which is characterized by a series of violent expiratory acts succeeding each other so quickly that the patient is unable to take an inspiration between them. The number of these acts varies, according to the severity of the paroxysms, from six to twenty. A long and labored inspiration then takes place, giving rise to a crowing sound evidently due to spasms of the glottis; this is the whoop which enters into the name of the affection. Another series of coughing expiratory acts succeeds, followed again by the sonorous inspiration or whoop; and these alternate acts of coughing and whooping are repeated until the paroxysm ends. The contraction of the lungs by the spasmodic acts of coughing interrupts not only inspiration, but the pulmonary circulation, so that an accumulation of blood takes place in the right cavities of the heart. These effects are shown by notable congestion and lividity of the face and turgescence of the cervical veins. Tears flow in abundance. The suffering from dyspnœa is in proportion to the violence and length of the paroxysm. The contents of the stomach are frequently expelled, and at the close of the paroxysm more or less mucous secretion is expectorated. In proportion to the violence and length of the paroxysm the patient is fatigued or exhausted. However severe the paroxysm, there is scarcely any immediate danger either from apnœa or syncope. I have never known of an instance of death in a paroxysm. A medical friend, however, has related to me a case in which the breath was lost, and was restored by resorting to the procedure known as Marshall Hall's ready method.

The paroxysms in different cases differ widely in severity, duration, the degree in which the characteristics are marked, and in frequency of recurrence. When extremely severe they occasion great distress, and serious incidental events are liable to occur. On the other hand, when quite mild they are comparatively trivial. Only one or two of the spasmodic expiratory acts may occur, or they may be repeated so that the paroxysm lasts for several minutes. The whoop is more or less loud and long in proportion to the amount of spasm of the glottis. Although generally present and marked—being, as the name implies, the most characteristic feature of the paroxysm—it is sometimes slight and sometimes wanting. In the latter case the character of the disease has to be determined by other diagnostic features. A few paroxysms only may occur in the twenty-four hours, or they may recur during night and day at short intervals. They may amount to even a hundred within the twenty-four hours. In almost all cases they occur in greater number in the night than during the daytime, and in some cases they occur only at night. Exceptionally they are more frequent during the daytime. The paroxysms which occur in sleep are, of course, not produced by any obvious exciting cause. This may be true of paroxysms occurring in the waking hours, but physical exertion and mental excitement, especially the latter, appear not infrequently to determine the occurrence of a paroxysm. Children are liable to experience a paroxysm whenever a fit of crying takes place. A patient may have a paroxysm on witnessing a paroxysm in another patient, this fact illustrating the power of involuntary imitation.

Various events are incidental to the paroxysms, especially when the latter are severe. One of the most frequent of the incidental events is hemorrhage. Epistaxis is the most common form of hemorrhage. Not infrequently blood flows from the nostrils in more or less abundance with every paroxysm, and, the loss of blood inducing notable anæmia, this favors the persistence of the hemorrhage. Hæmoptysis is an occasional form of hemorrhage. Blood sometimes escapes from the conjunctiva or accumulates beneath this membrane. Trousseau cites a case in which a nævus situated on the face was the seat of hemorrhage with each paroxysm. Blood has been known to be forced from the ears. The primary and chief cause of the hemorrhage is the venous congestion arising from accumulation of blood within the right cavities of the heart. The urine and feces, or both, are sometimes expelled involuntarily during the paroxysm. Intestinal hernia is sometimes produced. Rupture of air-vesicles is an accident which sometimes occurs, giving rise to interlobular emphysema; and the air, finding its way along the areolar tissue connected with the primary bronchi and trachea, may produce emphysema of the neck, and even extend over the entire body.[1] Dilatation of the air-cells, or vesicular emphysema, is another incidental event. It is questionable whether this be not due to the bronchitis associated with whooping cough rather than to the violent action of coughing; but the latter doubtless contributes to its production. When the paroxysms recur frequently, and are generally accompanied by vomiting, the system suffers from innutrition. Here is a source of anæmia in addition to the hemorrhages. Convulsions occur in some cases, especially in children, being attributable to the cerebral congestion occasioned by the venous obstruction at the right side of the heart.

In the intervals between the paroxysms, aside from complications or intercurrent affections, the general condition will depend on the frequency of the paroxysms, their severity, and the incidental events. The patient is enfeebled in proportion as the paroxysms are long, violent, and frequent, also in proportion to the amount of hemorrhage and the interference with nutrition by vomiting. The face is pallid in proportion to the anæmia. The veins of the neck furnish the venous hum, and the arteries a bellows murmur. Cephalalgia is a prominent symptom in some cases, arising from cerebral congestion. The febrile movement, which generally exists, in a greater or less degree, during the first or forming stage, as a rule disappears when the characteristic paroxysms are established; that is, in the second stage. If it continue into this stage or if it be reproduced, it is due to an unusual intensity of the bronchitis or to some inflammatory complication. The face is swollen or puffed, and this, with the pallor, renders the appearance quite characteristic. Certain complications are liable to occur which add much to the gravity of the affection. Bronchitis is a part of the affection.

If the affection be uncomplicated, physical exploration of the chest elicits good resonance on percussion, with perhaps the dry or moist bronchial râles. The bronchitis in some cases is unusually acute, giving rise to fever and abundant mucous secretions. Collapse of pulmonary lobules and bronchopneumonia are liable to occur in young children. The occurrence of vesicular emphysema has been already mentioned. The inflammation may extend into the minute bronchial tubes, giving rise to capillary bronchitis. This is denoted by fine bubbling râles diffused over the chest, in conjunction with great frequency of the respirations, notable acceleration of the pulse, and lividity, the resonance of the chest on percussion not being diminished. Pleuritis with effusion is another complication more likely to occur in adults than in children. These several pulmonary complications are rendered severe and dangerous by the recurrence of the paroxysms of whooping cough. Frequently the latter become less violent and frequent when any of the complications just named are developed, so that a premature improvement as regards the paroxysms of whooping cough may be an unfavorable omen. When whooping cough is accidentally associated with some acute disease, such as measles or scarlet fever, the paroxysms of cough, as a rule, become less frequent and violent; these intercurrent diseases interfering with the natural course of the affection. Finally, whooping cough appears to favor the development of phthisis in some cases in which the tuberculous diathesis exists.

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[1] Vide case reported by Dr. Wm. P. Northrup in *Am. Journ. of Med. Sciences*, July, 1883.

The duration of the affection is subject to considerable variation. Exceptionally the affection ends spontaneously in a few days. These cases are infrequent, but the fact that the affection is occasionally of short duration when let alone is to be borne in mind with reference to the supposed efficacy of remedies in abridging or arresting it. It is rare for the affection to end within a period of six weeks. In the majority of cases it continues for a longer period than this, and it sometimes persists for many months. As a rule, it gradually declines before it disappears, the paroxysms by degrees becoming less frequent and severe. Frequently, for a considerable period after the affection has ended, the characteristics are manifested, to a greater or less extent, whenever a bronchitis or common cold is contracted or cough is excited by any cause. Trousseau states that the affection continues for a long or short period in proportion to the duration of the symptoms prior to the characteristic paroxysms; that is, if the latter are delayed the affection will be protracted, but if they are quickly developed the affection is not likely to last long.

PATHOLOGICAL CHARACTER.—This remarkable affection consists of three pathological elements. Bronchitis is one of these. This is the initial element. It is stated that this element is sometimes wanting, but examples must be exceedingly rare. Another element is fever. This is more or less marked in different cases, but is seldom wanting during the first or forming stage. The fever, as a rule, is out of proportion to the bronchitis, and is therefore to be regarded as not symptomatic, but idiopathic or essential. The most prominent of the three elements relates to the nervous system. This is manifested by the spasmodic expiratory movements in the paroxysms of cough, and by spasm of the glottis giving rise to the whoop. Some have considered the affection as belonging among the neuroses, others have considered it to be an essential fever, and by others it has been considered as a peculiar variety of bronchitis. It is neither of these separately, but collectively they constitute the affection. Dependent, as will be seen presently, on a special cause, the primary, essential pathological condition is general or constitutional, of which the bronchitis and cough are the local expressions.

CAUSATION.—Whooping cough is an infectious disease, and it is communicated by a contagium present in the bodies of those affected with it. It probably originates in no other way. It is highly contagious, and few persons pass through childhood without contracting it. Young infants are liable to contract it. No period of life is exempt from susceptibility to the contagium. The infrequency of the affection after childhood is accounted for by the fact that the great majority of persons experience it before adolescence. Having been once experienced, the susceptibility thereafter ceases. This is the rule, but exceptionally the affection occurs more than once. The exceptions to the rule as applied to this affection are not more frequent than to the rule as applied to other affections in the same category; for example, the eruptive fevers. The average period of incubation is not well ascertained. It is variable, the limits being one and two weeks.

DIAGNOSIS.—When whooping cough has advanced beyond the first or forming stage and the characteristics pertaining to the paroxysms are well marked, it is recognized without difficulty. The whoop is a diagnostic criterion, but this is sometimes imperfect and even wanting. In these cases the diagnosis is to be based on the occurrence of paroxysms presenting the characters of well-marked whooping cough minus the whoop, on the duration of the affection, and on the fact of known exposure to the infection, together with knowledge of the fact that the patient has not already experienced the affection. The affection may be so mild and short that there is considerable doubt whether it have really occurred, but such cases are rare. It is desirable to make a probable diagnosis during the first or forming stage; that is, before the disease is fully declared by the characteristic paroxysms. It should be strongly suspected when cough and febrile movement are out of proportion to the bronchitis and unduly persistent. Of course the prevalence of the affection and known exposure are to be taken into account. Attention has been recently directed to the occurrence of ulcerations on either side of the frænum linguæ as a diagnostic circumstance. These are produced traumatically by the lower teeth, the tongue being partly protruded during paroxysms of cough. They occur only when, from the violence of the paroxysms, the diagnosis involves no difficulty. They are wanting, of course, in children before the period of dentition.

PROGNOSIS.—Whooping cough is rarely fatal *per se*, yet indirectly it leads to a considerable loss of life. Occurring in the course of other diseases—for example, measles or during the ailments incident to dentition—it may prove a serious affection. A fatal result is generally due to complications, the more frequent and important of which have been stated—namely, capillary bronchitis, broncho-pneumonitis, pleuritis, vesicular and interlobular emphysema, and phthisis. Young children, especially during dentition, are sometimes carried off by convulsions. As regards the affection itself, the gravity and danger are proportionate to the frequency and severity of the paroxysms, the duration of the affection, and the degree of anæmia and innutrition induced by hemorrhage and vomiting.

TREATMENT.—The treatment of whooping cough embraces curative and palliative measures. It must be admitted that there are no known means by which the affection may be arrested. Measures are curative if they abridge the duration of the affection or diminish notably its severity, and there are various remedies which possess more or less curative power.

Emetics were formerly considered as curative, given every other day for the space of a week or longer at the beginning of the affection. They were much extolled by Laennec, who preferred ipecacuanha as the emetic drug. Others have preferred the sulphate of zinc. Trousseau advocates, instead of the zinc or ipecacuanha, the sulphate of copper, given in solution in small doses, according to the age of the patient, the doses being repeated at short intervals until vomiting is produced. In view of the testimony in behalf of the treatment with emetics, its efficacy in some cases cannot be doubted, but owing to its severity it is nearly or quite obsolete in this country. This method of treatment, it is to be borne in mind, is not admissible except early in the career of the affection.

Of other curative remedies, some are addressed to the bronchitis and others to the neuropathic element. A combination of cochineal and the carbonate of potassa, commonly known as the cochineal mixture, has been much employed in this country. This mixture consists of cochineal, half a scruple; of carbonate of potassa, a scruple; and of white sugar, a drachm, in four ounces of water. A dessert-spoonful is to be given three times daily to a child a year old, and the dose increased in proportion as the age is greater. The disease is undoubtedly in some cases favorably modified by this preparation, as shown by diminution of the violence of the paroxysms and of the frequency of their recurrence: these effects of remedies can generally be appreciated when the affection has not already continued sufficiently long to attribute the improvement to a spontaneous decline. It is not easy to determine the fact of the duration being abridged, as the natural duration varies within wide limits. This remedy probably acts upon the bronchitis. Meigs considers the cochineal as inert, and states that he has found the same benefit from the carbonate of potassa alone, dissolved in syrup of gum and water.[1]

Alum, which was recommended highly by Golding Bird, is considered by Meigs as giving more decided and satisfactory results than any other remedy which he has employed. From one to six grains may be given every four hours, the dose being graduated to the age. Dissolved in some form of syrup and water, it is not an unpleasant remedy. This remedy probably exerts its curative effect by acting upon the bronchitis.

Belladonna has been recommended as a valuable curative remedy by many physicians in different countries. Trousseau lays down certain rules with regard to its administration which he deems essential in order to secure its curative efficacy. The dose at first should be quite small—one-tenth of a grain of the extract for a child a year old, and one-fifth of a grain for a patient four or more years of age. The quantity to be taken in the twenty-four hours is to be given in a single dose. The dose is to be gradually increased until its effect upon the severity or frequency of the paroxysms is apparent. It should then be continued steadily without further increase. The action of this remedy is doubtless upon the neuropathic element of the affection. Some have regarded conium, stramonium, and hyoscyamus as not inferior to belladonna in this affection. The leaves of the chestnut tree (*Castanea vesca*) have long had a popular reputation, and many physicians have confidence in their curative effect. The infusion is the form generally used, an ounce of the leaves to a pint of boiling water and given freely.

Strong testimony has been borne by not a few physicians to the curative efficacy of nitric acid. To a tumblerful of well-sweetened water the acid is to be added until the acidity is of the strength of pure lemon-juice; of this a dessert-spoonful is to be given to a child a year old every hour, and a larger quantity over that age.

Since the bromides were introduced into medical practice they have been employed considerably in this affection, and with apparent benefit. These remedies are probably useful as pharyngeal and laryngeal anæsthetics; hence they are addressed to the neuropathic element of the affection.

Other remedies which have been recommended, and which I shall simply name, are tannic acid, asafœtida, arsenic, quinia, chloroform, the oxide of zinc, and ergot. A method of treatment which it was claimed had proved remarkably successful was some years since brought before the French Academy of Medicine—namely, inhalation of the fumes disengaged in the purification of coal-gas. It having been observed that children living in the vicinity of gas-works suffered but little from whooping cough and recovered after a short career of the affection, the effect was tried upon a large scale, and, as stated by Commerege and Bertholles in their reports to the French Academy, with signal benefit to a large proportion. To secure the advantage of this measure patients should inhale the fumes at the place where the gas is purified, for two hours at a time, for twelve consecutive days.[1] It would appear from the statements by Blache, Barthez, and Roger that this measure is often inefficacious. It is, however, sometimes promptly curative.

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[1] *A Practical Treatise on Diseases of Children*, by J. Forsyth Meigs, M. D.

Of the different remedies which have been noticed, all are doubtless more or less curative in a certain proportion of cases. It may be doubted if any of them ever have a specific effect. They are, severally, useful in some and not in other cases; the degree of the usefulness of each varies in different cases, and with our present knowledge the physician cannot judge beforehand what particular remedy in any individual case will be most useful. Under these circumstances, if those first selected prove inefficacious, others are to be successively tried.

Palliative treatment is important. Relief may be afforded by opiates in small doses if they be well borne, by ethers, and by hydrocyanic acid. The latter remedy is, of course, to be given with great caution. Strong coffee has been found to be sometimes useful as a palliative. The hydrate of chloral has been found to be an effective palliative remedy, but the physician is not justified in incurring risk of the occasional danger attending its use, even in small doses, especially when the patient is a child and the object is simply palliation. Many of the antispasmodic remedies are useful as palliatives; of these, the value of musk in this as in other diseases is perhaps not sufficiently recognized in this country.

Prof. J. Lewis Smith has found the inhalation of carbolic-acid spray decidedly useful in diminishing the frequency and the severity of the paroxysms of cough. Spray from the following mixture was inhaled from the steam atomizer three times daily, from two to five minutes: Acid. carbolic. ʒss, potass. chlorat. ʒij, glycerinæ ℥ij, aquæ ℥vj.[2] Prof. Smith calls attention to the apparent effect of the treatment, which of late years has consisted chiefly of palliative and sustaining measures, upon the mortality from this disease. Fifty years ago, in the city of New York the proportion of deaths attributed to whooping cough to the deaths from all diseases was 1 in 76, whereas at the present time it is less fatal than any other severe contagious malady. Inasmuch as this is an infectious disease, and is therefore caused by the presence of a specific micro-organism, a curative disinfectant remedy administered internally or by inhalation may hereafter be discovered. Carbolic-acid spray and the fumes of coal-gas probably act as disinfectants.

Hygienic measures form an important part of the treatment. If there be no complications which interfere with exposure to the open air, this should by all means be advised and enforced. Under proper prudential restrictions the more out-of-door life the better. The diet should be nutritious. If from the frequency of vomiting the system suffer from innutrition, alimentation becomes an important object of treatment. The patient should take food often, and as soon after a paroxysm as possible. The times chosen for giving food should be as remote from an expected paroxysm as practicable, and eating at the most favorable times should be insisted upon despite the absence of appetite. Solid is to be preferred to liquid food, as less likely to be rejected by vomiting. Trousseau states that in some cases patients vomit with the paroxysms which occur in the daytime, and not with those occurring during the night; and in such cases food should be taken freely during the night.

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[1] Vide *American Journal of the Medical Sciences*, April, 1865.

[2] Vide article in the *American Journal of the Medical Sciences*, Oct., 1879.

The stomach is sometimes made to tolerate food by minute doses of opium. Change of air is sometimes of signal efficacy in modifying the severity of the affection and bringing it to a close.

Hemorrhages, if profuse or recurring frequently, claim hæmostatic measures of treatment.

Complications call for the therapeutical measures appropriate under other circumstances, making due allowance for the effects of this affection on the circulation, the blood, nutrition, and the general strength of the system.

There is no known method of preventing the disease other than by isolation as regards exposure to the contagium.

CHAPTER XI.

PULMONARY HEMORRHAGE.—PULMONARY GANGRENE.—PULMONARY ŒDEMA.—CARCINOMA.—HYDATIDS.

Pulmonary Hemorrhage.—Bronchorrhagia.—Pneumorrhagia.—Hemorrhagic Infarction.—Pulmonary Gangrene.—Pulmonary Œdema.—Carcinoma within the Chest.—Hydatids.—Syphilitic Disease of the Lungs.

Pulmonary Hemorrhage.

PULMONARY hemorrhage may have as its source—*first*, the bronchial mucous membrane; and *second*, the parenchyma of the lungs. When from the former source, it is called *bronchorrhagia*, and from the second, *pneumorrhagia*. The source of the hemorrhage also may be exterior to the lungs. The most frequent example of this is the rupture of an aneurismal tumor into a bronchus. Hemorrhage having its source in the pulmonary parenchyma may attend diseases in which the air-cells remain intact and diseases causing destruction of the pulmonary parenchyma, especially pulmonary tuberculosis and pulmonary gangrene.

Bronchorrhagia exists in the majority of the cases in which hæmoptysis occurs. The latter term should be limited to the spitting of blood, and should be applied only to the cases in which pure or unmixed blood is expectorated. It is not properly applicable to sputa streaked with blood in cases of bronchitis, or to blood intimately mixed with mucus and inflammatory products in the rusty expectoration of pneumonitis. A true hæmoptysis is the raising of blood, and blood only. In bronchorrhagia the seat of the hemorrhage is most frequently in the small and terminal bronchi. Hemorrhage from the larynx, trachea, or large bronchi is infrequent. In the rare cases in which the loss of blood has occasioned death it has not been found possible to demonstrate the source of the hemorrhage. The blood probably comes from rupture of capillaries and other minute vessels in the mucous membrane of the small bronchi.

It is important to determine, when blood is ejected from the mouth, whether it comes from the air-passages. It may come from the stomach, from the posterior nares, and from the mouth or fauces. If it come from the stomach, it is ejected by acts of vomiting; it is likely to be commingled with other contents of the stomach; it has the characteristic acid odor of the latter; and it gives an acid reaction, whereas pure blood is alkaline. Coming from the stomach, it usually has a black grumous appearance, due to the action of the gastric acids. In some cases of profuse bronchorrhagia considerable blood may be swallowed and afterward vomited. In a case which I saw in an emergency, the blood coming most profusely from the mouth and nose, the source of the hemorrhage was evidently within the air-passages. Some hours afterward, when the patient was seen by the attending physician, the hæmoptysis had ceased, and a considerable quantity of blood was exhibited which had evidently been vomited. In this case there were both hæmoptysis and hæmatemesis, the latter following the ingestion of blood derived from the air-passages. On the other hand, blood which has flowed into the stomach from the erosion of a large vessel, and is vomited before it becomes mingled with the gastric juice, may retain its bright-red color and its alkalinity. If it come from the posterior nares, it is in the form of dark, solid sputa which are removed by acts of hawking. If it come from the mouth or fauces, the fact may generally be ascertained by a close inspection of these parts. When it comes from the air-passages, it is raised by acts of coughing, which are generally not violent; the blood rises into the trachea and larynx and is expelled with slight efforts. In the majority of cases the blood is liquid, of a bright arterial hue, and contains air-bubbles in more or less abundance. If, however, the hemorrhage have taken place slowly into the bronchial tubes, and the blood have remained there for some time before being expectorated, it undergoes coagulation and acquires a dark or almost black appearance.

The amount of hæmoptysis varies much in different cases. It is sometimes quite small, only a drachm or so of blood being raised; usually, however, when the amount is relatively small or moderate, several drachms or a few ounces are expectorated. Not infrequently the amount is considerable. It is not rare for patients to lose during an attack of hæmoptysis from half a pint to a pint of blood, and sometimes a much larger quantity. The blood is expectorated with more or less rapidity. The continuous duration of an attack of hæmoptysis may vary from a few minutes to several hours, and even many days; in the latter case, of course, the blood escaping slowly. Occasionally the flow of blood is so rapid that it escapes simultaneously from the nose and mouth, and death by suffocation may result from its accumulation in the air-passages. A single attack only may occur, or attacks may be repeated at intervals varying greatly in duration in different cases. In some cases attacks recur daily or repeatedly during the day for a variable period. In other cases the attacks recur after days, weeks, months, or years. After an attack it is common for the expectoration to be more or less colored with hæmatin for several days. In proportion as it is rapidly discharged it is less frothy than when the quantity expectorated with each act of coughing is small. When the hæmoptysis is rapid and abundant, if the patient be not in advanced phthisis, the bursting of an aneurism should be suspected. The hemorrhage from this source, however, is not always at first abundant and rapid, the opening being for a time too small for the free escape of blood. In cases of phthisis advanced to the cavernous stage the hæmoptysis may be rapid and abundant, leading sometimes to fatal syncope, the hemorrhage being due to the opening of a vessel contained in one of the bands of pulmonary tissue which frequently traverse tuberculous cavities, or to the rupture of the minute aneurismal dilatations which late observations have disclosed in the walls of these cavities.

In the majority of cases bronchial hemorrhage is in some way pathologically connected with pulmonary phthisis, and from the frequency of this connection it is important as a diagnostic symptom of that disease. Not infrequently it is the first event which awakens the attention of patients to the existence of pulmonary disease. In a large proportion of cases it occurs more or less frequently during the progress of phthisis. In a certain proportion of cases it occurs when physical signs do not afford evidence of the existence of phthisis, these signs becoming developed sooner or later after its occurrence. In these cases it is probable that tubercles already exist in the terminal bronchi, and that the hemorrhage is due to a tuberculous ulceration into the blood-vessels in the coats of the bronchi. In many cases of initial hæmoptysis of phthisis tubercle bacilli have been found in the blood or sputum, and in this way valuable aid in diagnosis may be afforded by the examination of the sputum. Hæmoptysis should always excite a strong suspicion of tuberculous disease, yet its significance in this respect was undoubtedly overestimated by Louis in his treatise on phthisis. Louis, having questioned a large number of patients affected with various diseases other than tuberculosis, and finding that spitting of blood had taken place in no instance save after injury of the chest or when the catamenia were suppressed, concluded that this symptom is almost pathognomonic of phthisis. Since the publication of that work, however, it has been found that cases are not very infrequent in which hæmoptysis is not accompanied or followed by either the symptoms or signs of tuberculous disease. A paper communicated by the late Prof. John Ware contains statistical information of special value with reference to this point.[1] Ware in this paper gave the results of an analysis of 386 cases of hæmoptysis noted in private practice during a period of about forty years. Of these cases, in 62 recovery from the bronchial hemorrhage took place, and the patients afterward were either known to be living in ordinary health or to have died of other diseases having no connection with the existence of tubercles. The length of time during which this immunity continued varied from two to thirty-seven years. In addition to these cases, in 52 a similar complete recovery took place, and, so far as known, there was no development of tuberculous disease; but the entire subsequent history of these cases had not been obtained. Making allowance, in a certain proportion of the cases, for the probable occurrence of small phthisical affections which were arrested and did not return, these facts show that neither the existence of phthisis nor a strong proclivity thereto is to be positively predicated on the occurrence of bronchorrhagia. They show, moreover, that as regards any immediate or remote evils bronchial hemorrhage may be innocuous. These conclusions are corroborated by the facts developed by my own clinical studies relating to phthisis.[2]

Bronchorrhagia is incidental to certain cardiac lesions, especially those involving obstruction at the mitral orifice. These lesions lead to bronchial hemorrhage by inducing pulmonary congestion. It may occur in connection with morbid conditions of the blood and tissues, which lead to hemorrhages from mucous membranes in different situations, as in purpura hæmorrhagica and scorbutus. It occurs occasionally in asthma. It may be produced by violent and prolonged muscular exertions, without the coexistence of any pulmonary or cardiac affection, and it has been observed to occur from muscular exertions, not excessive, if conjoined with diminished pressure of the atmosphere in elevated situations, as in ascending high mountains. It may be produced traumatically by injuries and wounds of the chest. It may occur as a secondary hemorrhage when the menses are suppressed. Well-authenticated cases of this kind have been reported, but they are exceedingly rare. I have met with a striking example, the hæmoptysis occurring regularly for the four years following the suspension of the menses, and no other pulmonary affection either accompanying or following the hemorrhage. The

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[1] "On Hæmoptysis as a Symptom," by John Ware, M.D., etc., *Publications of the Massachusetts Medical Society*, 1860.

[2] Vide work on *Phthisis*, 1876.

386 cases analyzed by Ware did not include a single case in which the hemorrhage could be considered as compensatory for the menses. Exertion and mental excitement, by their effect on the circulation, may act as exciting causes if, from pulmonary disease or other circumstances, a predisposition exist; but clinical observation shows that in the larger proportion of cases the hemorrhage takes place without any apparent exciting cause. It takes place frequently during the night-time.

The occurrence of hæmoptysis generally occasions much alarm and anxiety. If patients be seen at the time, the nervous agitation and disturbance of the circulation, which are often present, are attributable in a great measure, if not altogether, to the mental condition induced by the attack. The first duty of the physician is to endeavor to remove needless apprehensions from the mind of the patient. Hæmoptysis proceeding from bronchial hemorrhage may destroy life either by suffocation or by exhaustion from the loss of blood, but the cases are so rare that the danger of fatal consequences is scarcely to be considered. The physician may assure the patient of the absence of any immediate danger; and, in general, the hemorrhage does not lead to any evil results. It is suggestive of the existence of phthisis, but aside from its symptomatic significance it affords little occasion for alarm. So far from favoring a tendency to tuberculous disease, there is ground for the conjecture that it is sometimes preventive of that disease; and when it occurs in connection therewith, clinical observation shows that it exerts no unfavorable influence on the progress of the disease, but, on the contrary, its influence seems, as a rule, to be favorable.

In exceptional cases bronchial hemorrhage apparently leads to either the development or an increase of pulmonary symptoms, together with more or less fever. The immediate local and constitutional sequences are sometimes exceedingly grave, and death may take place within a few days. I have met with several cases in which, dating from an hæmoptysis, rapid breathing, dyspnœa, frequency of the pulse, and a high temperature occurred, the cases ending fatally after a certain number of weeks or months. Such cases, however, are rare. Niemeyer held the opinion that the retention of blood in the bronchi and air-cells after an hæmoptysis excites inflammatory changes which lead to cheesy degeneration and phthisis. He considered phthisis to be the result, and not the cause, of this initial hæmoptysis. Experiments, however, have shown that the presence of blood in the bronchi and air-cells may excite a lobular, but never a caseous, pneumonia or tuberculosis. The view of Rindfleisch, therefore, is more probable, that the hemorrhage may hasten the development of phthisical symptoms by occluding the bronchi and causing atelectasis and lobular pneumonia, but that in these cases tubercles are already formed before the hæmoptysis and stand in a causative relation to it. In cases of death directly following a profuse hemorrhage the bronchial tubes have been found, post-mortem, filled with clots, and the obstruction of respiration caused by these doubtless was the immediate cause of death. Blood-casts of the bronchial tubes are sometimes expectorated.

The measures of TREATMENT directed to the hæmoptysis must have reference to the profuseness or otherwise of the hemorrhage and the associated symptoms. Venesection heretofore has been frequently employed. This, however, is indicated only in cases in which the patient is full-blooded or the pulse abnormally strong; and such cases are comparatively few. Even in these cases, unless the escape of blood be unusually rapid and abundant, a saline purgative and sedative remedies are to be preferred to bloodletting. If the hemorrhage be profuse and persisting, cold may be applied to the chest, as in hemorrhages in other situations. This may be done by means of compresses wet with iced water or with ether, which latter refrigerates by its rapid evaporation. The application of the ice-bag to the chest has been recommended, and I have resorted to it with apparent success and without any untoward consequences. Revulsive measures may be employed, consisting of stimulating pediluvia, sinapisms, and dry cupping. To diminish the amount of blood returned to the heart, temporary ligation of the extremities may be resorted to. I have witnessed a prompt arrest of the hemorrhage by this measure. A popular remedy is common salt, which usually has been freely given before patients come under the care of the practitioner. Its efficacy is doubtful, but its use serves to occupy the attention of the patient and friends until medical advice is obtained.

In all cases quietude of body is to be enjoined, the use of the voice is to be restrained, the apartment should be kept cool, the head and shoulders should be raised, the diet should be bland or unstimulating, drinks should be cold, and small pieces of ice may frequently be taken into the mouth. These measures are doubtless of more or less importance, yet I have repeatedly known patients who, having become accustomed to attacks of hæmoptysis, paid little or no attention to it, and kept about their ordinary pursuits as usual, without apparently increasing or protracting the hemorrhage by this course. I have known a lecturer continue the daily use of the voice in public speaking without any apparent injury. The measures just enumerated are often continued too long, patients being anxious to prevent a recurrence of the hemorrhage after it has ceased. They may be assured that moderate exercise out of doors, a nutritious diet, etc. will not be likely to reproduce an attack.

Cough, if present, should be quieted by some form of anodyne. Anodyne remedies are also generally indicated by the excited condition of the nervous system. If the hemorrhage be slight or moderate, as a rule nothing is required in addition to these remedies and such hygienic regulations as may be deemed prudent. If, however, the hemorrhage be considerable and prolonged, hæmostatic remedies are to be employed. Of these, probably ergot is the most efficient. From one to two drachms of the fluid extract may be given hourly or half hourly according to the urgency of the indication, the doses being repeated for several hours if required. Krameria and tannic or gallic acid are also efficient vegetable astringent remedies. Of the astringents belonging to the mineral kingdom, the acetate of lead has long been considered valuable in this application. The most efficient preparations of this class, however, are the persulphate and pernitrate of iron. The inhalation of astringent remedies in the form of atomized liquids or spray has been found effective for the arrest of hemorrhage. For this purpose solutions of alum, tannin, and the perchloride of iron may be employed.

I have met with a few cases of persistent bronchial hemorrhage which claim a separate notice. The hemorrhage in these cases was slight, but it continued for a long period, without other evidence of pulmonary disease. It was not referable to disease of the heart, to suppression of the menses, or to any appreciable cause. Moreover, the hemorrhage was confined to the bronchial mucous membrane, and the blood did not come from either the mouth, throat, or nasal passages. The hemorrhagic expectoration was not pure blood, but a thin, bloody liquid. I have notes of three such cases. In two of these cases, after expectorating almost daily more or less of a sero-sanguinolent liquid during several months, recovery took place under the use of tonics and invigorating hygienic measures. In the third case the hemorrhagic expectoration continued for six years, during which period the patient, a young woman, from time to time came under my observation. Repeated examinations of the chest by others as well as myself failed to discover any positive signs of pulmonary disease. During the greater part of the long period just named there was daily more or less expectoration of liquid containing blood-coloring matter, the quantity being always small. Associated with this symptom were anæmia and hysterical ailments. Aside from these nothing was discoverable. The subsequent history of this case is unknown.

Pneumorrhagia, or the extravasation of blood into the air-cells, and frequently also into the interstitial tissue, is commonly known as pulmonary apoplexy. There would be no impropriety in this use of the term apoplexy if we adopted the custom of French writers of expressing by it extravasation of blood in any situation; but with us when applied to a cerebral affection it includes cases in which there is no extravasation of blood, and it is not customary to apply it to extravasation elsewhere than in the brain, spinal cord, and lungs. The name pneumorrhagia is to be preferred.

Extravasation of blood into the pulmonary parenchyma may occur either with or without laceration of the pulmonary tissue. Extravasation with destruction of pulmonary parenchyma is called *diffuse* pulmonary apoplexy or *diffuse* pneumorrhagia. In *circumscribed* pneumorrhagia or hemorrhagic infarction the blood is in the air-cells and interstitial tissue, but there is no rupture of the lung-tissue. Hemorrhage with laceration of the pulmonary tissue is most frequently the result, either of traumatism, such as stab-wounds and gunshot injuries of the lungs, or of the rupture of an aneurism adherent to the surface of the lungs. The blood lies in a cavity of which the walls consist of torn lung-tissue. The tissue surrounding the cavity is usually diffusely infiltrated with blood. There may be at the same time rupture of the pleura followed by a discharge of blood into the pleural sac. This diffuse hemorrhage, with destruction of the pulmonary parenchyma, is less frequent, but more serious, than the circumscribed form.

In cases of bronchial hemorrhage the blood may be drawn by inspiration into the air-cells and produce the appearance of hemorrhagic infarction. Blood may be inspired likewise into the air-cells in cases of wounds of the lingual artery or of suicide by cutting the throat. Hemorrhage from phthisical cavities and from gangrenous disintegration of the lungs is noticed when treating of these affections. Excluding these conditions, the most important causes of pneumorrhagia without laceration of pulmonary tissue are active and passive congestion of the lungs, injuries and disease of the central nervous system, especially cerebral hemorrhage, diseases attended by a hemorrhagic diathesis, as scorbutus, purpura, yellow fever, and acute yellow atrophy of the liver, and either thrombosis or embolism of branches of the pulmonary artery.

The term *hemorrhagic infarction* is applied especially to hemorrhage following embolism of branches of the pulmonary artery, the most frequent and important of the causes just mentioned. These embolic infarctions are firm, dark-red in color, and wedge-shaped, with the base of the wedge toward the surface of the lung and the apex toward the hilum. In the majority of cases the base of the infarction reaches the surface of the lung, the pleura over the infarction being usually coated with a delicate layer of fibrin. These infarctions may be single, but they are frequently multiple. They may occupy the greater portion of a lobe, but usually they are about the size of a walnut or pigeon's egg. They are most common in the posterior portion of the lower lobe. The affected portion of lung is free from air. The microscopical examination shows the air-cells filled with blood and the alveolar septa compressed and infiltrated with red blood-corpuscles. There are fewer nuclei than normal in the walls of the alveoli, in consequence of coagulation necrosis (p. 52). The tissue surrounding the infarction is congested, œdematous, or solidified with pneumonic products. Emboli may lodge in branches of the pulmonary artery without causing hemorrhagic infarctions. Although the branches of the pulmonary artery do not anastomose with each other, and therefore belong anatomically to the terminal arterial system, blood may be brought to the capillaries of an occluded artery by means of anastomoses with the bronchial artery and other small arteries which enter the lung (Küttner), and by means of surrounding capillaries. In many cases these anastomoses suffice to carry on the circulation in a district, the main artery of which has been obstructed by an embolus; but if the circulation be enfeebled by weakness of the right side of the heart, or if there be obstruction to the flow of blood from the pulmonary veins into the left side of the heart, the anastomoses no longer are sufficient, and a hemorrhagic infarction results within the district the artery of which contains an embolus. Cohnheim's explanation has been generally accepted, according to which the blood in the infarction is derived by a regurgitant flow from the veins leading from the affected district; but according to Litten's experiments the blood is brought to the capillaries of the occluded artery by the small anastomosing arteries mentioned above and by adjoining capillaries, the pressure in which does not suffice to force the blood into the veins. The blood escapes by diapedesis and not by rhexis. (See *Embolism and Thrombosis*, Part I. p. 28.) Infarctions are more frequent in the periphery than in the interior of the lungs, because the anastomoses are fewer and the circulation feebler in the former situation. If life be prolonged, the blood in a hemorrhagic infarction may be in great part absorbed, and only pigmented fibrous tissue, or possibly only a pigmented stain, be left behind.

In the majority of cases hemorrhagic infarctions in the lungs are associated with cardiac disease, especially with mitral obstruction or regurgitation. They appear when the right cavities of the heart have become weakened and dilated and thrombi have formed in the right auricle or ventricle. These thrombi are the source of emboli which are carried into branches of the pulmonary artery. It is possible that thrombi may form in branches of the pulmonary artery in heart disease. Pulmonary infarctions may be the result also of emboli derived from thrombi in the peripheral veins, as in cases of thrombosis of the femoral vein. It is not always possible to demonstrate a source of emboli in cases of pulmonary infarction.

The DIAGNOSIS of either form of pneumorrhagia is by no means always easy. Hæmoptysis is of course present when the source of the hemorrhage is bronchial and the extravasation is due to the inhalation of blood into the cells. This symptom is also present in the majority of cases in which the hemorrhage takes place within the cells, but it may be wanting. When present, the hæmoptysis may be either small or abundant; and the amount of extravasation is by no means in proportion to the quantity of blood expectorated. The respirations are hurried and labored in proportion to the extent of lung solidified by the extravasated blood. Dyspnœa and a sense of oppression are also proportionate to the degree in which the respiratory function is compromised. These symptoms, however, are measurably due to the coexisting cardiac lesions when the latter stand in a causative relation to the hemorrhage. Dulness on percussion will be found over a space or over spaces corresponding to the situation and extent of solidification. This may not be appreciable if the extravasation be in the form of small disseminated nodules; but if the extravasation be diffused over a considerable space, the dulness becomes a very significant sign, provided tuberculosis, pneumonitis, and other affections involving solidification can be excluded. A feeble bronchial respiration may be associated with the dulness on percussion, or the respiratory sound may be suppressed over the extravasation. The presence of blood in the cells and smaller air-tubes will be likely to give rise to the subcrepitant, and perhaps to a well-marked crepitant râle within a limited space. These signs, suddenly developed in connection with hæmoptysis and with mitral cardiac lesions, may lead to a diagnosis; but in the cases in which no blood is expectorated a positive diagnosis is extremely difficult if not impracticable.

Circumscribed extravasation, incidental to bronchial hemorrhage, is not necessarily serious; and perhaps it occurs to a limited extent not infrequently in cases of abundant hæmoptysis followed by recovery. If there be no injury of the pulmonary structures and the blood simply infiltrate the air-cells, it may be gradually removed by absorption and expectoration, the normal condition being restored, as after solidification from pneumonitis. If due to embolism, the hemorrhage may lead to gangrene, or the infarcted portions may become the sites of embolic pneumonia and the so-called metastatic abscesses. The latter do not occur unless the emboli contain infectious matter. If these results do not follow, and if the number and size of the spaces in which the extravasation has taken place be not large, recovery may take place. When, however, the hemorrhage into the air-cells and into the areolar tissue is considerably diffused, the termination is usually fatal and death may take place speedily. Other things being equal, the gravity of the symptoms and the immediate danger are in proportion to the amount of extravasation.

The TREATMENT of different forms of pneumorrhagia must have reference to the symptomatic indications in particular cases, in addition to analeptic and supporting measures.

Pulmonary Gangrene.

Pulmonary gangrene may be defined as death or necrosis of lung-tissue, accompanied by decomposition. It is one of the infrequent pulmonary affections. Its occurrence in cases of lobar pneumonitis and of hemorrahgic infarction has already been referred to. Its most frequent causes are those which either lower the vitality of portions of the pulmonary tissue or favor the introduction into the lungs of decomposing or infectious substances. Among the causes may be mentioned interruption of the blood-current by compression or embolism of the pulmonary or bronchial vessels; infectious diseases, especially those accompanied by the formation of septic emboli; penetrating wounds of the lungs; severe contusions of the chest; decomposition of the contents of bronchiectatic and of tuberculous cavities; the entrance of foreign substances into the bronchi, such as bits of food in attempts to artificially feed insane patients; the ulceration of abscesses and of tumors (*e. g.* cancer of the œsophagus) into the bronchi, etc. Pulmonary gangrene occurs more frequently in males than in females, and also in those whose powers of resistance are enfeebled by alcohol, poverty, bad hygiene, or disease. It sometimes occurs in diabetes mellitus. It may occur as an idiopathic affection in previously healthy individuals; that is, without any apparent cause.

It is presented in two forms, which are distinguished as *diffused* and *circumscribed*. In the circumscribed form the gangrene occurs in sharply-defined spots of variable size, usually between that of a bean and that of a hen's egg. There may be only one gangrenous focus or there may be several foci. They are more frequently situated in the periphery than in the central parts of the lung, and more frequently in the lower than in the upper lobes. The gangrenous spot at first is dark-brown or greenish, dry and friable. Later it undergoes softening, and a cavity is formed, with sloughy walls, containing an extremely offensive greenish or brown fluid holding in suspension shreds of decomposed lung-tissue. The microscopical appearances of the fetid contents of the gangrenous cavities are identical with those of the sputa. The lung-parenchyma immediately surrounding the gangrenous centre is usually in the condition of lobular pneumonia; and not infrequently it is the seat of hemorrhages. The gangrenous process tends to spread peripherally. In the cases in which the disease terminates in recovery the walls of the cavity become cleaned off and free from the adherent shreds of lung-tissue; a kind of pyogenic membrane is formed; and finally, by adherence between the opposite sides of the cavity or by the growth of granulations, the cavity is entirely obliterated, only a cicatrix remaining. In the process of extension of the gangrenous process the blood-vessels are usually thrombosed before they disappear; but sometimes they are opened before they become impervious to the blood-current, and then severe and even fatal hemorrhage may ensue. When the gangrenous patch reaches the surface of the lung, the overlying pleura is covered with a layer of fibrin. Frequently, general pleuritis results, which may be of a gangrenous character. Sometimes the gangrenous focus opens into the pleural cavity, giving rise to pneumo-pyothorax. Thrombi may form in the veins in the neighborhood of the sloughy centres in the lung. From this source infectious emboli may be carried to the various organs of the body. Intense bronchitis, sometimes of a diphtheritic character, may result from the irritation of the gangrenous matter.

Diffuse gangrene may be secondary to the circumscribed form, or it may be diffuse from the first. The greater portion of a lobe, a whole lobe, or even a whole lung, may be involved. The pulmonary parenchyma is broken down into a putrid, greenish, or blackish pulpy mass, without an evident line of demarcation. It is always fatal.

The SYMPTOMS in cases of gangrene relate measurably to the general condition or to associated affections. The pulmonary symptoms due to the gangrenous condition at first are those which belong to circumscribed pneumonitis, consisting of cough, some pain, and accelerated breathing, and after a time an expectoration, more or less abundant, of characteristic appearance and odor. The gross and the microscopical appearances of the sputa have been studied carefully by Traube[1] and by Leyden and Jaffe.[2] The sputa are of a yellowish, greenish, or brownish color according to the varying amount of pus and of blood present. They emit a penetrating, intensely fetid odor. When fresh they are alkaline, but upon standing become acid. By allowing the expectoration to stand for a while in a glass dish three distinct layers become visible: the upper is frothy, opaque, and of a dirty gray or yellowish color; the middle is clear and watery; and the lower is greenish and purulent, or it may be brownish from admixture with pigment or blood. In this lower layer are shreds of lung-tissue visible to the naked eye, and gray or whitish masses. Microscopical examination reveals granular detritus, pus-corpuscles, triple phosphate crystals, drops of fat, and acicular crystals of the fatty acids, often in bunches. In addition, granular or angular fragments of brown or black pigment, round and rod-shaped bacteria, spiral bacteria, leptothrix threads, and infusoria are met with. The bacteria and fatty drops and crystals are often compacted together into little yellowish clumps visible with the naked eye. The shreds of lung-tissue seen with the naked eye are of a dirty gray or black color, and consist chiefly of granular detritus, dark pigment, and bacteria, with occasionally some elastic fibres. Elastic fibres are rarely present, according to Traube, in the gangrenous expectoration. Later observations show that Traube underestimated the frequency with which elastic fibres are found in gangrenous sputa; still, it remains true that the microscopical shreds of lung-tissue frequently found in the expectoration of pulmonary abscess contain many elastic fibres, often marking out the outlines of the alveoli, whereas elastic fibres are more scanty in the sputum of pulmonary gangrene.[1]

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[1] *Deutsche Klinik*, 1853, 1859, 1861 *et seq.*

[2] *Deutsches Archiv f. klin. Med.*, ii. p. 488.

Pulmonary gangrene may be confounded with fetid bronchitis, decomposition in the contents of dilated bronchi and of tuberculous cavities, and with pulmonary abscess. The diagnosis is not to be based upon the gangrenous odor of the breath and sputum alone. In fetid bronchitis and in cases of decomposition of the contents of pulmonary cavities microscopical shreds of lung-tissue are not present in the expectoration; in pulmonary abscess they are often present, but they contain abundant elastic fibres, which are less abundant and are often absent in pulmonary gangrene. Again, pulmonary abscess is not usually accompanied by the fetor of gangrene.

In cases of circumscribed gangrene the gangrenous portion of lung, together with the solidification from inflammatory exudation around this portion, furnishes dulness or flatness on percussion within a circumscribed space, which, in view of the most frequent situation of gangrene, will generally be over the scapula and below the spinous ridge. Within this space either the respiratory murmur is wanting or there is a feeble bronchial respiration, with, perhaps, either exaggerated vocal resonance or weak bronchophony. Moist bronchial or bubbling râles are likely to be heard within and around this space. If the patient survive the sloughing and removal by expectoration of the decomposed mass, the cavernous signs may be discovered. I have repeatedly found a well-marked cavernous respiration, and I have observed a circumscribed depression in front corresponding to the situation and size of the gangrenous cavity.

As regards PROGNOSIS, if circumscribed gangrene do not occur under circumstances which are dangerous, irrespective of the gangrene, recovery may take place. The chances of recovery are differently estimated by different writers, which perhaps may be accounted for by supposing that the diagnosis of gangrene is not infrequently based on insufficient grounds. It can hardly be doubted that the result is fatal in a large majority of cases. Hæmoptysis, as already stated, sometimes takes place in connection with the separation of the slough, and I have known the hemorrhage to be so abundant as to prove the immediate cause of death. Most of the cases end fatally in which the gangrene involves the pleura, perforation of the lung taking place, followed by pleuritis with pneumothorax.

The TREATMENT of pulmonary gangrene is to be directed more to the system than to the local affection. The affection never occurs under circumstances which render depletion or other debilitating measures appropriate; on the contrary, such measures can hardly fail to be pernicious. Tonic remedies and the sustaining treatment are indicated. The diet should be as nutritious as possible, and alcoholics are to be given with a freedom proportionate to the tendency to failure of the vital powers. The employment of opium in some form is important, in order to palliate pain or undue cough and to allay constitutional irritation. The chlorate of potassa has been suggested as likely to be beneficial, from its apparent usefulness in gangrenous affections of the mouth and throat. The tincture of the chloride of iron is suggested by the same analogy. Disinfectants by inhalation are indicated as long as the sputa contain decomposed matter and the breath has a fetid odor. The vapor of turpentine poured upon boiling water is a disinfectant, and has otherwise a salutary local effect. Tar vapor, produced in the same way, is useful. A solution of carbolic acid of the strength of from 2 to 4 per cent., atomized, and inhaled for a few minutes at short intervals, is probably the best disinfectant. Thymol and salicylic acid are recommended as appropriate as disinfecting inhalations.[1]

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[1] According to the experiments of Filehne—"Ueber die Vorgänge bei dem Lungenbrande," *Sitzungsberichte der Phys. Med. Soc. zu Erlangen*, Jan. 11, 1877—in the sputum of pulmonary gangrene a digestive ferment is present capable of dissolving in alkaline solution the elastic fibres and albuminous substances, but not acting on ordinary white fibrous tissue, which may be found in gangrenous sputum when the elastic fibres are absent. Elastic fibres resist putrefactive changes much longer than those induced by digestion.

Pulmonary Œdema.

Pulmonary œdema is always dependent on other pathological conditions, and is not entitled to be considered as an individual affection. It is, however, an event of frequent occurrence, and is important as interfering with the function of respiration, in not a few instances proving the immediate cause of death. In œdema of the lungs there is a transudation of serum into the interlobular tissue, the alveolar walls, and the air-cells.[2]

Œdema may be localized around some diseased centre in the lung, as, for example, in the neighborhood of a pneumonitis, an infarction, abscess, tumor, etc. This is distinguished as *collateral œdema*. In this case it probably results from a moderate inflammatory change in the vessel-walls, by which their permeability is increased. This variety has also been called inflammatory œdema. Hypostatic œdema, following hypostatic congestion and leading to the condition of lung called splenization, has already been referred to.

These forms differ from general pulmonary œdema, which is usually understood by the term œdema of the lungs. Here a serous transudation occurs throughout both lungs, except in portions which may perhaps present a mechanical obstacle to the entrance of the serum in consequence of consolidation, as from compression, inflammatory exudation, new growths, etc. Œdematous lungs are increased in volume and weight; retract less than healthy lungs upon opening the chest; have a characteristic boggy feel compared to that of a sponge filled with water; and pit on pressure. They are less buoyant than normal lungs, but they do not sink in water. The color is usually red, but it may be pale. On section, a serous or sero-sanguinolent liquid escapes in abundance, usually mixed with air-bubbles. The same frothy liquid is present in the bronchi and usually in the trachea. Microscopical examination in cases of acute general pulmonary œdema shows the pulmonary capillaries widely distended with blood, and the contents of the alveoli and bronchi to consist of serum containing a considerable number of red blood-corpuscles, some granular epithelial cells washed off from the alveolar walls by the transuding serum, and free granular matter. The red blood-corpuscles, which are always present in the serum, have escaped by diapedesis. The serum infiltrates also the alveolar septa.

Œdema of the lungs may occur under various pathological conditions. There is hardly a grave disease, acute or chronic, which it may not complicate. It is also true that there is no disease of which it is a necessary accompaniment. It occurs often during the death-agony, when it seems to be rather an accompaniment than the cause of death. Of the affections during the course or at the termination of which it most frequently occurs may be mentioned acute and chronic Bright's disease, valvular lesions of the heart, pneumonitis, fevers (especially those of an asthenic type), cerebral lesions (especially hemorrhage and traumatism), and cachexiæ giving rise to a hydræmic state of the blood. It occurs during these and other diseases, especially when the heart's action becomes enfeebled. It is frequently developed suddenly, and may disappear likewise with considerable rapidity. It may prove quickly fatal by interference with respiration. The great variety of conditions in which it appears, the inconstancy of its development under apparently similar conditions, the rapidity of its onset in many cases, and its production during the death-agony are among the considerations which at first seem to give to it a pathological position having no analogy to the dropsical accumulations in other organs, and which render difficult the explanation of its production.

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[1] For an interesting case of pulmonary gangrene treated surgically by thoracentesis and drainage, with practical remarks, vide article by Drs. Fenger and Hollister in the *Am. Journ. of Med. Sciences*, Oct., 1881; vide, also, the *London Lancet*, May 31, 1884, for cases treated in the same manner by Mr. A. Pearce Gould.

[2] Posner (*Virchow's Archiv*, Bd. 79, p. 362, 1880) finds that the dropsical effusion is first into the walls of the air-cells, and that it may be confined to them (interstitial œdema). Usually, however—and in the higher grades of œdema always—the serum escapes also into the air-cells.

That hydræmia, pulmonary congestion by obstruction at the mitral orifice, and general weakness of the heart are only predisposing, not immediate, causes of pulmonary œdema is evident from the fact that all these conditions may exist in a high degree without the development of œdema of the lungs. This fact is proved both by clinical observation and by the establishment of these states artificially in animals. There must be added some other factor which acts as the immediate and exciting cause—a factor which is capable of developing under various conditions, and one which can appear suddenly and as rapidly disappear. It is also to be assumed that this factor must be something which will increase the blood-pressure in the pulmonary capillaries; for that œdema of the lungs is a mechanical œdema—that is, a result of increased tension in the capillaries of the lung—is rendered probable by microscopical appearances and by our knowledge in general of dropsical effusions. That mechanical hindrance to the outflow of blood from the pulmonary veins, as by mitral stenosis, is capable of producing this elevated tension seems *a priori* probable; but this is a condition present in only a minority of cases of pulmonary œdema, and the experiments of Welch,[1] in reference to the cause of œdema of the lungs, have shown that the obstacle must be greater, in order to produce œdema, than can well occur in man. He, however, found that an efficient cause of pulmonary œdema exists in paralysis of the left side of the heart, the force of the right side being not at all or less impaired. Neither general paralysis of the heart nor weakness alone of the right ventricle suffices to produce pulmonary œdema. But, as was proved experimentally, it appeared when the left ventricle was so disproportionately weakened that it could not propel into the general circulation the same amount of blood propelled by the right ventricle into the lungs. The blood accumulated in the lungs until the tension in the pulmonary capillaries became sufficient for the transudation of serum through the vascular walls. The assumption that disproportionate paralysis of the left ventricle is the immediate cause of acute general pulmonary œdema in man would explain many of its singular phenomena. Such a condition of the heart can be readily supposed to develop suddenly, to rapidly disappear, and to arise during manifold diseases, especially when the general activity of the heart is impaired; for it is not denied that the general force of the heart is weakened in most cases of pulmonary œdema; and, indeed, it is hardly conceivable that the left ventricle should lose its wonderful power of adaptation to the obstacles which it has to overcome in propelling the blood, before the whole heart had suffered in its function. Œdema of the lungs occurs during the death-agony, when the left ventricle loses its power more rapidly than the right, or dies faster, so to speak. While the hypothesis of Welch as to the immediate cause of pulmonary œdema is not controverted by clinical facts, but, on the other hand, seems to explain them better than any other, we as yet know of no means of determining in man the relative force of the two sides of the heart, and upon the absolute strength of either right or left ventricle only subordinate weight is placed. Other influences adduced to explain the cause of œdema of the lungs are impaired nutrition of the walls of the pulmonary capillaries and vaso-motor disturbance.[1]

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[1] William H. Welch, "Zur Pathologie des Lungenödems," *Virchow's Archiv f. Path. Anat.*, Bd. 72, 1878.

The SYMPTOMS of œdema are increased frequency of the respirations and dyspnœa in proportion to the extent of lung affected, together with more or less cough and serous expectoration or bronchorrhœa. The displacement of air in the air-cells by liquid gives rise to dulness more or less marked on percussion over a space corresponding to the œdematous portion of lung, and within this space the respiratory murmur is weak or lost or it may be feebly bronchial or broncho-vesicular. The vocal resonance may be increased. The presence of liquid in the smaller bronchial tubes is denoted by fine mucous or subcrepitant râles. It is stated that a true crepitant râle may be produced, but this must be extremely rare. The diagnosis is to be based on dulness more or less diffused on both sides of the chest, associated with moist bronchial râles, pneumonitis being excluded, and the existence of Bright's disease, cardiac lesions, and other predisposing conditions being considered.

The TREATMENT must have reference to the circumstances under which the œdema occurs. Occurring in connection with disease of the kidneys, those measures are indicated which are applicable to dropsical effusion in other situations—namely, hydragogue cathartics and sudorifics selected and regulated according to the circumstances belonging to individual cases. When incidental to disease of the heart the main reliance is upon revulsive measures, in connection with such remedies as the condition of the heart may claim irrespective of this result. In the cases in which it is dependent on adynamia and an impaired condition of the blood, tonic and sustaining measures are called for. The liability of its occurrence in connection with hypostatic congestion in fevers and other protracted diseases, accompanied by feebleness of the circulation and depression of the vital powers, renders it an important part of the treatment of these diseases to see that the patient be not allowed to lie constantly in one position. The position of the body should be frequently changed, in order to obviate the gravitation of blood to the dependent portions of the lungs.

The name *acute* pulmonary œdema distinguishes cases in which the œdema occurs suddenly to such an extent as to give rise to great dyspnœa, sometimes quickly causing death by apnœa. In such cases prompt and efficient measures of treatment are called for. Venesection, if the action of the heart be not extremely feeble, is the most promptly efficient measure for relief, and should be resorted to at once when the symptoms denote imminent danger. Perhaps under no other circumstances is it more apparent that the physician has saved the life of a patient than in certain cases of acute pulmonary œdema in which venesection was employed. Occurring in connection with dilatation of the heart, or when accompanied with great feebleness of the pulse and cyanosis, the application of a considerable number of dry cups to the chest may be substituted for bloodletting. To these may be added mustard to the chest and stimulating foot-baths. Ethereal and alcoholic stimulants should be given freely. I have known the inhalation of oxygen to afford marked relief.

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[1] See article by H. F. Borden, M. D., *Boston Med. and Surg. Journal*, 1880.

New Growths within the Chest.

Intrathoracic tumors include nearly all the different varieties of new growths with which we are acquainted. Their consideration in detail belongs to works on pathological anatomy. Reference here will be made only to those rendered practically important by reason of their frequency or their gravity.

Pulmonary *carcinoma* may be primary or secondary. Primary carcinoma of the lungs is a very rare affection. As a rule, only one lung is involved, the right lung more frequently than the left. Primary cancer of the lungs may be either of the cylindrical-celled variety, in which case the tumor originates in the larger bronchi, or it may be of the flat-celled variety, in which case the tumor originates in the respiratory bronchioles or the air-cells. In the latter form of carcinoma the stroma consists of the pre-existing alveolar walls, and the appearances are sometimes not unlike a diffuse pneumonia. Primary cylindrical-celled carcinoma of the lungs usually appears as a circumscribed tumor; primary flat-celled carcinoma may be either circumscribed or infiltrated. Hemorrhages are likely to occur in primary cancers of the lungs. Sometimes softening and breaking down of the cancerous mass occurs, whence may arise cavities, abscesses, or gangrenous foci. Secondary inflammation is present in the lung-parenchyma surrounding the cancerous deposit.

Secondary cancer of the lungs is common. It is frequently consecutive to cancer of the stomach and to cancer of the breast, and it may be secondary to cancer in any part of the body. The relation of the lungs to the venous system of the body renders these organs the frequent seats of secondary tumors in general. Secondary pulmonary cancer is usually in the form of nodules varying in size from that of a pea to that of an orange, and disseminated throughout both lungs. Sometimes the lungs, as well as other organs of the body, are studded with small cancerous nodules (general or miliary carcinosis). Sometimes the lymphatic vessels of the pleura and of the lungs are filled with cancerous cells, in which case these vessels appear as a network of white varicose bands. The secondary deposits in the lungs usually reproduce the structure of the primary tumor.

In pulmonary carcinoma the bronchial lymphatic glands, and not infrequently the mediastinal, the cervical, and the axillary glands, are involved.

A form of cancer, which is sometimes described as *endothelioma* or endothelial cancer, occurs primarily in the pleura. The same form of tumor is also described as originating in the lymphatic vessels of the pulmonary parenchyma.

The pleura may be studded with secondary cancerous nodules without any tumors in the lungs. Cancer of the pleura is often accompanied with abundant liquid effusion in the pleural cavity. The effusion is frequently hemorrhagic, and usually contains clumps of large epithelial cells, described as grouped cells and budding cells. The recognition of these clumps of cells in fluid withdrawn from the pleura during life may give valuable aid in diagnosis.

Of the malignant tumors within the chest, *lympho-sarcoma* is among the most important. Its takes it origin in the cervical, the mediastinal, or the bronchial lymphatic glands. Lympho-sarcomata grow more frequently in the anterior than in the posterior mediastinum. These tumors frequently extend into the lungs, advancing chiefly along the adventitia of the blood-vessels and of the bronchi. The tumor is composed of cells, mostly small round cells, enclosed in a network of anatomosing fibres like the framework of the lymph-glands. Caseous degeneration is rare, but a fibroid metamorphosis is more common.

Enchondroma may be a primary, but it is usually a secondary, tumor of the lung.

The SYMPTOMS of intrathoracic tumors are both general and local. In suspected carcinoma the existence of the cancerous cachexia and the presence of carcinomatous tumors in other parts of the body must be considered in arriving at a diagnosis. Lympho-sarcomata present often the history of pseudo-leucocythæmia or adenia of Trousseau. In carcinoma of the lungs some diagnostic value is to be attributed to enlargement of the supraclavicular lymphatic glands and to a muco-hemorrhagic expectoration compared in its appearance to currant jelly. These two symptoms are, however, not pathognomonic or always present in pulmonary carcinoma. The chest-wall on the affected side may bulge; sometimes, however, especially in hard cancer accompanied by extensive induration, it is contracted.

The DIAGNOSIS of new growths within the chest is to be based on the symptoms taken in connection with the signs of solidification, and sometimes the cavernous signs. The physical evidence is often not very distinctive. Pulmonary phthisis is to be excluded by a careful investigation with reference to the history as well as to the existing symptomatic phenomena. The coexistence of carcinoma in some other situation or its previous existence is to be taken into account in arriving at a diagnosis of carcinomatous intrathoracic disease.

The presence of tumors, great or small, gives rise to dulness or flatness on percussion, with either suppression of the respiratory murmur or the modifications which denote solidification. The tumors may be so situated as to give rise to symptoms and signs proceeding from pressure on important parts other than the lungs. The heart may be displaced. The calibre of the trachea or bronchi may be diminished. Aphonia or laryngeal spasm may be induced if the recurrent nerve be involved. Congestion limited to the upper extremities, head, and neck, and subcutaneous œdema, denote obstruction of the superior vena cava. Pressure on the pulmonary veins may give rise to bronchorrhagia and œdema of the lungs. Compression of the œsophagus occasions difficulty in the ingestion of food and drinks. These results of pressure on adjacent parts also belong to the history of aneurismal tumors. A tumor situated in the tract of the aorta may simulate the physical signs of aneurism. It may pulsate and give rise to a murmur. I know, from my own observations, that it may occasion a double murmur; that is, a systolic and a diastolic murmur. The enlargement of one lung by a new growth, together with either pleural transudation or suppuration, may lead the physician to suppose that he has to deal simply with a case of pleurisy or empyema. The existence of the underlying graver affection can be determined only after removal of the liquid by aspiration. On the other hand, unilateral contraction of the chest is suggestive of fibroid phthisis.

As regards TREATMENT in carcinoma and other new growths, all that can be hoped for is the prolongation of life by a diet as abundant and nutritious as the digestive powers will allow, together with the best available hygienic influences and tonic remedies. Palliative treatment is to be employed in accordance with the symptomatic indications.

Hydatids—Echinococci.

Among the extremely rare affections of the pulmonary organs belong hydatid productions. These may either be developed within the lungs, or they may have been developed in the liver and have made their way through the diaphragm into the pulmonary organs in the manner in which hepatic abscesses are sometimes evacuated into the bronchial tubes, circumscribed peritonitis and pleuritis taking place and leading to adhesions which prevent the evacuation from taking place into either the peritoneal or the pleural sac.

Acephalocysts within the lungs may remain latent for a variable period; that is, giving rise to few or no pulmonary symptoms. Sooner or later, however, they excite inflammation of the surrounding parenchyma and bronchial tubes, and then occasion pain, cough, expectoration, febrile movement, etc. They may be discharged by ulceration into the bronchial tubes, leaving pulmonary cavities. The affection is likely to be mistaken for pulmonary tuberculosis. Microscopical examination of the matter expectorated may show the hooklets of echinococci or pieces of the sac with its characteristic striation and hyaline appearance. Without the demonstration of one of these the diagnosis is impracticable. The question as to the primary seat of the cysts is to be settled by the previous history, which, if they come from the liver, will show the existence of hepatic trouble prior to the occurrence of pulmonary symptoms.

The presence of hydatids in the lungs always involves great danger; and the danger is greater when they are derived from the liver than when they are developed within the lungs. There is no special plan of treatment. Palliative and sustaining measures are to be adapted to the circumstances of individual cases.

Syphilitic Disease of the Lungs.

The most common manifestation of syphilitic disease of the lungs is in the form of a new growth of connective tissue (chronic interstitial pneumonia) beginning at the root of the lung and following the course of the larger bronchi and blood-vessels. By extension of the process to the pulmonary parenchyma more or less extensive patches of indurated connective tissue are produced in the central part of the lung. In rare cases the growth of fibrous tissue is so great as to cause irregular lobulation of the lungs similar to that found in syphilitic livers.

In another form of pulmonary syphilis there is new growth of fibrous tissue around the small bronchi and the blood-vessels, producing peribronchitic and perivascular fibrous nodules studding the lung.

Syphilitic ulcers may occur in the trachea and in the bronchi. These often cicatrize, and may thus lead to serious stenosis of the trachea and of the bronchial tubes.

Gummata are found, although rarely, in the lungs, both in congenital and in acquired syphilis.

There is a peculiar form of syphilitic pneumonia, sometimes described as white hepatization, which is not uncommon in new-born children affected with syphilis. In an early stage of the affection small nodules occur in the lungs; later, a large portion or even the whole of the lungs is involved. The lesion consists in an infiltration of the alveolar walls with lymphoid cells, and an accumulation of cells, chiefly epithelial in character, in the air-cells. This white hepatization, so often found in stillborn syphilitic infants, is of more pathological than clinical interest.

Of late years many cases have been reported as examples of syphilitic phthisis. Some of these cases are instances of syphilitic fibroid induration with chronic bronchitis and bronchiectatic cavities. In most of the cases which have been described as syphilitic phthisis, and which have been examined post-mortem, the lesions are not distinguished from those of chronic pulmonary tuberculosis with caseous pneumonia, and no satisfactory proof exists of the syphilitic nature of the disease. No positive proof of the occurrence of a genuine pulmonary phthisis of syphilitic origin exists. As we now possess in the tubercle bacillus, and perhaps in the bacillus of syphilis, means of diagnosis of these morbid processes, we may hope for a solution of the question as to the occurrence of syphilitic phthisis.

The diagnosis of syphilitic disease of the lung can hardly be made with certainty. Gummata and patches of central induration may be present in the lungs without causing any symptoms. If pulmonary symptoms be present, such as cough, expectoration, dyspnœa, pain in the chest, they are not distinctive. The physical signs of consolidation or of cavity (in case of bronchiectasis) may be discovered. A probable diagnosis may be based upon the history of syphilis; the recognition of syphilitic lesions in other parts of the body, especially in the throat and in the larynx; the exclusion of tuberculous disease of the lungs, particularly by examination of the sputum; and the amelioration or cure of the symptoms by antisyphilitic treatment.

Pulmonary tuberculosis may coexist with syphilis.

CHAPTER XII.

DISEASES OF THE LARYNX AND TRACHEA.

Affections of the Larynx and Trachea.—Points relating to the Anatomy and Physiology of the Larynx, which are Involved in the Consideration of Diseases in this Situation.—Acute Simple Laryngitis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Subacute Laryngitis.—Chronic Laryngitis.—Laryngitis Hypoglottica.—Laryngitis with Fibrinous Exudation: Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Œdema of the Glottis.—Spasm of the Glottis.—Nervous Cough.—Nervous Aphonia.—Paralysis of the Laryngeal Muscles.—Morbid Growths within the Larynx.

IMPORTANT diseases affecting the respiratory apparatus are seated in the larynx and trachea. Diseases of the larynx frequently involve the trachea, but it is rare for the latter to be affected without the former, and it will suffice to consider tracheal affections incidentally in treating of those of the larynx.

Certain anatomical and physiological points pertaining to the larynx are to be kept in mind with a view to a clear apprehension of the diseases in this situation. The larynx is composed of a number of cartilages—namely, the cricoid, thyroid, arytenoid, and epiglottis. The epiglottis was formerly supposed to be essential as a protection against the entrance of food and drink into the laryngeal cavity during the act of deglutition; the removal of this cartilage, however, in inferior animals has shown that its loss does not occasion serious inconvenience. A hospital case in which it was completely destroyed by syphilitic ulceration, as determined by the touch and the laryngoscope, has fallen under my observation. So long as ulceration existed there was considerable difficulty arising from spasm of the glottis excited by contact with food and drink, and deglutition was performed with the least annoyance while the patient was lying on the back. But after the ulceration had healed the difficulty nearly ceased, liquids and solids being swallowed without much inconvenience. The quality of the voice underwent some change, and the patient, a young woman, stated that she had not the power of producing notes in singing as before.

The small size of the rima glottidis is an important point in connection with laryngeal diseases. In the adult male, after death, it is a triangular space an inch in length, and at the base about a quarter of an inch in width, the size being still smaller in women and children. The dimensions in life vary with the two respiratory acts. When examined in a living animal, the vocal cords are found to separate widely in inspiration and to return in expiration, forming the respiratory movements of the glottis. The respiratory movements take place from a reflex influence communicated through the recurrent nerves. These points are of interest and importance in their practical applications.

Other movements of the muscles of the larynx are produced by volition in the acts of speaking. The larynx being the seat of the voice, modifications of vocal sound constitute important symptomatic phenomena of disease. In the movements concerned in phonation the will acts through branches of the spinal accessory contained in the recurrent laryngeal nerves. The laryngeal muscles are subject to spasm and paralysis.

The solidity of the walls of the larynx is an important point in connection with certain affections. The cricoid and thyroid cartilages do not readily yield to internal pressure, and hence arises obstruction from the presence of morbid products which encroach upon the small space between the vocal cords. Another point to which reference will be made is the abundance of areolar tissue beneath the mucous membrane above the vocal cords. This tissue is more abundant in the adult than in the child—a fact which will serve to account for certain differences as regards the effects of disease during and after infantile life.

The mucous membrane of the larynx is covered, for the most part, with ciliated cylindrical epithelium. Over the upper portion of the epiglottis, the true vocal cords, and a portion of the posterior surface of the laryngeal cavity, however, the epithelium is of the pavement variety. The mucous membrane is in most places loosely attached by lax connective tissue to the subjacent muscles and cartilage, but over the true vocal cords it is firmly adherent. Hence in the latter situation submucous inflammatory and serous effusion cannot well occur. Racemose mucous glands are abundant in the larynx. None are present in the true vocal cords. The mucous membrane and the submucous tissue are rich in elastic fibres.

Of the affections of the larynx, those involving inflammation will be first considered. Inflammation here may be simple; that is, not distinguished by any unusual features save those which are incident to the situation of the affected mucous membrane. On the other hand, it may be accompanied by fibrinous exudation. The latter will be considered under a separate head. Simple inflammation may be either acute, subacute, or chronic. These three varieties are to be considered separately. The appropriate name for inflammation affecting the larynx is *laryngitis;* this name should take the place of the terms *angina* and *cynanche*, which were formerly in vogue, but are now rarely used. The term *croup* is applied to laryngitis with fibrinous exudation, and it has also been applied to simple laryngitis and to a non-inflammatory affection—namely, spasm of the glottis—occurring in children. The first of these affections is sometimes distinguished as *true croup*, while the others are called, by way of distinction, *false croup*. The term is an unfortunate one and tends to produce confusion. I shall consider the several affections to which the term has heretofore been applied separately under their appropriate names.

Acute Simple Laryngitis.

ANATOMICAL CHARACTERS.—This variety of laryngeal inflammation is often called acute catarrhal laryngitis. During life the mucous membrane is reddened and swollen, as demonstrated by means of the laryngoscope; but after death the congestive hyperæmia is less marked, and may even entirely disappear under the pressure of the abundant elastic fibres. Small ecchymoses are not infrequently observed. The inflammatory products consist of serum and pus mingled with an abundant secretion of mucus. The first product is chiefly mucus; later, this becomes opaque from admixture with a larger number of pus-cells, and in severe cases it becomes distinctly purulent. Ciliated epithelial cells do not appear in the expectoration, and they are not usually desquamated. The mucous glands are swollen, and through their orifices often little drops of muco-pus can be pressed. The mucous membrane and submucous tissue always contain some pus-cells. The amount of swelling of the loose submucous tissue varies in different cases. In severe cases it becomes so swollen from infiltration with serum and pus as to endanger life by interference with respiration. Sometimes erosions or even superficial ulcerations of the mucous membrane are formed. They either have their origin from ecchymoses or they are follicular and seated in the mucous glands.

CLINICAL HISTORY.—The symptoms of acute laryngitis are intelligible when the morbid changes are considered in connection with the small size of the rima glottidis and the unyielding walls of the larynx. Simple swelling of the mucous membrane may produce more or less distressing symptoms and danger due to narrowing of the aperture of the glottis; but with submucous infiltration added the obstruction is increased, and may prove fatal by suffocation. The obstructive effect of the morbid changes explains the symptomatology and danger. The chief element of danger is the submucous infiltration. This occurs in varying degrees, the severity of the local symptoms and the danger being in proportion to its amount.

The development of the disease may be preceded by subacute laryngitis or a common cold. The voice is at first hoarse, and, with the development of acute inflammation, is lost. The aphonia is accompanied with a stridulous cough and a small expectoration of glairy mucus, which may be streaked with blood. Febrile movement is more or less intense or it may be wanting. These symptoms may exist without notable difficulty of breathing, provided the changes be limited to the membrane and the swelling be not great.

With a greater amount of swelling, together with submucous infiltration, labored respiration and dyspnœa are added. The obstruction affects both inspiration and expiration, but especially the former, owing to the fact that the infiltration is greatest above the rima, where the areolar tissue is most abundant. The respiration is stridulous or noisy. The patient complains of a sense of constriction at the larynx, or feels as if the cavity were filled with a foreign substance. The latter symptom may be called *laryngeal tenesmus*. There is frequently more or less difficulty in deglutition. Liquids, by coming into contact with the inflamed epiglottis, excite cough and add to the distress. The labor of breathing and the dyspnœa are increased by the occurrence of spasm of the arytenoid muscles. The exacerbations of distress from obstruction are due chiefly to a spasmodic element. Paralysis of the abductor muscles may in some cases contribute to the obstruction. The obstruction will then be manifested more in the act of inspiration than in expiration.

In cases of acute laryngitis with great obstruction the suffering is intense. The face and eyes are swollen and congested, the countenance denotes anguish, and after a time lividity occurs. The distress is much increased during the exacerbations caused by spasm, and in the intervals of comparative ease the patient manifests drowsiness, but is unable to obtain refreshing sleep. With the muscular efforts to enlarge the aperture of the glottis, the larynx rises and falls in the acts of inspiration and expiration. There are frequent efforts to cough and expectorate, as if to get rid of an accumulation within the larynx. The cough may be dry and croup-like, but is oftener husky or abortive. The larynx is sensitive to pressure. Pitting over the pomum Adami has been observed. The respiratory acts are less frequent than in health. In a case which I have recorded they were 12 per minute.

With these symptoms in a large proportion of cases the disease ends fatally if tracheotomy be not resorted to. The mode of dying is by apnœa, and it may take place either gradually or suddenly in an exacerbation of unusual violence.

Assuming that other affections do not coexist, the symptoms, in addition to those which relate directly to the laryngeal obstruction, are incidental to fever—deficient oxygenation and decarbonization of the blood, together with the suffering which belongs to the disease. Slight delirium may occur toward the close of the disease if it be prolonged, but in general the mental faculties are unaffected.

PATHOLOGICAL CHARACTER.—In simple acute laryngitis the inflammation has no special characteristics. The severity and danger are not owing to the intensity or extent of the inflammation. The same amount of inflammation in other situations would constitute a trivial affection. The gravity is due to the fact that the usual results of inflammation in this situation obstruct the passage of air through the larynx, in consequence of the small size of the rima glottidis and the unyielding laryngeal walls. In children simple acute laryngitis gives rise to croupy cough and respiration, and hence this is one of the different affections formerly embraced under the name croup.

CAUSATION.—Acute laryngitis, with the symptoms stated in the Clinical History, in the adult is extremely rare.[1] It is less rare in infants and children than after adult age. Its infrequency is remarkable in view of the frequent occurrence of inflammation in the pharynx; and the fact exemplifies the principle of conservatism involved in the law by which inflammation does not tend to extend from one part to other parts, although in immediate proximity. I have seen a case in which the pharynx was laid open by a gunshot wound in the neck, so that the top of the larynx could be seen through the wound, and yet during the progress of recovery there was no evidence of the slightest laryngeal inflammation.

Occasionally, however, laryngitis is developed in connection with pharyngitis. This was observed in some cases of the so-called epidemic erysipelas, or "black tongue," which formerly prevailed in many parts of this country. I have known it to occur in connection with the pharyngeal affection in scarlatina. The laryngitis which, in a subacute form, is common in rubeola sometimes becomes acute. Acute inflammation may be excited in variola by the occurrence of the eruption in this situation. Under the several circumstances just named it is a secondary affection. It may occur as a primary affection after exposure to cold and wet, as in the case of Washington.

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[1] Some writers reckon as cases of acute laryngitis those in which the symptoms denote a less dangerous form of the disease than the one here considered under this heading. Of course the disease is less rare if the term acute be extended to cases of moderate severity.

It might be conjectured that excessive use of the voice would be likely to predispose to this affection, but clinical observation furnishes little proof that such is the fact. It may be produced traumatically by the inhalation of irritating vapors. Many cases have been reported in which young children have produced the disease by sucking boiling liquid and steam from the spout of a water- or tea-pot.

DIAGNOSIS.—The diagnostic characters of acute laryngeal inflammation are the loss of voice, and the husky, stridulous cough, in connection with the fever and other symptoms. That the labor of breathing and the dyspnœa are due to the obstruction of the larynx is shown by the sensations of the patient, the diminished frequency of the respiratory acts, and the absence of physical signs denoting pulmonary disease. In proportion as the aperture of the glottis is narrowed the respiratory murmur will be weakened, and this affords a better criterion of the amount of obstruction than the sufferings of the patient. The only difficulties in diagnosis relate to the discrimination of simple acute laryngitis from laryngitis with fibrinous exudation, from œdema of the glottis, and from spasm of the laryngeal muscles. In arriving at the conclusion that simple acute laryngitis exists, the affections just named are excluded by the absence of distinctive circumstances belonging to the clinical history of each. Suffice it to say here that the presence or absence of fibrinous exudation in cases of laryngitis is generally determinable; that œdema of the glottis may be ascertained; and that spasm without inflammation or with only subacute inflammation is wanting in characters which are essential to the diagnosis of acute laryngitis. If a satisfactory laryngoscopic examination can be made, the diagnosis is demonstratively established.

PROGNOSIS.—Acute laryngitis in the adult is a grave affection. The gravity, as already stated, depends in a great measure on the submucous infiltration. Cases without this contingency occur, and may end favorably without very alarming symptoms. The suffering in these cases arises measurably from spasm. If the constant obstruction—*i. e.* obstruction exclusive of that due to spasm—be sufficient to interfere greatly with respiration, the tendency is to a fatal result. A proportion of such cases will end fatally if life be not saved by timely surgical interference. The course of the disease in fatal cases is rapid. It has been known to prove fatal in seven hours. The duration rarely exceeds a week.

In young subjects this disease is less severe and less likely to prove fatal than in adults, notwithstanding the smaller size of the rima glottidis in early life, the difference being probably due to the fact that submucous transudation is less in children.

TREATMENT.—The treatment of acute laryngitis embraces general and local measures. The general measures are those designed to lessen the intensity of inflammation and to promote resolution and the absorption of serous transudation. The employment of bloodletting is to be guided by the same principles as in other inflammations, but the evils and danger of abstracting blood are not so great as in certain other inflammations—for example, lobar pneumonitis—because there is not the amount of morbid effects to be recovered from, and it is therefore not so important to spare the vital powers for the processes of restoration. The danger in acute laryngitis, it is to be borne in mind, is not from the extent or degree of the inflammation or from the lesions which it may induce *per se*, but it is incidental to the seat of the inflammation. And if a fatal result take place, the dying is by apnœa, not by asthenia. But in the use of bloodletting and other depressing measures this consideration must be kept in view: the patient's strength is not to be so far diminished as to incapacitate for the prolonged muscular exertions which the labor of breathing may require. Bloodletting should never be practised after the occurrence of lividity of the prolabia and face. Under these circumstances a fatal result would be hastened by abstracting blood.

The bowels should be moved efficiently by cathartics of the saline class. Nauseant or the direct cardiac sedatives are indicated as in other inflammations. Nauseant remedies should not be carried so far as to produce vomiting, which will add greatly to the distress and tend to aggravate the inflammation. If antimony be used, it should be given largely diluted, to avoid its local action on the throat.

Mercurialization may be advocated in this as in some other affections on the following ground: Although the antiphlogistic and sorbefacient powers of mercury have doubtless been heretofore greatly overestimated, it is by no means certain that it exerts no remedial influence in these directions. So long, therefore, as there is room to suppose that it may be useful in any degree, it should be employed in an affection like this, which frequently tends rapidly to destroy life by apnœa. The general depressing effect and other evils of mercurialization are of little moment, provided the remedy prove in any degree useful in its influence on the local affection. If mercurialization be determined on, it should be induced early and rapidly, either by large doses guarded with opium or by small doses repeated at short intervals, mercurial inunction being added.

Remedies to allay spasm and cough are indicated. The spasmodic element may be more or less prominent. Its degree of prominence is shown by the frequency and severity of the paroxysmal distress, whereas the amount of obstruction due to the swelling and infiltration is shown by the labor of breathing in the intervals between the paroxysms or exacerbations. Opium and antispasmodic remedies are important in proportion to the amount of suffering from spasm, but opiates should not be carried so far as to blunt the perception of the want of breath. If belladonna be found to meet the indication, it is to be preferred to opiates.

In the foregoing remarks on the general measures of treatment reference has been had especially to idiopathic or primary laryngitis. When the affection occurs as a complication of scarlatina, rubeola, or variola, and if it be associated with pulmonary or any other important disease, the same measures may not be admissible. This statement applies to bloodletting, mercurialization, and other depressing measures. Inflammation of the trachea frequently, if not generally, coexists, and bronchitis may be developed in accordance with the law respecting the extension of inflammation in the air-passages—namely, that it tends to travel downward and but rarely upward. Of course the general symptoms, febrile movement, etc. will be increased in proportion to the extent of mucous surface inflamed.

The local measures of treatment embrace fomentations or poultices applied to the neck, with a blister, perhaps, applied over the sternum; and the atmosphere of the apartment should be charged with steam or the vapor of hot water should be inhaled by means of a convenient apparatus. The former is vastly more effective, and is therefore to be preferred. The application to the throat of compresses dipped in ice-water and renewed at intervals of a few minutes is a measure which may be tried with safety, and if it afford relief it may be often repeated, being continued as long as it is a source of comfort to the patient. Early in the disease it may act favorably as an antiphlogistic, and afterward it may afford relief by diminishing spasm. According to Cohen, it is indicated when the obstruction is caused in a measure by paralysis of the abductors.

When the membrane, on laryngoscopic examination, shows much tumefaction above the vocal cords, Cohen advises scarification of the parts. If this measure be not practicable or if it prove ineffectual, tracheotomy is to be employed, provided the obstruction become so great as to render danger of death imminent. Patients should never be allowed to die from strangulation for want of timely surgical interference. If the danger be purely or chiefly from apnœa, this operation will prove the means of saving life. The important question is, When is it to be performed? It should not be delayed until permanent lividity occurs; that is, lividity due to the obstruction caused by swelling and infiltration irrespective of spasm. Persisting lividity denotes imminent danger, and impairs the probability of success from the operation. It is advisable not to delay either scarification or tracheotomy, or both, until lividity occurs, but to resort to surgical interference whenever the obstruction is sufficient to occasion sinking in of the soft parts and retraction of the lower part of the chest in inspiration.

After convalescence is established the recovery of the voice is slow, the pitch and quality remaining altered for some time.

The treatment of acute laryngitis in children, owing to its being a less severe and grave affection than in the adult, claims less vigorous treatment. Cases frequently, if not generally, do well with simple palliative measures. Emetics may be important in order to effect the removal of viscid mucus from the larynx. Death may be produced solely by obstruction due to the accumulation of mucus in this situation. It will hardly be supposed that the larynx may be so tolerant of obstruction from this source that, occurring in a healthy child, efficient efforts of coughing would not be excited and life be destroyed by slow apnœa; yet a case exemplifying the fact has fallen under my observation.

Subacute Laryngitis.

Subacute inflammation of the larynx is of frequent occurrence, either as preceding the development of bronchitis, or the inflammation not extending to the bronchial tubes. It characterizes certain cases of the affection commonly known as "a cold." The inflammation is of a low grade of intensity, and is not accompanied by submucous effusion. There is either hoarseness or aphonia, with cough and an expectoration at first transparent and viscid, and afterward opaque, thick, and loose. There is no obstruction to respiration. Laryngeal spasm in the adult is rarely excited. There is little or no fever.

It is important only as involving some liability to an increase in the intensity of the inflammation or the development of the acute affection; but the liability to this is small. The measures of treatment are those indicated in cases of bronchitis or a "cold."

Subacute laryngitis exists in a certain proportion of the cases formerly included under the name croup. The croup phenomena are due to spasm, the laryngitis being an unimportant element. This variety of so-called croup is unattended with danger, however violent may be the symptoms referable to spasm.

Chronic Laryngitis—Laryngeal Ulcers.

Chronic laryngitis occurs much more frequently than the acute form; it occurs, therefore, often without having been preceded by acute laryngitis.

Acute laryngitis, in fact, if it do not destroy life, ends in recovery without eventuating in the chronic affection, and, on the other hand, in chronic laryngitis the inflammation rarely becomes acute.

In chronic inflammation the hyperæmia is less intense than in acute laryngitis. The mucous membrane has a grayish color, and often presents to view dilated veins. A mucous or purulent secretion coats its surface, which is often granular from enlargement of the mucous glands. The vocal cords lose their smooth, glistening appearance, and are grayish or opaque. The flat epithelium covering them becomes thickened, and often in other portions of the larynx the normal cylindrical epithelium is replaced by the pavement variety. By development of new connective tissue the mucous membrane and submucous tissue often become thickened, and occasionally papillomatous growths project into the cavity of the larynx. Instead of being hypertrophied, the mucous membrane in some cases is atrophied. Ulcerations more or less deep and extensive are frequent in chronic laryngitis due to specific causes.

In the majority of cases chronic inflammation in this situation occurs in connection with either syphilis or pulmonary phthisis. The localization of syphilis in this part is rare, so that the existence of chronic laryngitis constitutes presumptive evidence of the existence of phthisis. In cases of pulmonary phthisis with chronic laryngitis it was formerly supposed that the latter precedes the former, and that the disease extends from the larynx to the lungs. The term *laryngeal phthisis* was used to distinguish the affection in these cases. Improvement in the means of detecting a tuberculous deposit in the lungs, however, has led to a knowledge of the fact that the laryngitis in nearly all these cases is secondary, the pulmonary disease being first developed. The laryngeal ulcers which complicate pulmonary phthisis are most frequently seated on the posterior surface of the larynx, just above the commissure of the vocal cords. In many cases a single small ulcer in this situation is found. This may, however, extend in depth and surface, and others may likewise appear. The vocal cords may be nearly destroyed by phthisical ulceration, and one or more small ulcers are often found on the epiglottis. The smaller ulcers are usually round, and the larger ones have festooned borders. The edges are thickened. The ulcers appear to be always due to the breaking down of tuberculous products. Tubercle bacilli are found in the walls of the ulcer and in the secretion from its surface.

While phthisical ulcerations most frequently begin just above the vocal cords, syphilitic affections are usually first developed in the upper portion of the larynx. *Syphilitic laryngitis* in general follows syphilitic disease of the pharynx and base of the tongue. In the later stages of syphilis laryngeal ulcerations, often of the most destructive character if not properly treated, may be developed. These usually begin on the epiglottis and extend downward. They may attack the vocal cords, and even extend into the trachea. They are accompanied usually with much induration and thickening of the submucous tissues. When the ulcers heal, sometimes deforming cicatrices are formed, which may lead, especially on or about the vocal cords, to dangerous and even fatal stenosis.

Inflammation of the tissues immediately surrounding the laryngeal cartilages is called *laryngeal perichondritis*. Suppurative perichondritis is most frequently the result of syphilitic or phthisical ulcers in the larynx. It occurs as a very rare complication in infectious diseases of an asthenic type. The non-vascular cartilage suffers in its nutrition, and often becomes partly or wholly necrotic, so that loose pieces of cartilage, usually calcified or ossified, are found bathed in pus. If the collection of pus open into the laryngeal cavity, the dead pieces of cartilage may be coughed out. A necrosed fragment of cartilage has been known to become lodged in the glottis and to cause death by suffocation. Sometimes the perichondrial abscess opens externally, and through the opening necrosed cartilage may be discharged.

The most characteristic of the SYMPTOMS of chronic laryngitis relate to the voice. It becomes either hoarse, husky, or stridulous, and it may be lost; that is, there is either difficulty of speech (dysphonia) or its extinction (aphonia). If aphonia exist, the patient speaks in a husky whisper. The extent to which the voice is affected is not a criterion of the extent to which the vocal organs are damaged by ulceration. Slight ulcerations and thickening may lead to great huskiness, and even extinction of the voice. If notable hoarseness exist, more or less destruction of the vocal cords, enlarging the rima glottidis, may be suspected.

As regards cough, its prominence as a symptom varies in different cases. It is modified like the voice; that is, it is either hoarse, husky, or stridulous. The expectoration also varies. It may be slight or more or less abundant. Its characters may be those of mucus, muco-purulent matter, or pus. Not infrequently it presents bloody streaks, and it may be fetid. The bloody streaks and pus point to ulceration; the fetor points to caries of the cartilages. Cough and expectoration, however, it is to be borne in mind, are measurably due to the tuberculous affection of the lungs with which the laryngitis is generally associated. Some cases of tuberculous laryngitis are characterized by violent paroxysms of cough resembling those of whooping cough, and vomiting may occur during the paroxysms.

A marked difference in different cases relates to deglutition. In some cases the act of swallowing is attended with no inconvenience, but in a certain proportion of cases it is a source of great distress. The passage of food and drink causes pain, excites spasms of the glottis, thus occasioning distressing dyspnœa, and frequently liquids are returned through the nostrils. Patients are obliged to confine themselves to the blandest articles of diet, and even these are sometimes ingested with so much difficulty that death is hastened by innutrition. The difference in different cases depends on the situation of the ulcerations, these extending sometimes above the vocal cords and upon the epiglottis, where the ulcerated surface comes in contact with the food and drink in the act of swallowing, and sometimes being sufficiently below the top of the larynx to be secure from this source of irritation.

The DIAGNOSIS of chronic laryngitis is easy; the morbid characters of the voice at once indicate the affection. Its connection with pulmonary phthisis is to be determined by a physical exploration of the chest and examination of the sputa, together with an examination into the previous history and symptoms. If pulmonary disease be excluded, the syphilitic source of the affection is to be investigated. The affection may be regarded as idiopathic in cases in which both syphilis and phthisis can be excluded; but, as already stated, such cases are rare.

The PROGNOSIS in cases of chronic laryngitis will of course have reference to its pathological associations. Its connection with phthisis does not render the latter disease more rapidly progressive; on the contrary, the progress of the tuberculous affection of the lungs is, as a rule, more slow with than without this complication, provided it do not interfere with alimentation. Thus associated, however, the laryngitis is rarely recovered from. The inflammation and ulcerations usually continue, but if not, the voice remains permanently more or less impaired. Considerable improvement may take place in cases of tuberculous laryngitis, although the affection continue. Chronic laryngitis dependent on syphilis offers a better prospect of recovery. The voice, however, after recovery will be very likely to remain permanently affected.

In my experience cases of tuberculous laryngitis have not presented notable embarrassment of breathing from obstruction. In not a few of the cases, however, of syphilitic laryngitis which I have seen there has been sufficient stenosis to render respiration more or less labored, and in some cases obstruction threatened life by apnœa. In a number of cases which have come under my observation at Bellevue Hospital the operation of tracheotomy has been resorted to, and, apparently, the lives of patients thereby saved. It has sometimes been resorted to more than once in the same case.

The TREATMENT of chronic laryngitis, as regards general measures, will have reference to its pathological relations. In the great majority of cases the treatment must be that indicated by the coexistence of pulmonary phthisis. If the affection be traceable to syphilis, the antisyphilitic remedies are called for—namely, mercury and the iodide of potassium.

As regards local measures, counter-irritation over the neck by means of the croton oil or small blisters is sometimes useful, even in cases of phthisis. The local treatment, however, relates especially to applications to the affected part: that is, within the larynx. The injection of medicated liquids and the insufflation of remedies in the form of a dry powder are objectionable, owing to the difficulty of limiting the application to the diseased parts and to the cough and spasm which they are liable to excite. A more satisfactory method is the introduction of a sponge attached to a probang properly curved. Owing to the enlargement of the glottis which takes place in the act of inspiration, the introduction must be made during the act of inspiration. During the act, as is well known, foreign bodies of large size are sometimes drawn with the inspired breath into the air-passages. The nature and seat of lesions within the larynx may be ascertained by means of the laryngoscope, and the knowledge thus obtained affords aid in judging as to the propriety of topical applications and in showing the particular points at which they are to be made.

The topical remedy most frequently applied by means of the sponge is the nitrate of silver. A strong solution is commonly used—namely, from one to two scruples to the ounce of distilled water. The sponge, moistened by this solution, is carried between and below the vocal cords. Spasm of the glottis occurs, and the liquid, being expressed from the sponge, comes into contact with the whole of the interior surface of the larynx. This application is sometimes beneficial, affording immediate and marked relief. It may then be repeated from time to time. In many cases, however, no relief is afforded, and in these cases after a fair trial it is useless to persevere in it. In the cases in which the laryngitis interferes with deglutition, relief may sometimes be afforded by narcotics and probably by codeia. The topical application of the hydrochlorate of cocaine has recently been found serviceable.

Tracheotomy is to be resorted to in cases of syphilitic laryngitis whenever obstruction endangers life. The propriety of this operation as a means of promoting recovery has been advocated. For this purpose the opening is to be maintained for an indefinite period. The advantage consists in the arrest of the respiratory movements of the laryngeal muscles, which is a result of breathing through the trachea.[1] This measure has also been advocated in cases of laryngitis associated with pulmonary phthisis, the objects being complete rest of the larynx and the prevention of the passage of the matter of expectoration over the ulcerated surfaces. It has proved a useful measure in some reported cases.[1] An objection to it in cases of phthisis is the obstacle which a tube in the trachea offers to free expectoration. If found to be useful, its usefulness will probably be limited to the cases in which the laryngeal affection involves dysphagia and in this way interferes with alimentation.

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[1] Vide article by Thomas Bryant, in the *Trans. Clin. Soc. of London*, vol. i., 1868.

Inflammations may occur in that part of the larynx situated below the vocal cords. Inflammation in this situation is called *laryngitis hypoglottica.* The inflammation may be confined to this region. Acute subglottic laryngitis is often attended by great swelling of the submucous tissue in consequence of infiltration with serum and pus-cells. The affection is a grave one on account of the obstruction to respiration. Usually, both acts of respiration are difficult. The diagnosis is readily made by means of the laryngoscope, which reveals the situation of the swelling to be just below the true vocal cords. Tracheotomy is frequently indicated by the severity of the symptoms.

Chronic inflammation of the mucous and submucous tissues of this part of the larynx is characterized by a new growth of connective tissue (*laryngitis hypoglottica hypertrophica*). The affection is rare and of a peculiar nature, being in some cases apparently dependent upon the disease called rhinoscleroma.

Laryngitis with Fibrinous Exudation (Membranous Croup).

In Part First of this work (p. 37) the anatomical characteristics of the so-called croupous, diphtheritic, and pseudo-diphtheritic inflammations have been briefly mentioned. It is important to distinguish between the anatomical signification of these terms and the clinical application of the names croup and diphtheria. In croup we may have diphtheritic as well as croupous inflammation, the difference depending chiefly upon the seat of inflammation. Decided as are the differences between membranous or true croup and diphtheria as regards etiology and clinical history, essential points of difference in respect of their pathological anatomy have not as yet been established. In both diseases there is a fibrinous exudation. In croup the larynx is by preference attacked, in diphtheria the pharynx and sometimes the nasal passages; but croup is usually accompanied with fibrinous exudation in the pharynx, and diphtheria frequently with pseudo-membranous laryngitis. In both diseases the fibrinous exudation, when seated in the trachea or the lower portion of the larynx, is usually of the so-called croupous variety, and, when seated in the pharynx or fauces, of the diphtheritic or of the pseudo-diphtheritic variety. By so-called true or membranous croup, however, is understood a local disease characterized by fibrinous inflammation of the upper air-passages; by the name diphtheria, an infectious disease with, in certain cases, a similar localization of the fibrinous exudation. In this article reference is had only to the affection heretofore known as true croup. Diphtheria is more appropriately considered under general diseases than under diseases affecting the respiratory system.

The term croup is of Scotch origin, and signifies strangulation. As already stated, it has been used in a comprehensive sense to signify a variety of affections in children characterized by a peculiar shrill, barking cough called the croupal cough, and by difficulty in breathing. These symptoms may accompany in children simple acute or subacute laryngitis, and also spasm of the glottis without laryngeal inflammation. To such cases the name *false croup* has been applied in distinction from *true croup*, the latter term being employed only for the pseudo-membranous variety of inflammation.

The extent of the fibrinous exudation varies in different cases. The exudation, in the form of a false membrane, is found in the larynx and trachea. It is also present, as a rule, in the pharynx, on the tonsils, soft palate, base of tongue, or epiglottis. The larger bronchi are often involved, and in rare instances those of small size. The larynx may be primarily affected, or the false membrane may appear first in either the pharynx or the trachea. The false membranes are whitish or grayish, or brownish-red from admixture with blood. They vary greatly in extent, thickness, and consistence. On the mucous membrane of the pharynx or fauces the fibrinous exudation appears usually in patches. In the larynx the favorite seat of the exudation is the vocal cords, which are often uniformly coated, and sometimes so thickly that the glottis is nearly occluded. The false membrane may be expectorated in the form of shreds or of tubular casts of the larger bronchi, or rarely as solid cylinders from the small bronchi. The membrane, when recent, is of tolerably firm consistence; it may be found on autopsy, however, as a soft, pultaceous mass coating the mucous membrane. In the trachea and bronchi it can, as a rule, be readily stripped off from the subjacent mucous membrane; in the larynx it can also often be removed without tearing the mucous membrane; but it is usually closely adherent on the epiglottis and true vocal cords; in the pharynx and on the soft palate also it is intimately connected with the mucous membrane. In the bronchi, trachea, and lower portion of the larynx the fibrinous exudation is of the so-called croupous variety—that is, the false membrane consists chiefly of fibrillated fibrin holding in its meshes pus-cells, red blood-corpuscles, and sometimes epithelial cells; it lies directly upon the basement-membrane in most places, the epithelium being absent; the mucous membrane itself is swollen, hyperæmic, and contains a variable number of emigrated white blood-corpuscles, and sometimes ecchymoses, but no fibrinous exudation. On the epiglottis and in the other parts of the throat the exudation is often of the pseudo-diphtheritic variety—that is, the false membrane cannot be detached without loss of substance from the subjacent mucous membrane—but there is no fibrinous material in the mucous membrane itself. The exudation, however, may be diphtheritic; that is, the fibrinous substance is present not only upon the surface, but also in the tissues of the mucous membrane. As has been explained in Part First, the fibrinous material in the diphtheritic and pseudo-diphtheritic forms of inflammation consists in part of fibrillated fibrin and in part of epithelial, pus-, and connective-tissue cells which have undergone the peculiar metamorphosis called coagulation necrosis. During the course of the disease the pseudomembranes may be repeatedly exfoliated and new ones formed in their place.

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[1] Vide article by Dr. Beverley Robinson, *Am. Journ. of Med. Sciences*, April, 1879.

Both rod-shaped and spherical bacteria are found in the false membranes, but it is not proven that they are concerned in the causation of the disease.

The lungs after death from fibrinous laryngitis often present spots of atelectasis, also an emphysematous condition, especially of the upper lobes, hyperæmia, and sometimes lobular pneumonitis, rarely the lobar form.

CLINICAL HISTORY.—The symptomatic phenomena in this variety of inflammation at the outset are the same as in simple laryngitis. The local and general symptoms may not be as marked as in cases of simple acute laryngitis. The disease is often supposed to be nothing more than a common cold. There is more or less cough, and the cough is hoarse or barking. Hoarseness or huskiness of the voice exists, and moderate fever. The symptoms denoting gravity of disease arise from obstruction due to the exudation, in connection with swelling of the glottis and the occurrence of either spasm or paralysis of the laryngeal muscles. One, two, or three days may elapse before the disturbance of respiration is sufficient to create any apprehension. Not infrequently the general symptoms do not prevent the child from being up and engaging in play even after a certain amount of obstruction has taken place. The obstruction due to the exudation and swelling is shown by labored breathing in both acts and by dilatation of the nostrils. These evidences may be apparent before the obstruction is sufficient to occasion suffering from dyspnœa. The additional obstruction due to laryngeal spasm is shown by the dyspnœa, which occurs in paroxysms or exacerbations. Spasm may be more or less prominent as an element of this form of disease; but, as a rule, it is far less prominent than in the affections heretofore known as false croup. Hence, of all the varieties of so-called croup, this, in its development and early progress, is the one least likely to excite alarm.

As the disease advances the obstruction is increased. The labor of breathing becomes greater, and is attended with manifestations of distressing dyspnœa. The current of inspired air is insufficient for the free expansion of the lungs, as denoted by contraction of the lower part of the chest, together with sinking in of the soft parts above the clavicles and sternum in the act of inspiration. The cough becomes stridulous and abortive. The voice or cry is reduced to a husky whisper. The face is congested, and an expression of anxiety and distress is marked. More or less fever continues. The sufferings are increased at variable intervals in proportion as the spasmodic element is prominent. If life be prolonged, the cough at length becomes loose, and portions of the false membrane are expectorated from time to time. The obstruction may then gradually decrease and recovery take place. If the disease prove fatal, the mode of dying is by slow apnœa; but not infrequently the suffering from dyspnœa diminishes toward the close of life, and the labor of breathing may be so far lessened as to excite delusive hopes of improvement. Convulsions occasionally occur toward the close of life.

A cause of obstruction in certain cases is paralysis of the posterior crico-arytenoid muscles. As a result of paralysis of these muscles, dilatation of the glottis does not take place in inspiration, and obstruction during the inspiratory act occurs as after section of the recurrent laryngeal nerves. Immobility of the glottis is observed by laryngoscopic examination. In so far as the obstruction is due to this cause, it will be manifested in the inspiratory act. This act may be accompanied by a laryngeal sound suggestive of a sucking inward of the glottis. Another important incidental circumstance increases the interference with respiration—namely, the accumulation of muco-pus in the trachea and bronchial tubes, the presence of the false membrane in the larynx, and the feeble expiratory efforts preventing expectoration. The danger from accumulation below the larynx is of course incidental to the cases in which bronchitis coexists.

Pathological Character.—Fibrinous exudation occurs exceptionally in inflammation of a mucous membrane. It denotes a peculiar kind of inflammation. This peculiarity of the inflammation belongs to it from the beginning. It is not evidence of intensity of the inflammation; on the contrary, the inflammation in this variety of laryngitis may be less intense than in the simple acute variety. Acute simple laryngitis has no tendency to eventuate in fibrinous exudation; and in the other affections of the larynx which have been commonly embraced under the name croup the form of disease under consideration, distinguished as true or membranous croup, rarely supervenes.

Causation.—This form of disease is generally met with in young children, but rarely in infantile life. The age, in the great majority of cases, is between two and seven years. The disease, however, may occur at any period from birth to the age of two years, and at any period after seven years. Boys are oftener affected than girls. It occurs oftener in some situations than in others; a cold, humid, changeable climate apparently contributes to its occurrence. Cases occur oftener in the spring and autumnal months than at other portions of the year. Happily, it is everywhere a rare disease, excluding, it will be borne in mind, its occurrence as a complication of diphtheria. It may occur as a primary or a secondary affection. Exclusive of its occurrence in diphtheria, it is sometimes developed in connection with the affection of the pharynx in scarlatina. It occasionally follows measles. The inflammation and exudation, as already stated, generally involve more or less of the pharynx, the point of departure being in some cases the larynx, and in other cases the pharynx, oftener the latter.

DIAGNOSIS.—Considering the great fatality of this disease, the diagnosis is of great importance with reference to the prognosis and treatment. It is to be discriminated from acute simple laryngitis, from subacute laryngitis with spasm, and from a purely spasmodic laryngeal affection which is presently to be considered. In each of these affections laryngeal spasm is more or less involved, and the character of cough and respiration distinguished as croupal is chiefly attributable to that element. The spasmodic element is more likely to be prominent, especially at the outset, in the other affections just named than in this affection; in other words, in this, the so-called true croup, the croupal phenomena are less marked than in either of the forms of the so-called false croup. The insidious development of this disease, which alone of the affections formerly embraced under the name croup is attended with danger, renders it liable to be overlooked until the disease has progressed so far as to give rise to serious obstruction.

As contrasted with the other affections just named, the slow, insidious development is one of the points involved in the differential diagnosis. In the so-called catarrhal and the spasmodic variety of croup the croupal phenomena occur suddenly and violently, the attack almost always being in the night. When such an attack occurs without having been preceded by cough, febrile movement, and alteration of the voice, it is almost certain that the affection is not true croup. Alteration of the voice is an important diagnostic feature. This will distinguish membranous croup from a purely spasmodic affection, but not from simple laryngitis, either acute or chronic; the voice, however, is affected in a more marked degree in laryngitis with fibrinous exudation.

The presence of fibrinous exudation about the epiglottis, and more or less within the pharynx, is a point of great importance in the differential diagnosis. With reference to this point the observations of Ware are valuable. In his analysis of cases of the several varieties of croup, out of 33 cases of true croup—*i. e.* laryngitis with exudation—a pharyngeal exudation was observed in all save a single case. On the other hand, of 45 cases of the affections included under the head of false croup, an exudation within the pharynx was sought for and found to be wanting in every case. Of 19 cases observed by Meigs with respect to this point, in all but 3 an exudation occurred in the pharynx either before or after the development of the laryngitis. In view of these facts, the presence of an exudation within the pharynx, taken in connection with the symptoms which denote laryngitis, renders it extremely probable that exudation exists within the larynx. It is to be borne in mind, however, that the pharyngeal exudation is sometimes wanting, and that its absence in the larynx cannot be inferred from its non-existence in the pharynx with as much positiveness as its presence in the larynx is to be inferred from its existence in the pharynx.

In order to render the diagnosis positive, if the co-operation of the patient can be obtained, the laryngoscope should be employed. The existence of the disease is of course demonstrated if the intralaryngeal space can be inspected.

In the progress of the disease, if it do not destroy life, the discovery of patches of false membrane in the matters expectorated should be considered as the only reliable test of the correctness of the diagnosis. The non-observance of this test has led not infrequently to cases of simple laryngitis being regarded as cases of laryngitis with fibrinous exudation successfully treated.

With capillary bronchitis this affection need never be confounded. In capillary bronchitis the respirations are frequent, panting, and gasping; in laryngitis with exudation the acts are labored and difficult, and, as a rule, not notably frequent. The embarrassed, noisy breathing, especially during sleep, in cases of enlargement of the tonsils may at first suggest the affection under consideration, but it is readily excluded by the absence of the croupal cough and of hoarseness. Retro-pharyngeal abscess is to be excluded.

PROGNOSIS.—The tendency of this disease is to a fatal result. Of 22 cases analyzed by Ware, 19 proved fatal. Even if the affection be limited to the larynx and uncomplicated, it will destroy life in the great majority of cases if patients be not saved by surgical interference. The danger is of course increased if the affection extend to the bronchial tubes, and in proportion as it extends toward the smaller tubes. Not infrequently broncho-pneumonia exists as a complication, and this of course augments the danger. The duration of the disease in fatal cases is usually from four to six days. In a small proportion of cases it kills in less than four days, or life is prolonged beyond six days. If recovery take place, convalescence is reached after a gradual improvement, and is generally slow, the voice remaining more or less affected for a considerable period.

TREATMENT.—Under measures of treatment heretofore relied upon this disease proved fatal in the vast majority of cases. The treatment was based on the supposition that the exudation depends on the intensity of the inflammation, and with a view to reduce this intensity the measures known as antiphlogistic were actively employed. They have been employed sufficiently to show that they are not successful, and if they do not do good they can hardly fail to do harm. With a view to the proper objects of treatment, it is to be considered that cases are rarely seen prior to the occurrence of exudation, and that the prevention of exudation, therefore, is not the end to which therapeutical measures are to be directed. It is also to be considered that, exudation having taken place, recovery is effected by the separation of the false membrane, and that its separation takes place as a result of a suppurative process beneath it, this process requiring several days. These facts being considered, the rational objects of treatment are to prolong life until the separation of the false membrane can be effected, and to promote the process by which this is effected.

Depletion by bloodletting or other means is not indicated. By reducing the strength of the patient it conflicts with the first object—namely, the prolongation of life. On the contrary, the strength is to be supported, as far as practicable, by concentrated nourishment and the judicious use of alcoholics. Emetics, which have heretofore been used excessively in this disease, are to be employed with circumspection in order not to conflict with the first object. They have been given, and frequently repeated, in order to relieve spasm, and it is undoubtedly true that for a short period after their operation the breathing is comparatively easy; but this end may be attained by other measures less objectionable on the score of depressing the powers of life. Another purpose of emetics is to aid in detaching the false membrane. But it is useless to give emetics for this purpose until the exudation has been loosened by the process of suppuration. Emetics, however, are not to be entirely withheld. In children they compensate for the want of voluntary efforts of expectoration. They may aid in the expulsion of the false membrane when it becomes detached. A fact stated under another head in this chapter is not to be lost sight of—namely, the tolerance of the larynx in young children of an accumulation of mucus sufficient to occasion fatal obstruction. Emetics, then, may be given from time to time, but not too frequently, and the emetic substances used should be those which excite prompt and efficient vomiting without producing prolonged nausea and depression. This will exclude antimony. Ipecacuanha, powdered alum, turpeth mineral, the sulphate of copper, and the sulphate of zinc are among the articles which may be selected. Of these articles, the turpeth mineral acts promptly and efficiently, without much depression or nausea. This remedy was recommended as an efficient emetic in croup forty years ago by Dr. Hubbard of Maine. It is to be borne in mind that the accumulation of muco-pus in the trachea and bronchial tubes in certain cases furnishes an indication for the use of emetics.

Anodyne remedies are indicated to allay spasm. Opium, however, is to be given with great circumspection. Belladonna and other of the narcotics, if they be found to answer, are to be preferred. Mercury is admissible on the ground stated under the head of Acute Simple Laryngitis; that is, the objections to its use are not to be taken into account in so dangerous a disease as this, so long as there is ground to suppose that it will do any good.

In the early part of the disease cold applications to the neck may be tried, as in the treatment of acute simple laryngitis. These, as in cases of the latter form of laryngeal inflammation, have reference, *first*, to lessening the intensity of the inflammation—that is, to an antiphlogistic effect; and *second*, to a sedative effect as regards spasm of the laryngeal muscles. To employ cold efficiently, compresses dipped in ice-water may be applied and removed at intervals of a few moments. The continuance of these applications and their repetition are to be determined by the relief which they appear to afford. They should not be persisted in if the patient resolutely resist them or if they do not seem to give relief.

Topical measures of an opposite character have reference to the second object—namely, promotion of the process by which the false membrane is separated. The neck may be enveloped in a poultice or the water-dressing for the same reason that these are deemed useful in phlegmonous inflammation—namely, to hasten suppuration. But the most efficient topical application is the inhalation of steam, and the best method is to charge the atmosphere of the room with as much of it as possible. In a room at a temperature of 80° or 85°, filled with steam, patients are free from spasm and breathe with comparative ease. I have observed cases in which patients passed safely through the disease, the inhalation of steam being the chief measure relied upon.

From the fact that pseudo-membranous exudations are rapidly dissolved in lime-water, the inhalation of spray from lime-water has been resorted to as a means of removing the false membrane from the larynx and trachea, and cases in which this treatment was successful have been reported. It has proved successful in cases under my observation. Pepsin has been employed for this end. Trypsin, one of the digestive ferments of the pancreas, has recently been used with success.[1] Dr. E. M. Moore recommends the insufflation of the bicarbonate of soda for this object. Slaking quicklime is an

[1] Vide *Diseases of Children*, by J. Lewis Smith, 6th ed., p. 581.

effective mode of securing this object, and at the same time of furnishing a warm, pleasant vapor. Breathing pure oxygen from time to time, and diffusing it in abundance in the atmosphere of the room, afford marked relief of dyspnœa.

The topical application of a strong solution of the nitrate of silver has been advocated. The introduction of the sponge probang into the larynx of the child is difficult, owing to the small size of the glottis and frequently the want of co-operation on the part of the patient, yet it may be effected. I have witnessed recovery in a case in which this was the chief measure employed. The applications were made twice daily. When the false membrane is loosened, the introduction of the sponge may be useful mechanically, detaching the membrane and pushing it below the glottis, thus leading to its expectoration.

Lastly, the propriety of tracheotomy is to be considered. For statistics bearing on this subject the reader is referred to treatises on the diseases of children. The propriety of the operation hinges, not on the number of cases in which it has proved successful, but on the answer to the question whether lives are saved by it. Now, it cannot be doubted that patients with this disease have been snatched from impending death by opening the trachea. It is, therefore, not only justifiable, but the practitioner is bound, if possible, not to allow patients to die from suffocation for want of this operation. Of course, the duty of resorting to it is the more imperative the greater the probability of its success; and a fair examination of statistics will show that not a very small proportion of those who would otherwise die may be rescued by it.

The success of tracheotomy depends much on its timely performance. Its failure in many cases is attributable to its having been too long deferred, but it has proved successful repeatedly as a *dernier ressort* under the most unpromising circumstances. As soon as the persisting obstruction—that is, the obstruction due to the exudation, associated perhaps with paralysis of the dilating muscles of the glottis—compromises respiration sufficiently to render it probable that the patient's life will not be prolonged until the separation of the false membrane can take place, the operation is called for. Its success doubtless depends much on the manner of its performance and the subsequent management. (For information on these points the reader is referred to surgical works.)

Sir William Jenner has pointed out an objection to a premature performance of tracheotomy—namely, the effect of the operation on the efficiency of cough. In an efficient act of coughing first a deep inspiration is taken and the glottis closes; then by means of forcible expiratory efforts the current of expired air, dislodging from the bronchial tubes their contents, carries them into the trachea, and, the glottis suddenly opening, expectoration is effected. Now, with an opening in the trachea an efficient act of coughing cannot take place; hence there is a greater liability to accumulation in the bronchial tubes than if tracheotomy had not been performed. The danger from this effect of the operation thus relates to the bronchial tubes in the cases in which bronchitis is associated with the laryngitis.[1]

If the operation prove unsuccessful, it diminishes greatly the suffering from obstruction, and is fully justifiable with a view to euthanasia.

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[1] Tubage of the larynx, proposed by Bouchut as a substitute for tracheotomy, has been successfully employed by Dr. Joseph O'Dwyer of New York. (Vide *Diseases of Children*, by J. Lewis Smith, 6th ed., p. 589. Vide, also, article by E. Fletcher Ingals, in *Journal of the Am. Med. Association*, Feb., 1886.)

Œdema of the Glottis.

This name denotes a serous or, more frequently, a sero-purulent effusion in the submucous areolar tissue above the vocal cords. In the majority of cases, therefore, œdema of the glottis is an inflammatory affection. The effusion is situated chiefly in the ary-epiglottic folds, about the ventricle of Morgagni and the base of the epiglottis. On the vocal cords the mucous membrane is too intimately adherent to the subjacent dense fibrous tissue to admit of submucous effusion. The ary-epiglottic folds and adjacent tissue are swollen, soft, and project from each side as two semi-transparent, gelatinous tumors, as large as, or even larger than, a pigeon's egg, which, lying upon the glottis, are sucked inward with the inspiratory act, and thus occasion obstruction. The current of air in expiration separates and pushes the tumors upward, so that in cases of œdema without laryngitis there is little or no obstruction in the expiratory act. Upon cutting into the œdematous tissue a serous or sero-purulent liquid escapes. It is rare for pus-corpuscles to be so abundant that the effusion is distinctly creamy and purulent. In most cases of Bright's disease complicated with œdema glottidis the effusion is thin and watery, with comparatively few blood-corpuscles. The œdema usually exists on both sides, but it is sometimes unilateral. Œdema may also occur in the lax submucous tissue just below the vocal cords (subglottic œdema).

When œdema of the glottis is due to inflammation of the mucous membrane in proximity to the seat of the effusion, it is analogous to the subcutaneous œdema which accompanies inflammation of the skin in situations where the areolar tissue beneath is abundant and lax, as, for example, around the eyes and on the penis—the collateral œdema of Virchow. This so-called collateral œdema is, however, always of inflammatory origin. (See Part I. pp. 34 and 36.)

As causes of œdema glottidis may be mentioned laryngitis, pharyngitis, tonsillitis, retro-pharyngeal and cervical abscesses, injuries to the neck (particularly cut-throat wounds), swallowing irritant poisons, such as ammonia, sulphuric acid, etc., and erysipelatous inflammation of the neck. It also occurs in connection with certain general diseases, especially those complicated with laryngitis, as typhus and typhoid fevers, smallpox, septicæmia, phthisis, heart disease, and both the acute and chronic forms of Bright's disease. It has been attributed to the action of cold. It probably never occurs as an idiopathic affection. It is important to add that it may occur as the first and only dropsical affection connected with disease of the kidneys. Its gravity and the necessity of prompt and efficient interference render it vastly important to appreciate the pathological condition and to recognize its existence.

The occurrence of the œdema may be sudden, and life be destroyed in a few hours or even minutes.[1] In a case occurring in connection with mild pharyngitis the suffering from obstruction was developed during the night, and the patient, leaving his apartment to seek for aid, fell upon the floor, and died before any assistance could be rendered. In a case in which it occurred in connection with tonsillitis the patient was left to sleep in an apartment by himself, there being no difficulty of breathing, and in the night-time a relative in an adjoining room was awakened by his noisy breathing. He was found to be unconscious, and, although medical aid was promptly called and the trachea opened without delay, the operation was too late to prevent a fatal result.

Difficulty of breathing, due to obstruction at the glottis, is a prominent symptom. This is associated with a sense of a foreign substance at the seat of the obstruction, and a strong disposition to hawk and swallow. The diagnostic point relating to the difficulty of breathing is, the inspiratory act is alone or chiefly obstructed, expiration being free. The inspiration is arrested before it is completed, being, as it were, suddenly cut short, and both the inspiration and expiration are accompanied by marked stridor. If laryngitis do not coexist, the voice is devoid of hoarseness or huskiness. This will serve at once to exclude laryngitis. I have met with a case, ending fatally, in which there was no hoarseness or huskiness of the voice and no appearance of laryngitis after death. The diagnosis may be made positive by means of the touch. With the forefinger introduced into the mouth, carried to the base of the tongue and applied below the epiglottis, the œdematous tumors may be distinctly felt.

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[1] A case is reported in *Guy's Hospital Reports*, April, 1855, which terminated fatally within five minutes after difficulty from obstruction was experienced.

If the embarrassment of breathing be not too great to permit a satisfactory laryngoscopic examination, this will render the diagnosis positive. The tumors may sometimes be seen by depressing the tongue, without the aid of the laryngoscope.

Statistics have heretofore shown a large fatality. Bayle found that in 17 cases only 1 recovery took place. Of 40 cases collected by Valleix, 31 were fatal. Of 168 cases collected by Sestier, 127 died. With a correct appreciation of the pathological condition, a prompt recognition of it, and timely surgical interference, many, if not most, patients may be saved. It is fair to attribute the large proportion of fatal cases heretofore to an imperfect knowledge of the affection, or delay in the diagnosis, or want of promptness in resorting to efficient interference.

The affection kills by apnœa. The object of treatment is to prevent suffocation, and if the obstruction be sufficient to occasion much labor of breathing, lividity, etc., no time is to be lost in efforts to obtain relief by medication. Either the larynx or trachea must be opened, or the method of scarification, as practised by Dr. Gurdon Buck, must be resorted to. Buck's method consists in introducing a curved bistoury, properly guarded, into the mouth, carrying it below the epiglottis guided by the left index finger, and scarifying on both sides, so as to give exit to the effused liquid. The scarification may be repeated if required.[1]

The difficulty in the way of the performance of this operation is greater than might be supposed. The increase of suffocation caused by introducing the fingers into the mouth, the movements of the larynx incident to the labored breathing, and the acts of vomiting which are excited, render it not easy to deliberately scarify the œdematous parts. In a case under my observation the result was unfortunate. The tumors were not sufficiently scarified, but wounds were made which led to hemorrhage and the flow of blood into the air-tubes. Tracheotomy was subsequently performed, but too late to save the patient. This case is referred to in order to enforce the importance of being prepared to meet with more difficulty than might be anticipated. Without assuming to adjudicate in a matter which is purely surgical, I am led to think that the simpler as well as the more effectual operation is to open the larynx or trachea. Niemeyer stated that in some cases the finger-nail will suffice to open the œdematous tumors. Strong pressure with the finger has succeeded.

A case at Bellevue Hospital exemplified the importance of opening the trachea before the obstruction threatens immediate death. A patient was suffering considerably from difficulty of breathing, but, as was decided, not sufficiently to require laryngotomy. The house physician, however, made every preparation to perform the operation at an instant's notice. Suddenly the difficulty of breathing increased, and the physician was in a few moments at the bedside, but too late to save the patient. The liability to a very rapid increase of obstruction proving quickly fatal is to be considered in deciding upon the propriety of opening the larynx or trachea. The operation may be advisable, as a precautionary measure, even if life be not at the moment threatened, when the practitioner cannot remain with or near the patient until danger is passed. In the event of a physician being summoned to a case in which life is momentarily in danger, and when appropriate surgical instruments are not at hand, the larynx or trachea should be opened at once, without dissection, with a pocket-knife.

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[1] Vide *Trans. Am. Med. Association*, vols. i. and iv. Several cases successfully treated by this method are reported by Dr. Buck. Cohen (*op. cit.*) has used with success, for scarification, a common gum-lancet.

Œdema of the glottis rarely occurs in infancy or childhood, the reason probably being that the areolar tissue at the situation where the œdema occurs is less abundant and loose in young subjects than after puberty. The period of life in which it is most liable to occur is between the ages of twenty and forty. It occurs much oftener in men than in women.

Œdema in very rare instances occurs below the vocal cords. This is distinguished as subglottic œdema. Its occurrence in this situation is not determinable by the touch. The diagnosis can only be made by means of the laryngoscope. Scarification is not practicable. Tracheotomy is indicated under the same circumstances as in cases of obstruction from œdema above the cords. The obstruction calls for this operation, although the diagnosis of subglottic œdema may not have been made.

Spasm of the Glottis.

Spasm of the muscles closing the glottis is an important element in most of the affections of the larynx which have been considered. To this element are attributable the croupal cough and the paroxysms or exacerbations of dyspnœa in simple acute and subacute laryngitis in children and in laryngitis with fibrinous exudation. But spasm occurs also as a purely functional affection; that is, irrespective of inflammation or lesion within the larynx. It is an element of several of the functional affections of the nervous system (neuroses) to be hereafter considered—namely, hysteria, epilepsy, epileptoid convulsions, rabies, and tetanus. Occurring irrespective of these connections, it may be reckoned among the functional affections of the larynx.

Spasm occurs in young children, affecting the respiration without inducing cough. This is the pathological condition when the infants are said to have fits of "holding the breath." Respiration is arrested until the face becomes congested and livid, and when the spasm relaxes the inspiration may be sonorous, as in pertussis. In some children these paroxysms occur more or less frequently. They are sometimes accompanied by carpo-pedal spasm. Convulsions may occur. Death may take place in a prolonged paroxysm or in consequence of repeated paroxysms in rapid succession. I have known an instance of this kind. The affection is commonly known as *laryngismus stridulus*. From a fancied connection with enlargement of the thymus gland persisting after birth, it has been called *thymic asthma*.

This affection is purely neurotic. It involves a peculiar susceptibility to spasm in the laryngeal muscles. This susceptibility may be incident to a certain period, as during weaning or dentition, but in some cases it appears to be an idiosyncrasy of infantile life. When the predisposition exists, paroxysms are liable to be excited by various causes acting physically and mentally. This susceptibility is sometimes observed in different children of the same family—a fact in confirmation of its innateness.

During a paroxysm it is customary to employ means to produce a strong impression on the surface, as slapping the back or sprinkling cold water on the face. A ready and effective mode of arresting a paroxysm is to introduce a finger into the throat. The treatment otherwise involves the removal of all local causes of irritation, such as the distension of the gums and the retention of fecal matter, avoidance of causes inducing mental excitement, and measures to invigorate the system.

Spasm of the glottis occurring in children over two years of age, accompanied by laryngeal irritation and cough, is a frequent affection, known as *spasmodic croup*. A child is suddenly attacked during the night, having perhaps gone to bed apparently in perfect health. The breathing is labored and sonorous; the cough presents in a marked degree the shrill, ringing, croupal character. Great alarm is produced, and the physician is sent for in urgent haste. The surface of the body is cool; the pulse is small and perhaps not accelerated; the voice or cry is not hoarse nor husky as in largyngitis; the affection, in short, is purely spasmodic. It is unattended by danger. When the violent symptoms are relieved respiration is found to be unobstructed. There is no liability to its eventuation in so-called true croup; that is, to laryngitis with fibrinous exudation. It is to be discriminated from the latter affection. The points involved in this discrimination are as follows: The abruptness of the attack, which is not preceded by symptoms denoting laryngitis; the violence of the attack, in this respect differing from so-called true croup; the absence of hoarseness or huskiness of the voice, of febrile movement, and of fibrinous exudation in the pharynx; and, finally, the speedy and complete relief. The paroxysms may recur for two or more successive nights.

The TREATMENT consists of a mild emetic if there be reason to suppose that the stomach is overloaded, a hot foot-bath, and warm fomentations to the neck. These measures are uniformly successful. The treatment of these cases is sometimes needlessly active, under the impression that a serious affection is threatened; and practitioners are apt to congratulate themselves and the friends of the patient on their success in preventing true croup.

Spasm of the glottis may occur as a functional affection in the adult. It is occasionally incidental to hysteria, and may simulate, as regards some of the symptoms, laryngitis or œdema of the glottis. The diagnostic points are as follows: The coexistence of hysterical phenomena; the absence of huskiness or hoarseness of the voice; intermittency of the difficulty of breathing; the speedy relief obtained by anodyne remedies; and the sudden development of the affection. I have been consulted in several cases in which spasm of the glottis occurred in male adults without any evidence of disease within the larynx or of intrathoracic disease.

The measures of treatment are anodyne or antispasmodic remedies for immediate relief, and afterward those indicated by the general condition and those employed in analogous nervous affections.

Spasm of the glottis may be occasioned by irritation of the recurrent laryngeal nerve from the pressure of a tumor or other causes. It is one of the symptoms of aortic aneurism, and, occurring in a person over forty years of age, it should always excite a suspicion of aneurism. I have reported a case of aortic aneurism in which the life of the patient was destroyed by frequently recurring paroxysms of laryngeal spasm.[1]

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[1] *American Medical Times*, 1865. Another similar case has fallen under my observation. It is questionable whether in these cases the attacks of laryngeal obstruction be not due to paralysis of the abductor muscles. This inquiry will be referred to in connection with Aortic Aneurism.

Nervous Cough.

The term "nervous cough" is used to designate a neuropathic disorder characterized by a dry, peculiar cough occurring without laryngitis, bronchitis, or any pulmonary affection. It may be noticed with propriety among laryngeal affections, inasmuch as the distinctive character of the cough relates to the larynx. In most of the cases which I have seen the cough has had a peculiar barking tone, and the pitch has been low, showing that the glottis was dilated at the instant of coughing. In some cases, however, the tone is shrill and the quality of sound croupal, showing spasm of the glottis. In a case recently under observation the cough consisted of a single, short, hoarse bark, often repeated several times in a minute. The cough is somtimes in paroxysms, having a resemblance to whooping cough. The peculiar sound of the cough, together with its frequent recurrence and sometimes its violence, renders it distressing to those whose sympathies are excited, and annoying to others. Cohen, who observed the movements of the glottis in a well-marked case by means of the laryngoscope, states that the vocal cords came together with force, and suddenly separated at the instant of the cough.

This disorder rarely occurs in males. It is often associated with more or less of the symptoms embraced under the name hysteria. The cough has been called *tussis hysterica*, and it is usually included by medical writers among the diversified hysterical manifestations. It occurs chiefly in chlorotic girls. I have known it to occur before the age of puberty. It is likely to persist for a considerable period.

The DIAGNOSIS is to be based on the peculiarity of the cough, its dryness—except that the acts of coughing, if frequent and violent, may occasion some mucous expectoration—and the negative result of careful physical explorations of the chest. The diagnosis is strengthened, on the one hand, by the coexistence of anæmia and of hysterical phenomena, and, on the other hand, by the absence of rational symptoms pointing to phthisis. The laryngoscope should be employed in order to render positive the exclusion of other laryngeal affections.

The indications for TREATMENT relate to anæmia if it exist, and to other morbid conditions affecting the nervous system. Tonic remedies, a nutritious diet, out-door life, and, in short, invigorating measures, are indicated. In addition to chalybeates may be mentioned quinia, small doses of strychnia, and the preparations of zinc as appropriate remedies. Hygienic measures, however, are of the first importance. A change of climate is sometimes advisable if other measures fail. A sea-voyage proved effective in a case under my observation after a long persistence of the cough in spite of various measures of treatment. As palliatives for the cough antispasmodic remedies may be prescribed, but opiates are not advisable.

This is one of the affections which may be induced by involuntary imitation. Cohen states that within his knowledge a school for girls was suspended in consequence of the large number of cases.

Nervous Aphonia—Paralysis of Laryngeal Muscles.

Loss of voice, or aphonia, is incidental to the varieties of laryngitis which have been considered. But it occurs when not dependent on either inflammation or lesions within the larynx; in other words, as a functional affection. It is then known as nervous aphonia. The loss of voice is due to paralysis affecting the nerve of phonation—namely, the spinal accessory. The affection may be limited to the voluntary movements of the laryngeal muscles, the reflex movements involved in respiration being unaffected.

Functional aphonia is met with not infrequently in women, and is often, but not always, associated with phenomena embraced under the name hysteria. As a purely neurotic affection—that is, not dependent on any lesion of the nervous system—it is extremely rare in the other sex. An obvious distinction is between loss of voice and loss of speech, or aphasia. Aphonia may occur from pressure on the recurrent laryngeal nerve by an aneurismal or other tumor, and the loss of voice should direct attention to this as a possible or probable cause.

The DIAGNOSIS of nervous aphonia may be made by attention to the character of the whispered voice. The patient speaks in a pure, soft whisper, without effort. On the contrary, if the aphonia be due to laryngitis, the whisper is stridulous or husky and labored. Moreover, in aphonia due to laryngitis there is more or less cough and expectoration, symptoms generally absent in nervous aphonia. As the question is usually to decide between nervous aphonia and chronic laryngitis, and as the latter affection is generally associated with pulmonary phthisis, the absence of the symptoms and signs of the latter disease will serve to confirm the diagnosis. Examination with the laryngoscope will establish the diagnosis by showing that the larynx is free from lesions; and the absence or the incompleteness of movements of the glottis when an effort to speak is made may be ascertained by inspection.

Inspection with the laryngoscope shows that paralysis of laryngeal muscles may be bilateral or unilateral, and that the different muscles—namely, the adductors and abductors, the tensors and the laxators—may be affected separately. Unilateral paralysis of course points to some cause affecting the pneumogastric nerve on one side only, and it is especially suggestive of an aneurismal tumor pressing upon one of the recurrent laryngeal nerves. Bilateral paralysis, however, may be caused by pressure upon the recurrent laryngeal or the vagus nerve on one side. A case under my observation exemplified the correctness of this statement.

The localization of paralysis in the different muscles of the larynx, singly or severally, is to be made by laryngoscopic examination. As a rule, in functional aphonia the adductor muscles are affected. The patient is unable by an effort of the will to approximate the vocal cords sufficiently for phonation. In paralysis of the abductor muscles the voice is more or less affected. This form of paralysis will claim separate notice. (For an account of the laryngoscopic appearances, the symptoms, etc. of paralyses affecting the different muscles separately, the reader is referred to works which treat of diseases of the larynx *in extenso*.[1])

If aphonia be purely neurotic—in other words, involving no lesion of the nervous centres or pressure on either the par vagum or the recurrent laryngeal nerve—recovery may be expected after a duration of the affection varying much in different cases. The voice is sometimes restored instantaneously and unexpectedly. I have met with cases in which repeated attacks had occurred.

TREATMENT in these cases should be directed to the general condition. Tonic remedies and invigorating hygienic measures are indicated. Moderate counter-irritation may be useful. Electrization of the laryngeal muscles has been found signally useful. Both the induced and the direct current are used with success. The recovery of the voice sometimes follows a single application or even a single electrical shock; but in some cases the treatment to be successful must be continued for several weeks. In some of the cases in which electricity at once effects a cure there is reason to think that the success is due to a moral influence. Other measures often effective are evidently operations through the mind. Cohen states that he has repeatedly effected a cure by the use of the laryngoscopic mirror, the patient being led to suppose that its introduction was a curative procedure. The late Professor Ware sometimes succeeded by telling patients that they could recover the voice by an energetic act of the will. A violent mental emotion has sometimes produced sudden aphonia, and, aphonia existing, has sometimes occasioned a sudden restoration of the voice.

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[1] Cohen, *op. cit.*, treats fully of laryngeal paralyses.

Whenever there is a partial restoration of the voice complete recovery may be expected by systematic efforts of speech, in the same way as the control of the will over other paralyzed muscles is effected by means of persistent voluntary exercise. Beginning with the vowels most easily spoken, the practice should extend to those more difficult of utterance, then to consonants and words of one syllable, and finally to sentences. After recovery the exercise of the voice within the limit of fatigue is to be advised.

Aphonia is sometimes feigned. The malingerers are women actuated by a desire to excite sympathy or to become objects of interest. I have met with two cases in which the patients declared their inability even to whisper. One of these patients communicated by signs and by writing, and the other by silent, inarticulate movements of the lips. A curious feature in the first of these cases was that during a second visit, accompanied by her husband, while I was in conversation with him, she began to speak in a low voice and took part in the conversation during the remainder of the interview. No comment was made on this sudden recovery of voice either by herself, her husband, or by me. I met her casually some years afterward; there was then no impairment of voice, and she made no reference to her aphonia except to ask if I recollected her consulting me. The subsequent history in the other case is unknown. That the inability to speak is in some instances a self-delusion is probable. This may have been the explanation in the cases just noticed.

Paralysis affecting the abductor muscles has important distinctive features when contrasted with the paralysis of the adductors. The effect of bilateral paralysis of the abductors is the approximation of the vocal cords, giving rise to more or less obstruction of respiration. The obstruction is manifested especially in inspiration. The condition is analogous to that observed after the physiological experiment of dividing both recurrent laryngeal nerves. The vibration of the cords with the current of inspired air causes stridor. There is dysphonia, but not complete loss of voice. Dyspnœa exists in proportion to the obstruction. There is danger of death from suffocation, especially if, as may happen, spasm of the adductors be added to the paralysis of the abductors.

In view of the indication for prompt treatment whenever the obstruction is sufficient to involve danger, the diagnosis is extremely important. The laryngoscope renders it positive, and it cannot be made so otherwise than by inspection of the larynx. In marked cases the vocal cords are in close proximity, the aperture being reduced to a narrow slit, and no dilatation takes place in the act of inspiration. Without a laryngoscopic examination the noisy, stridulous, inspiratory sound might suggest either laryngitis or œdema of the glottis.

Tracheotomy is indicated whenever the obstruction involves immediate danger. The indication derives great force from the fact that with an opening into the trachea the affection is tolerated indefinitely. Cohen cites a case within his cognizance in which a tracheal tube had been worn for more than twelve years, the patient a judge who was able to continue his duties on the bench. He also cites three cases in which life was lost from a refusal to submit to the operation of tracheotomy. If, from the occurrence of spasm of the adductors, paroxysms be liable to occur threatening life, there can be no doubt concerning the propriety of tracheotomy as a prophylactic measure, provided the patients be not under constant observation.

Cases in which spasm of the adductors is not an element may not require surgical interference. A case came under my observation at my college-clinic in which recovery took place without an operation, the treatment consisting chiefly of quietude and asafœtida. Dr. George M. Lefferts has reported two cases of recovery without tracheotomy under the use of the iodide of potassium, the patients having had syphilis.[1]

The PROGNOSIS as regards recovery from the paralysis will depend on the causative conditions. If the paralysis depend on lesions, either centric or peripheral, which are permanent, recovery is not to be expected. Danger of death from obstruction is permanently averted by a tracheal opening. If, however, the paralysis be functional, or if it depend on syphilitic changes, as in the cases reported by Lefferts, the prognosis as regards recovery is favorable.

If the paralysis be unilateral and remain so, there is little or no danger from obstruction. The voice is weak and stridulous, simulating chronic laryngitis. In a patient forty years of age aneurism of the aorta should always be suspected, and should be sought for, especially in the site of the descending portion of the arch; but the paralysis may depend on other causes, either centric or peripheral, generally the latter. An enlarged bronchial gland, carcinoma of the œsophagus, and a mediastinal tumor may cause unilateral paralysis by pressure on the recurrent laryngeal nerve.

Bilateral paralysis of the abductor muscles is extremely rare. The number of reported cases is small; but it is probable that the pathological character of the affection was not infrequently overlooked prior to the date of laryngoscopy.

Morbid Growths.

The most common tumor within the larynx is papilloma. It takes its origin by preference from that portion of the laryngeal mucous membrane which is covered with flat epithelium. It may have a broad base or may be connected with the mucous membrane by a distinct pedicle. It consists essentially of hypertrophied and newly-formed papillæ covered with flat epithelium. Small papillomatous growths are not infrequent attendants upon tuberculous and syphilitic laryngeal ulcers and chronic laryngitis. Papillomatous growths, however, may occur without inflammation. Other important forms of new growths are the laryngeal polypi, which may be fibrous, myxomatous, or glandular. Fibroma, myxoma, and adenoma do not, however, necessarily assume a polypoid form in the larynx. Carcinoma of the larynx, when primary, is usually of the flat-celled variety, and takes its origin from that portion of the larynx lined with squamous epithelium, especially from the true vocal cords. Cancer seated primarily in the larynx is rare, the disease, in the cases in which this part becomes affected by it, generally originating in the œsophagus or in the neck exterior to the larynx. Syphilitic and tuberculous new formations have already been referred to. A detailed consideration of these and other rarer forms of laryngeal tumors must be sought in works treating specially of diseases of the throat.

Morbid growths occasion inconvenience, suffering, and death by producing obstruction. Surgical interference becomes necessary when the obstruction is sufficient to endanger life. A tube may be worn in the trachea for an indefinite period. In a case in which a permanent artificial opening becomes advisable, it may be a question whether a large fistulous orifice might not be established, rendering the tracheal tube unnecessary. As bearing on this question, the following case is of interest: A patient, aged about forty, admitted into one of my wards in Bellevue Hospital and suffering from the effects of a debauch, had a fistulous orifice in the anterior portion of the trachea large enough to admit the end of the forefinger. This fistula followed a wound received in a fracas, and had existed for seventeen years. It occasioned little or no inconvenience in respiration. There were no symptoms of inflammation or irritation of the trachea or bronchial tubes, and he was entirely free from pulmonary disease; the only inconvenience which it occasioned was in speaking. In order to direct a current of air through the glottis sufficient for the production of the voice, he was accustomed to approximate the chin to the sternum, and in this way he was able to close the fistula.

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[1] *New York Med. Journal*, Dec., 1878.

The laryngoscope has proved to be of great practical value in determining the existence, situation, size, and character of morbid growths within the larynx, and in facilitating surgical operations for their removal through the laryngeal aperture. Cohen states that prior to laryngoscopy probably not more than seventy cases of morbid growths within the larynx were to be found in medical literature, whereas within the last twenty years thousands of such cases have been reported.

The larynx has been extirpated for the removal of carcinoma, and an artificial larynx has been substituted. The first operation of this kind was performed successfully by Billroth. (Surgical works are to be consulted for this and other operative procedures for tumors of the larynx.)

SECTION SECOND.

DISEASES AFFECTING THE CIRCULATORY SYSTEM.

CHAPTER I.

PERICARDITIS.

Introductory Remarks.—Acute Pericarditis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Chronic Pericarditis.—Pneumo-Pericarditis.

DISEASES affecting the circulatory system are seated either in the blood-vessels or in the central organ of the circulation—the heart. There is a group of affections existing independent of the organs of the circulatory system and involving certain morbid changes of the blood. The affections belonging in this group are simple or benign anæmia, chlorosis, leucocythæmia, pseudo-leucocythæmia, lymphatic anæmia, or Hodgkin's disease, pernicious anæmia, and to these may be added Addison's disease. These affections proceed from defect or disturbance referable to the blood-producing organs. They will be considered in a separate section (Section III.), under the heading Diseases affecting the Hæmatopoietic System. Morbid conditions of the blood, not recognized as individual affections, being common to different diseases, are considered in Part First of this work. Diseases of the vessels, entering, as they do, much more largely into maladies which belong to the surgeon than into those which fall within the province of the physician, are fully considered by surgical writers. Of the diseases of the arteries, aneurisms situated within the chest and abdomen will alone claim attention in this treatise. Thoracic aneurisms will be noticed in this section, and those situated within the abdomen will be referred to in connection with abdominal tumors in Section IV. This section will be devoted chiefly to diseases affecting the heart.

The diseases of the heart are conveniently arranged in the following groups: 1. Inflammatory affections; 2. Structural lesions; 3. Functional disorders. Inflammation affecting the heart gives rise to different diseases according to the structure in which it is seated. Inflammation of the serous membrane investing the heart (the pericardium) gives rise to the disease called *pericarditis*. Inflammation of the membrane lining the cavities of the heart (the endocardium) is another disease, called *endocarditis*. Inflammation of the substance of the heart (the myocardium) is distinguished as *myocarditis*. These three inflammatory diseases claim separate consideration.

Acute Pericarditis.

Pericarditis occurs as an acute and as a chronic affection. Acute pericarditis will be now considered, and afterward the chronic form of the disease.

ANATOMICAL CHARACTERS.—Acute pericarditis may be either diffuse or circumscribed. When circumscribed, the inflammatory products are usually deposited at the base of the heart, near the origin of the great vessels. Diffuse pericarditis is the usual form. The morbid appearances resulting from acute inflammation in this situation are essentially the same as in inflammation of other serous membranes—for example, the pleura. The serous membrane is at first reddened in consequence of congestion and small ecchymoses. The inflammatory exudation consists of fibrin, serum, pus-cells, and red blood-corpuscles. In the majority of cases it is sero-fibrinous, the pus-cells not being sufficient in number to render opaque the fluid part of the exudation. The exudation may consist almost wholly of fibrin, with very little serum. This has been called dry pericarditis. Under exceptional circumstances the red blood-corpuscles are present so abundantly as to give to the serum a reddish or brownish coloration. This constitutes hemorrhagic pericarditis. It occurs especially in connection with tuberculosis or cancer of the pericardium and also as a complication of scorbutus. Tuberculous pericarditis may occur, although very rarely, without tuberculous deposits in any other part of the body. In rare instances the exudation is purulent. This may occur in pyæmia and in consequence of the extension of a purulent inflammation in the neighborhood, as in cases of suppurative pleuritis, hepatic abscess, etc. In acute diffuse pericarditis the fibrin is deposited in the form of a layer or a series of layers on the visceral and on the parietal surface of the membrane. It is a peculiarity of this fibrinous pseudo-membrane that its free surface has a rough, shaggy appearance, due to the projection of a number of filaments and villous processes (*cor villosum*, hairy heart). This peculiar disposition is attributable to the constant churning action of the heart.

The quantity of liquid effusion in different cases of pericarditis varies greatly, amounting in some cases to a few ounces only, and in other cases to a pint or more. It is usually of a yellowish color, and contains flocculi of fibrin. Wandering cells or pus-cells are present in the fibrinous layer and in the serum, also in varying numbers in the tissue of the serous membrane itself. Whether these have any other origin than emigration from the blood is still a matter of dispute.

If recovery take place, the serum is absorbed; the fibrin undergoes a molecular disintegration, and is absorbed. At the same time new connective tissue in the form of adhesions is developed. The process of their formation is the same as in pleurisy (p. 117). The pericardial sac may be completely obliterated by the firm adhesion of its two layers, or it may be partially obliterated. The adhesions may also develop in the form of threads and bands stretching between the pericardial surfaces.

Purulent pericarditis runs a less favorable course. Several pints of pus may accumulate in the pericardial sac. It sometimes happens that the parietal layer of the pericardium ulcerates, and the pus may even escape externally by a fistulous opening. It is possible for only small accumulations of pus to be absorbed.

The muscular substance of the heart suffers more or less in its nutrition in all cases of pericarditis. The most frequent change is parenchymatous and fatty degeneration of the muscular fibres situated just beneath the pericardium. The fibrous thickening of the visceral pericardial layer which follows pericarditis is sometimes accompanied by the growth of fibrous tissue into the subjacent muscular substance, and a consequent atrophy of the muscular fibres in this situation.

The most convenient mode of dividing acute diffuse pericarditis into stages is the same as that adopted in pleuritis. The first stage extends to the time when liquid effusion takes place to an extent sufficient to be appreciated by the physical signs. The second stage, or the stage of effusion, continues until the liquid is absorbed. The third stage extends from the absorption of the liquid to the recovery, and this is generally the stage of convalescence.

CLINICAL HISTORY.—The symptoms are almost invariably made up, to a greater or less extent, of those arising from coexisting affections; for in the great majority of cases the disease is associated with articular rheumatism, Bright's disease of the kidneys, or pleuritis with or without lobar pneumonitis. Under these circumstances it is not always easy to determine to what extent certain symptomatic phenomena are due to the cardiac disease. As a rule, the development of the inflammation is attended with more or less pain, which in some cases is acute and lancinating, like the pain in pleuritis, and increased by forced breathing, so that the disease has not infrequently been supposed to be inflammation of the pleura. The intensity of the pain varies much in different cases; it is not always a prominent symptom, and it is sometimes slight or even wanting. The pain may be increased by acts of deglutition, by eructations, and it sometimes is analogous to that in angina pectoris. The facies is that of suffering and anxiety. A dry, irritable, suppressed cough is generally present. Tenderness over the præcordia is more or less marked, and pressure in the epigastrium upward in a direction toward the heart sometimes occasions acute pain. The pain produced by a deep inspiration may cause the patient to shorten this act, and hence, by way of compensation, the number of respirations per minute is increased. The alæ nasi may dilate in inspiration. Patients sometimes manifest suffering from an indefinite sense of distress, without acute, localized pain, and in women hysterical phenomena may be associated with the development of the disease. The action of the heart is increased, amounting sometimes to palpitation. The pulse is more or less accelerated, quick, and vibratory. Pyrexia and its concomitants—namely, anorexia, debility, etc.—are present. These are symptoms belonging to the first stage.

The duration of the first stage is usually short. An appreciable amount of effusion may take place in a few hours, and it is rarely delayed beyond one or two days. The acuteness of the local symptoms—namely, pain and soreness —then diminishes, and if the amount of liquid effused be sufficient to fill or distend the pericardial sac, symptoms are added which proceed from the pressure of the liquid upon the heart. These symptoms are—a sense of oppression referable to the præcordia; a tendency to syncope on exertion, which leads the patient to refrain as much as possible from movements of the body; feebleness and irregularity of the pulse, with a notable increase on emotional excitement or any muscular effort; dyspnœa, sometimes amounting to orthopnœa if the accumulation of liquid be large and rapid; feebleness of the voice or even aphonia; and dysphagia, in some cases produced by pressure of the distended sac on the recurrent laryngeal nerves and on the œsophagus. Cyanosis and turgescence of the cervical veins are sometimes marked, due probably to pressure on the auricles and the venæ cavæ. Venous pulse on the neck is sometimes observed. Vomiting is an occasional symptom. Singultus may be produced by pressure on the phrenic or branches of the pneumogastric nerve. Some cases are characterized by remarkable disturbance of the nervous system. Mental aberration, consisting in obstinate taciturnity and indifference, alternating with paroxysms of maniacal excitement under the influence of delusions which excite terror, is occasionally observed. Coma, chorea, and tetanic convulsions have been known to occur. These symptoms are likely to mask the cardiac disease and lead the practitioner to suspect disease of the brain. Autopsical examinations show that in these cases neither inflammation nor any appreciable lesion of the nervous system exists, and hence the phenomena are to be referred to functional disturbance of the nervous system.

The severity of the disease, as denoted by the symptoms, corresponds to the intensity of the inflammation and the amount of effusion. If the inflammation be slight or moderate and the quantity of effused liquid small, the disease may run its course without any symptoms denoting gravity, and the symptoms may not even point to the existence of any affection within the chest. In other cases it is one of the most distressing and formidable of diseases. The symptoms due to compression of course diminish as the liquid is absorbed. The absorption sometimes goes on very rapidly, and in this respect different cases present great variations. If, instead of being absorbed, the liquid continue to accumulate and life be not rapidly destroyed, the pericardial sac may become greatly dilated and the affection become chronic. The rapidity and completeness of recovery after absorption will depend on the amount of inflammatory exudation. This may be too abundant to be absorbed, and the disease may end fatally after continuing for a greater or less period in a chronic form. In cases which go on favorably toward recovery the action of the heart is feeble and easily excited during the stage of convalescence.

Pathological Character.—Acute pericarditis does not differ essentially in character from other serous inflammations. The same series of processes which take place in the serous inflammation considered in the preceding section (Pleuritis) occur in this inflammation—namely, fibrinous exudation and liquid effusion, agglutination followed by permanent adhesion of the free surfaces of the membrane, and sometimes the formation of pus. Suppurative pericarditis, however, occurs in a much smaller proportion of cases than suppurative pleuritis, or empyema. As in cases of pleuritis and other serous inflammations, so in different cases of pericarditis, both the actual and relative amount of fibrin and serum differ greatly. In some cases the quantity of fibrin is small and the effusion of liquid abundant; in other cases the fibrin is abundant with but little liquid; and, again, in other cases both fibrin and liquid are either abundant or small. Judging from the number of cases which I have seen, *dry* pericarditis—that is, the presence of fibrin without serous effusion—is not extremely rare.

Causation.—Acute pericarditis may be produced traumatically by perforating wounds of the chest or contusions. In one of the cases which I have observed it was produced by a wound inflicted by a one-tined fork. Walshe refers to a case in which the pericardial sac was perforated in the juggler's trick of swallowing a sword, and fatal pericarditis was induced. In the museum of Bellevue Hospital is a remarkable specimen in which a set of false teeth is contained within the pericardial sac. The teeth were swallowed during alcoholic coma, and, lodging in the lower part of the œsophagus, produced ulceration through this tube and into the pericardium, giving rise to fatal pericarditis.

Exclusive of its traumatic origin, the disease is almost always secondary, and it is developed in the course of acute articular rheumatism oftener than in connection with any other affection. Occurring in the course of rheumatism, it is distinguished as rheumatic pericarditis. It would appear, from statistical data, to occur in cases of acute rheumatism in a proportion of about 1 to 6. Sibson, out of 326 recorded hospital cases under his care, found evidence of pericarditis in 63.[1] Without having noted sufficient data for analysis, I am sure that this proportion is considerably higher than in my experience. The comparative infrequency of this complication in the cases which have come under my observation I am disposed to attribute to the prompt and efficient employment of the alkaline treatment which for many years has been in vogue in this country. Generally, the development is in the early part of rheumatism, after a greater or less number of the joints have been affected, but occasionally the pericarditis precedes the articular affection. When it follows the affection of the joints, it is not due, as was formerly supposed, to a metastasis of the articular affection, but it proceeds from the same morbid condition which underlies the latter. Rheumatic pericarditis is generally associated with endocarditis. The latter existed in 54 of the 63 cases analyzed by Sibson.

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[1] *Reynolds's System of Medicine.*

Next in frequency to its development in the course of rheumatism, it occurs in connection with either acute or chronic disease of the kidneys. It is not very infrequent in cases of chronic Bright's disease; but it is a rare concomitant of the renal affection which follows scarlatina and in other cases of acute diffuse nephritis. It occurs most frequently with the small kidney of chronic Bright's disease. Sibson's statistics show that it occurs in as many as 8 or 9 per cent. of all fatal cases of Bright's disease.

Pyæmia, scorbutus, the continued and eruptive fevers, erysipelas, and influenza are other affections in connection with which this disease has been observed to occur. It is a not very infrequent concomitant of pleuritis and of pneumonitis, affecting either the right or left lung—oftener the latter. Pericarditis may be secondary to peritonitis and to inflammation in the mediastinum.

I have met with a few cases in which a careful examination failed to discover any pre-existing or coexisting disease, nor was any other affection developed so long as the patients remained under observation, the periods extending only to the time when they had recovered sufficiently to leave the hospital. The extreme infrequency of such cases renders it extremely probable that whenever pericarditis exists some one of the affections just named will be found associated with it.

DIAGNOSIS.—The events belonging to the clinical history of acute pericarditis are not sufficiently diagnostic for the disease to be discriminated, by the symptoms alone, from pleuritis, pneumonitis, or pleurodynia. Its coexistence with the two former of these affections could not be ascertained, and its existence would surely be overlooked in the cases in which the symptoms are but little marked. The diagnosis must rest on physical signs. The presence of inflammatory exudation gives rise to an adventitious sound or murmur caused by the rubbing together of the pericardial surfaces in the movements of the heart. This sign is proof of the existence of pericarditis, and, as the exudation takes place usually within a few hours after the beginning of inflammation, the sign becomes available for diagnosis at an early period of the disease. The sign is called the *pericardial* or *cardiac-friction murmur*, or sometimes, in contradistinction from the murmur produced by blood-currents within the heart, an *exocardial murmur*.

The friction murmur is to be distinguished from the murmur produced within the heart, called *endocardial* or *bellows murmur*, by attention to the following points: It is double; that is, consisting of two adventitious sounds for each beat of the heart, one produced by the systolic and the other by the diastolic cardiac movements. The character of the murmur denotes friction; that is, it is rubbing, grazing, or grating, and at a later period in the disease it may be creaking, like the sound of new leather. It is limited to the præcordia, not propagated beyond the borders of the heart, and it may be limited to a portion of the præcordial space. As regards its intensity, it differs much in different cases, and it may vary in the same case during an examination, the variations being dependent on the force of the heart's action. It has not the same fixed connection with the normal heart-sounds as have the endocardial murmurs; in other words, it does not observe the same regularity in rhythm as the latter. This is a striking point of distinction. It is intensified, and sometimes changed in character, by firm pressure with the stethoscope. In connection with a rough friction murmur, fremitus is sometimes felt by the hand applied to the præcordia.

A friction murmur, if ever wanting in the first stage of pericarditis, is present so generally that we are warranted in basing an exclusion of the disease on its absence, provided sufficient time have elapsed for inflammatory exudation, and limiting the question of diagnosis to the first stage. The murmur is almost pathognomonic taken in connection with the symptoms and history; yet there is one source of fallacy—namely, the production of an intrapleural friction murmur by the movements of the heart in cases of pleuritis. A friction murmur accompanying the systolic and diastolic movements of the heart, continuing while the patient's breathing is voluntarily suspended, may be thus produced irrespective of pericarditis. I have met with several examples, the absence of pericarditis, notwithstanding a well-marked murmur with cardiac rhythm, having been determined by examination after death. This is called a *cardiac-pleural friction murmur.* It is to be suspected, if pleuritis be known to exist with or without pneumonitis, when the murmur is limited to the border of the heart, the intensity of the murmur varying much more than the ordinary cardiac murmur during the periods of examination, it being heard in some cases only at the end of inspiration, and generally being affected more or less by the respiratory movements.

In the second stage the occurrence of liquid effusion and its amount are determined by percussion. Abnormal dulness or flatness exists over an increased area in the præcordial region. The pericardial sac, if filled with liquid, forms a pyriform tumor, the boundaries of which are readily defined by percussion. The base is situated below the level of the apex-beat in health; the left border extends beyond the left nipple to a greater or less extent, and the right border between the right margin of the sternum and the right nipple; the apex is near the sternal notch. The situation, size, and shape of the area of dulness or flatness point to pericardial effusion. If the pericardial sac be partially filled with liquid, the area of dulness in the præcordia is abnormally widened at and below the apex of the heart, the widening extending more or less upward in proportion to the quantity of effusion. Vocal resonance is diminished or absent within the area of dulness or flatness, and also vocal fremitus. If the sac be distended with liquid, the præcordia may be abnormally prominent and the intercostal depressions abolished, as in pleuritis, within this region. Mensuration will show the increased size of the præcordia. The extent of dulness or flatness at different periods of the disease will show, on the one hand, progressive increase, or, on the other hand, diminution of the quantity of liquid, and at length its complete removal. Dr. J. M. Rotch has called attention to the fact that the pericardial sac, when filled, gives rise to flatness on percussion in the fifth intercostal space on the right side—a fact which I have clinically verified.

The friction murmur either diminishes or disappears after considerable effusion of liquid has taken place. Not infrequently it continues, although the pericardial sac may be filled with liquid and even much dilated. Under these circumstances it is sometimes heard when the patient is raised to the sitting posture, and it may disappear when the patient lies on the back. If it have disappeared or been diminished during the stage of effusion, it returns with increased intensity after the absorption of the liquid, and it then persists until agglutination of the pericardial surfaces takes place. Occasionally it continues during convalescence and after apparent recovery.

The apex-beat of the heart is weakened and may be suppressed by the presence of liquid. If the accumulation of liquid be considerable, the situation of the beat is altered. It is raised to the fourth intercostal space, and carried to the left as far as, or even beyond, the line of the nipple. The sounds of the heart are rendered feeble and distant by the presence of liquid, especially the first sound, and this becomes short and valvular like the second sound.

By means of these signs the diagnosis of pericarditis is generally made without difficulty. Bearing in mind the frequency of its occurrence in articular rheumatism, daily examinations of the chest in cases of that disease with reference to this complication should not be omitted. The fact that it occurs in cases of Bright's disease should also be borne in mind; in fact, it is a good rule, in examining a patient with any disease, not to omit an exploration of the præcordia. Developed in connection with pleuritis or pneumonitis, it may be overlooked, and the diagnosis involves more difficulty than when it occurs in other pathological connections. The determination of pericardial effusion, if a large effusion also exist in the left pleural sac, is not always easy, and the chief reliance in such a case must be on the presence of a friction murmur, which is produced, not without, but within, the pericardial sac. I have reported a case in which the heart was enveloped in either a carcinomatous or sarcomatous growth, completely filling and distending the pericardial sac without altering its form.[1] In this unique case the signs were those of pericardial effusion, and this was the diagnosis. It is difficult to see how pericardial effusion could be excluded in such a case, except by means of an exploratory puncture, which might have been resorted to without harm.

In a case under my observation the pericardium became agglutinated over the left half of the heart, and the right half of the sac was filled with effused liquid. Such a case, if not observed from the beginning, offers a puzzling problem in diagnosis.

The occurrence of delirium, coma, etc. in some cases of pericarditis masks the symptoms pointing to the latter. An examination for the signs of pericarditis should not be omitted in cases presenting notable disturbance of the nervous system.

PROGNOSIS.—Acute pericarditis is in some cases a very dangerous disease, but in other cases comparatively mild. The gravity depends much on the intensity of the inflammation, the amount of inflammatory exudation, and more especially the quantity of liquid effusion, together with the rapidity with which it takes place. A large quantity of liquid, rapidly effused, may lead quickly to a fatal termination. Death in such cases is caused by paralysis of the heart from compression, and cases have occurred in which life was destroyed in this way after the lapse of a few hours only from the time of the attack. Generally, however, the duration of the disease in fatal cases is from one to two weeks, and death takes place by slow asthenia. In such cases the immediate cause of death is either the prolonged compression of the heart by liquid, or weakening of the heart's action as a direct effect of the inflammation on the muscular fibres, or both causes combined. Sudden death by syncope is liable to be produced by muscular exertion, especially if there be much liquid effusion. A patient under my observation, affected with mild pericarditis, died instantly on getting out of bed to go to stool.

The fatality in cases of acute pericarditis is often due, not so much to the disease *per se*, as to coexisting affections. In fact, exclusive of the accidents just referred to, the disease tends to recovery. The danger varies greatly with its different pathological connections. Rheumatic pericarditis, not associated with pleuritis or pneumonitis, is rarely fatal. On the other hand, pericarditis developed in the course of Bright's disease proves fatal in a very large proportion of cases. This difference is explained by the ability of the system to sustain any important intercurrent affection being widely different in these two diseases. Cases in which the pericarditis occurs as a complication of pleuritis or pneumonitis show a large fatality. Traumatic cases generally end in recovery, provided the heart be not injured. Occurring as an idiopathic affection and uncomplicated, the prognosis is favorable.

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[1] Vide *Treatise on Diseases of the Heart*, 2d ed.

If the disease do not prove fatal during its acute course, it either ends in recovery or it becomes chronic. Chronic pericarditis will claim separate consideration. In the cases which end in recovery the usual result is adhesion, by the intervention of newly-formed tissue, of the pericardial surfaces over the whole or the greater part of the organ. In a small proportion of cases the adhesion is limited to a few small spaces. Occasionally, patches of fibrous tissue, without adhesion of the two surfaces, are the only permanent effects of the disease. It is doubtful if complete absorption of the inflammatory exudation ever takes place, followed by no adventitious tissue and leaving no trace of the disease.

TREATMENT.—The widely different circumstances under which acute pericarditis occurs must of course be taken into account in the treatment. The question as to the propriety of bloodletting and other measures heretofore distinguished as antiphlogistic can only be entertained in the first stage of the disease and when it is idiopathic or traumatic or connected with rheumatism. These measures are undoubtedly inappropriate when it is developed in the progress of Bright's disease or when it is secondary to pleuritis or pneumonitis. In general terms, the propriety of these measures admits of consideration only when the pericarditis is either primary, or secondary to affections which do not in themselves impair considerably the ability of the system to sustain an additional disease. When not associated with other diseases which are in themselves dangerous, it is to be borne in mind that pericarditis tends to recovery.

In the cases to which bloodletting must be limited the general principles which are to govern its employment are the same as in other acute inflammations. These principles have been considered.[1] Some importance, perhaps, belongs to this consideration—namely, by diminishing the mass of blood the heart is less excited and its labor diminished. On the other hand, it is to be considered that the danger incident to the disease is mainly from paralysis of the heart or impairment of its muscular power. Giving due weight to these considerations, bloodletting should be employed with great circumspection, even in the few cases in which the symptoms relating to the circulation, in the first stage of the disease, may appear to indicate it. It is never indicated after considerable effusion has taken place.

Depletion by means of saline purgatives is indicated, in the first stage, by the same symptoms as in other acute inflammations. This remark is alike applicable to nauseant and other sedatives. These should be limited to the first stage. They might prove pernicious if continued after effusion has occurred, and hence the great importance of determining the presence of liquid and its amount by physical signs.

Shall the system be brought under the influence of mercury in the first stage? This is a question which different writers and practitioners have answered differently, some advocating rapid mercurialization as highly important, others regarding it as needless and hurtful. In the cases in which mercury is by some considered important, and by many admissible, my experience leads me to be satisfied to forego its use.

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[1] Vide p. 125 *et seq.*

Opium is of great value in this as in other acute inflammations. It is indicated by pain and constitutional disturbance, and is to be employed sufficiently to relieve the general and local symptoms. The form of opiate and the mode of administration are to be determined by the circumstances in individual cases.

Locally, mild revulsive and soothing applications are useful in the first stage. Sinapisms, a poultice, the water-dressing, or the spongio-piline saturated with a warm anodyne infusion may be employed. Blisters in this stage should not be employed. Aside from their interference with the daily physical explorations, which are vastly important, they do more harm by adding to the pain and constitutional disturbance than good by way of revulsion. By German writers cold applied to the præcordia, as in the treatment of acute pleuritis and pneumonitis, is said to be useful. The evidence of its usefulness is the relief of pain, together with diminished frequency of the heart's action.

The indications in the second stage relate, *first*, to the liquid effusion. If the amount of effusion be sufficient to enfeeble the heart by compression, it is highly important to effect the removal of the liquid as speedily as possible. For this end the præcordia may be painted daily with the tincture of iodine, or small blisters may be applied, removing them as soon as vesication begins, and allowing the blistered surface to dry up rapidly. Hydragogue cathartics and diuretics may be employed, as in pleuritis, taking care not to carry these measures so far as to depress the vital powers and weaken the heart's action. This caution is of great importance, and it suggests a *second* indication which may be present in the second stage—namely, to strengthen the heart's action and support the powers of life by the timely and judicious use of alcoholics, tonics, and nourishment. Keeping in view the fact that the disease, if it end fatally, destroys life by affecting directly the heart's action, the measures just named are indicated with an urgency proportionate to the degree of feebleness of the circulation. All measures which tend directly or indirectly to weaken the circulation or depress the vital powers are contraindicated in the second stage. The liability to fatal syncope on exertion is to be borne in mind, and quietude should be rigidly enjoined. Feebleness and irregularity of the pulse in the second (and sometimes in the first) stage constitute an indication for digitalis, which, however, should not be given sufficiently to retard unduly the heart's action.

During the stage of convalescence precautions against undue excitement of the action of the heart by exercise, mental emotions, over-repletion of the stomach, etc. are more important than during recovery from most other acute inflammations. At the same time, convalescence will be expedited and complete recovery secured by a nutritious diet, tonic remedies, gentle gestation out of doors, and other hygienic measures to promote speedy recuperation.

In the treatment of pericarditis developed in the course of either rheumatism or renal disease it may be important to take into account the supposed presence in the blood of a morbid material upon which the pericardial inflammation depends. The indication derived from this view of the causation is, in general terms, to remove, if possible, the continued operation of the cause by measures addressed to the morbid conditions of the blood belonging to those affections. This part of the treatment, however, will be more appropriately considered hereafter in connection with rheumatism and renal disease. The importance of measures to prevent the development of pericarditis in the course of these diseases will also be then considered.

If the disease end in recovery without becoming chronic, adhesion of the pericardial surfaces to a greater or less extent may be expected to take place. This result was formerly supposed to lead to progressive enlargement of the heart, and therefore pericarditis was regarded as a highly serious disease with reference to its remote effects. This is an error which arose from imputing to pericardial adhesions the consequences of the valvular lesions which often coexist. It may be doubted if adhesions alone tend to produce enlargement, and there is reason to believe that if not associated with valvular lesions they may give rise to little inconvenience. The pericardial surfaces are not infrequently found, in autopsical examinations, universally adherent as a result of ancient pericarditis, without any other evidence of cardiac disease, when no symptoms referable to the heart had existed during life. Universal adhesions may be diagnosticated by finding the apex of the heart immovable when the body is placed in different positions; by a visible retraction of the chest-wall and epigastrium synchronously with the ventricular systole; and by the superficial cardiac space remaining unchanged by a deep inspiration. These physical signs denote adhesions not only of the visceral and parietal surfaces of the pericardium, but of the external surface to the adjacent lung and the parietes of the chest. If the latter adhesions have not taken place, the data for a diagnosis are wanting.

Chronic Pericarditis.

Chronic pericarditis may be subacute from the beginning, or it may follow the acute form of the disease. If acute pericarditis do not end in convalescence after the lapse of from two to three weeks, it becomes a chronic affection. As regards anatomical characters, cases differ. In some cases of chronic pericarditis there is no liquid effusion, but the pericardial surfaces are agglutinated by several layers of fibrinous exudation, which collectively may be half an inch or more in thickness. Under these circumstances the exudation is not absorbed, a low grade of inflammation continues, and the disease often ends fatally after a variable duration.

In other cases liquid effusion remains and progressively accumulates; the pericardial sac becomes more or less dilated, and it is sometimes so much enlarged as to depress the diaphragm and occupy the greater part of the thoracic space. In a case reported by Dr. T. P. Satterthwaite the liquid removed after death amounted to eight pounds and four ounces.[1] In a case reported by the late Dr. Swett the quantity of liquid was at least a gallon.

In chronic pericarditis without liquid effusion pain is rarely prominent. The symptoms are those dependent on feebleness of the circulation due to the impaired muscular power of the heart, and on constitutional irritation. In the cases in which large effusion exists the heart is weakened by compression, and the patient suffers from dyspnœa caused by interference with the respiratory function. The dyspnœa may amount to orthopnœa if the accumulation of liquid be large. Pain under these circumstances is rarely a prominent symptom. The symptoms mentioned as due to the pressure of the pericardial sac filled with liquid in the second stage of acute pericarditis may be still more marked when the sac is dilated by effusion in the chronic form of the disease. These symptoms are—aphonia, from pressure on the recurrent laryngeal nerves; dysphagia, from pressure on the œsophagus; cyanosis and turgescence of the cervical veins, from pressure on the venæ cavæ and the yielding walls of the auricles; and singultus, from pressure on the phrenic nerve or branches of the par vagum.

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[1] *Richmond and Louisville Med. Journal*, April, 1872.

Chronic pericarditis with effusion is not to be confounded with pericardial dropsy (hydro-pericardium). The latter occurs in connection with general dropsy, and the accumulation of serum is rarely, if ever, sufficient to dilate the sac. In chronic as in acute pericarditis the liquid is generally sero-fibrinous. In rare instances it is purulent (pyo-pericardium). The effused serum is sometimes colored with blood. This variety of pericarditis, called hemorrhagic, does not claim separate consideration. The term hæmo-pericardium denotes the presence of blood either with or without pericarditis. Without pericarditis it may be a consequence of wounds which penetrate the pericardial sac, and of the rupture of either the heart or an aneurism. In most, if not all, cases of so-called hemorrhagic pericarditis the liquid is bloody serum. This occurs especially in pericarditis associated with purpura hæmorrhagica and scorbutus.

The DIAGNOSIS, if liquid effusion be not present, must often be inferential, being based mainly on the knowledge of the existence of antecedent acute pericarditis. Creaking friction murmur exists in some cases, notwithstanding the agglutination of the pericardial surfaces. If friction murmur be not present and acute pericarditis be not known to have existed, a positive diagnosis is extremely difficult, if not impossible. The heart is found to be more or less enlarged, but there are no means of determining that the enlargement proceeds from a deposit of fibrin. If liquid effusion be present the physical signs render the diagnosis easy and positive. Flatness on percussion exists on the anterior surface of the chest, extending on either side of the sternum toward the axillary region in proportion as the pericardial sac is dilated. Obliteration of the intercostal depressions, and even bulging, may be observed. In a young subject there may be notable projection of the præcordia. Fluctuation in the intercostal spaces is sometimes felt. Laterally, beyond the limit of the flatness on each side, resonance on percussion and the respiratory murmur show the presence of lung. The heart's impulse is suppressed, but a shock communicated by the action of the heart may be felt. A friction murmur is sometimes heard, even when the quantity of effused liquid is very large. The sounds of the heart are feeble, the first sound being distant, more weakened than the second, and short and valvular like the second sound. These signs render the diagnosis sufficiently clear.

The PROGNOSIS in cases of chronic pericarditis is always extremely unfavorable. The disease ends fatally after a duration varying much in different cases.

The indications for TREATMENT are to improve the tone of the system and impart vigor to the heart by tonic remedies, together with nutritious diet and other hygienic measures. The propriety of much counter-irritation is doubtful. Everything which tends, directly or indirectly, to weaken the heart or the vital powers is to be avoided. In this point of view mercurialization is objectionable. If there be much liquid effusion, diuretic remedies and small blisters may be tried. Hydragogue cathartics in these cases, if the patient be feeble, are to be employed with great circumspection. In a case under my observation elaterium, given in small doses and producing but a moderate effect, appeared to hasten the fatal termination. Iodine may be employed as a sorbefacient, externally and internally.

The question as to the propriety of paracentesis relates chiefly to cases of chronic pericarditis. Instances, however, may occur in which, owing to a rapid and large effusion of liquid, this question may arise in connection with the acute disease. If life be in immediate danger from the quantity of liquid, whether the inflammation be acute or not, there can be no room for doubt concerning the propriety of withdrawing the liquid by aspiration; but this question is not restricted to the cases in which it is indicated to avert impending death. May not the rules of practice now generally accepted in cases of pleuritis with effusion and empyema be applied to similar conditions in cases of pericarditis? From a rational standpoint the answer is affirmative. Following these rules, if a large accumulation of liquid remain undiminished by other measures of treatment continued for a reasonable period, aspiration should be employed, and the latter is always indicated in suppurative pericarditis.

As preliminary to the operation there is apparently no objection to an exploratory puncture in order to demonstrate the presence of liquid and to ascertain its character. No harm can arise from the puncture with a needle attached to a hypodermic syringe. Even should the heart be punctured therewith, no serious consequences would follow. A needle, however, should not be used in aspiration, except for the objects just stated. A small-sized trocar is unobjectionable. The suction force by means of Davidson's syringe, properly adapted, is preferable to that produced by Dieulafoy's apparatus or its modifications, the former being simpler and more easily managed.

Roberts has tabulated 60 cases of paracentesis of the pericardium. Of these cases, the number of recoveries was 24. Of the fatal cases (36), 23 survived the operation for one day or longer, the average duration of life being over twenty-seven days, and the longest duration one hundred and sixty days.[1] These results strongly sustain the propriety of the operation, especially when it is considered in how large a proportion of cases pericarditis is associated with other grave affections, and that up to the present time the operation has generally been *en dernier ressort.*

The most eligible point for making the puncture is the fifth intercostal space between the sternum and the linea mammillaris, somewhat nearer to the former than to the latter. It is probably an important precaution to withdraw the liquid very slowly. If the liquid be purulent, and, after repeated aspirations, pus continue to reaccumulate, it is very evident that a permanent opening into the pericardial cavity affords the only hope of effecting a cure.

Pneumo-Pericarditis.

This name denotes the presence of air or gas within the pericardial sac in cases of pericarditis. Air or gas may find its way into this situation through a wound of the thoracic walls or of the œsophagus, and through a fistulous communication between the lungs or the stomach and the pericardial cavity. Air may get access into this cavity through a wound of the chest, and pericarditis not be developed. The condition then is expressed by the term *pneumo-pericardium.* I have reported a case exemplifying this statement.[2] The presence of air and liquid gives rise to splashing and sometimes metallic sounds with the movements of the heart, which are heard on auscultation. Percussion over the præcordia elicits a tympanitic resonance. These cases belong among the curiosities of clinical experience. The treatment of pneumo-pericarditis involves the same principles as the treatment of pericarditis without the presence of air or gas.

Of 28 cases collected by Müller,[3] 9 terminated in recovery. The cases due to traumatism offered the most favorable prognosis. Of 14 such cases, 6 recovered.

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[1] *Paracentesis of the Pericardium*, by John B. Roberts, A. M., M. D., etc., 1880.

[2] Vide *Practical Treatise on Diseases of the Heart*, 2d ed.

[3] Müller, "Drei Fälle von Pneumopericardie," *Deutsches Archiv für klinische Medicin*, Bd. 24, S. 158, 1879.

CHAPTER II.

ENDOCARDITIS.—MYOCARDITIS.—DISEASES OF THE CORONARY ARTERIES.

Simple Endocarditis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Acute Ulcerative Endocarditis.—Myocarditis.

INFLAMMATION of the membrane lining the cavities of the heart, or the endocardium, is called endocarditis.

The inner surface of the endocardium is lined by a single layer of flat, polygonal endothelial cells. Beneath this is a layer of connective tissue extremely rich in elastic fibres. This elastic layer, which with the endothelium constitutes the endocardium, is connected with the muscular wall of the heart by a layer of ordinary fibrillated connective tissue in which may be found scattered muscular fibres. Blood-vessels are present in this layer, but the endocardium itself is non-vascular. The valves may be regarded as duplicatures of the endocardium, between the lamellæ of which is a strengthening layer of fibrous tissue derived from the fibrous rings of the ostia. The semilunar valves are devoid of blood-vessels, and the auriculo-ventricular valves usually contain only a few blood-vessels near their attached borders (Langer). Inflammation of the valves often leads to a new formation of blood-vessels in their substance.

The inflammatory lesions of the valves of the heart are most frequently situated, not upon the free edge of the valve, but upon a thickened line a short distance from this edge, known as the line of closure of the valves.

The inflammations of the endocardium may be divided into simple endocarditis, which may be acute, subacute, or chronic, and acute ulcerative, or malignant endocarditis.

Simple Endocarditis.

A sharp line cannot be drawn between the acute and the chronic manifestations of this disease. A subdivision into verrucous or papillary endocarditis, in which the anatomical lesions appear as warty excrescences on the endocardium, and sclerotic or contracting endocarditis, in which the valves become thickened and shrunken, has been made, but these two varieties of lesions are so intimately related to each other, and so frequently combined, that they will be considered under a common heading.

ANATOMICAL CHARACTERS.—Endocardial inflammation, in the great majority of cases, is situated on the valves and chordæ tendineæ. It is relatively infrequent on the endocardium lining the cavities of the heart. After birth it is generally limited to the left side of the heart. In the rare cases of endocarditis of the right side after birth the left is likewise generally involved. Before birth endocarditis of the right ventricle is most common. Fœtal endocarditis has been often observed, and in the vast majority of cases it is confined to the right side.

Redness, which plays such an important part in the anatomical appearances of the early stage of acute inflammations in most situations, is not of much importance in endocarditis. A diffuse reddish staining of the endocardium, due to post-mortem imbibition of dissolved blood-coloring matter, must not be mistaken for inflammatory hyperæmia. Nothing is known as to the existence of inflammatory exudation upon the free surface of the endocardium. The essential anatomical characteristic of endocarditis, especially in its early manifestations, is the appearance of so-called vegetations upon the endocardium. These vegetations may be minute and in great number, giving only a roughened appearance to the affected endocardium, or they may attain the size of a pea or be even larger. They are of all shapes, and may be connected with the tissue of the endocardium by a broad base or by a narrow pedicle. They cannot be removed from the endocardium without leaving a loss of substance. This fact, together with their microscopical examination, shows that they are outgrowths from the tissue of the endocardium, and not merely fibrinous deposits from the blood, as was once supposed. They are formed by a nodular accumulation of young cells, partly emigrated white blood-corpuscles and partly young connective-tissue cells, in the upper connective-tissue layer of the endocardium. They constitute a kind of granulation-tissue over which the endothelium has disappeared. New blood-vessels are frequently found in these vegetations. A more diffuse infiltration with leucocytes, especially around the vegetations, is also met with in the endocardium. Upon the roughened surface of the vegetations white thrombi are deposited from the blood. These thrombi, which may be of considerable thickness, are formed chiefly of blood-plates, white blood-corpuscles, and fibrin (p. 28). The vegetations, with their caps of fibrin, are favorable points for the detachment of emboli, which may consist either of broken-off thrombi or of fragments of the vegetations. Less frequently than in acute ulcerative endocarditis, loss of substance of the endocardium may occur from breaking off of the vegetations or by necrosis and fatty disintegration of the surrounding infiltrated endocardial tissue. Thus, there may result perforation of the valves, or, on the other hand, valvular aneurism in consequence of bulging of their thinned and softened coats.

In the subacute and chronic forms of endocarditis the vegetations are firmer in their texture, and do not consist so much of granulation-tissue as of fibrillated connective tissue. These firm vegetations may be developed out of the soft, acute vegetations, or they may have been slow in their growth from the beginning. The valves are also much deformed by the thickening of their substance in consequence of the development of new connective tissue, which shrinks and causes retraction of the curtains. At the same time, the chordæ tendineæ are often shrunken and thickened. Abnormal adhesions between the edges of the valves or between the valves and the heart-wall are frequently formed. Regressive metamorphoses, in the form of necrotic foci, of fatty degeneration, and of calcification of the vegetations and of the thickened endocardium, are usually observed. These various changes in acute and chronic endocarditis lead to the development of valvular stenosis and insufficiency.

CLINICAL HISTORY.—Authenticated cases of endocarditis disconnected from other diseases, which serve to mask its symptoms to a greater or less extent, are wanting. The clinical history of the disease, therefore, as far as it is at present known, will require but a brief space. Our knowledge of the disease is confined chiefly to the cases in which it occurs in connection with acute articular rheumatism, and in a certain proportion of these cases it is associated with pericarditis. Pain referable to the præcordia is rarely, if ever, a prominent symptom. The patient may complain of an obscure sense of distress in the præcordia, not amounting to pain. The action of the heart may be morbidly excited. The organ beats with abnormal quickness and frequency, and its action may be irregular. The action of the heart may be out of proportion to the force of the pulse. The local symptoms, in short, as regards the action of the heart, are those of palpitation. In proportion to the acuteness of the inflammation, doubtless febrile movement is induced, together with the symptoms of constitutional disturbance which accompany fever symptomatic of inflammation in other situations. The local and general symptoms, however, are often not prominent, and as far as these are concerned the disease is not infrequently latent. Hence the existence of the disease was unknown prior to the early part of the present century.

PATHOLOGICAL CHARACTER.—The morbid changes which belong to simple endocarditis are in many respects the same as in serous inflammations. Inflammatory products on the surface of the membrane, if they occur in this situation, are carried by the blood-current into the circulation. The local effects of the endocardial inflammation are warty growths, fibrinous deposits, adhesions, etc., an account of which has been given under the heading Anatomical Characters.

CAUSATION.—Endocarditis, in the great majority of cases, is not a primary disease. There is reason, however, to believe that it occurs not very infrequently, and is overlooked in consequence of its latency as regards cardiac symptoms. This may be inferred from the number of cases in which valvular lesions referable to inflammation are found in persons who have never had the disease in the course of which endocarditis is likely to be developed—namely, articular rheumatism. As recognized in practice, it is generally incidental to the disease just named. The proportion of cases of rheumatism in which it occurs is not settled. The statistics of different observers show a variation of from $\frac{2}{3}$ to 9 per cent. A reason for this variation is that the existence of the disease has been often assumed on insufficient evidence. The ground for this statement will appear in connection with the consideration of the diagnosis. The analysis by Sibson of 325 cases of acute rheumatism, observed by him, gave 79 cases in which there was no evidence of endocarditis; in 161 cases the evidence of endocarditis was considered as positive; it probably existed in 13 cases, and it was noted as threatened in 63 cases. It is probable that in some cases the evidence was considered as complete on insufficient grounds. This statement will apply to cases in which the only evidence was a tricuspid regurgitant murmur or an aortic direct murmur, and to cases in which valvular lesions may have existed prior to the rheumatism. The cases of probable and threatened endocarditis cannot be said to have any statistical value. The frequency of rheumatic endocarditis is doubtless affected in not a small degree by the treatment adopted in the early period of rheumatism. It suffices to say that endocarditis occurs frequently in cases of rheumatism. Endocarditis, like pericarditis, may perhaps in some cases precede the affection of the joints. It may occur at any time in the course of a rheumatic attack, but it is usually developed in the early or middle period.

When developed in the course of rheumatism, it is not from the transference of the affection of the joints to the endocardial membrane—that is, not by a metastasis—but, like pericarditis developed in the same connection, it is produced by the causative condition which gives rise to the articular affection.

Endocarditis and pericarditis are not infrequently associated in cases of rheumatism, and this compound affection is called *endo-pericarditis*. Rheumatic pericarditis rarely exists without endocarditis. The reverse of this, however, does not hold good: endocarditis is not infrequently developed without pericarditis. In Sibson's cases endocarditis without pericarditis occurred in 107, and endo-pericarditis existed in 54.

Endocarditis, either with or without pericarditis, is developed, in a certain proportion of cases, in the course of Bright's disease. It occurs occasionally in the eruptive and continued fevers and in cases of pyæmia. It may possibly be produced by contusions of the chest. Its occurrence in chorea is supposed to be frequent. Evidence of its existence was regarded as sufficient in 13 of 34 cases observed by Sibson. The sufficiency of the evidence, however, in all of the 13 cases admits of doubt. This frequency of its occurrence is consistent with the supposed relationship of chorea to rheumatism. Recurring attacks of endocardial inflammation not infrequently take place in connection with existing valvular lesions of the heart.

DIAGNOSIS.—The symptoms in cases of endocarditis are insufficient for a positive diagnosis. The symptoms are even less diagnostic than those which belong to the clinical history of pericarditis, and the disease is oftener completely latent. The diagnosis of this disease, as well as of pericarditis, must rest on physical evidence. The vegetations and fibrinous deposits give rise to an endocardial murmur, and the diagnosis is to be based on the development of this murmur, taken in connection with the symptoms. As the anatomical changes are situated especially upon the curtains of the mitral valve, at least in cases of rheumatic endocarditis, the murmur is usually of mitral origin. The murmur is of a soft or bellows character. It accompanies the first sound of the heart; that is, it is systolic. It is heard loudest at or near the apex of the heart, and may be limited to this situation. As a rule, if prior to the endocarditis the patient were free from valvular lesions, the systolic murmur is not propagated far without the left border of the heart. The anatomical changes which occur during the progress of simple endocarditis do not involve immediate valvular insufficiency either at the mitral or aortic orifice. Hence, the mitral murmur does not proceed from an actual regurgitant current from the ventricle to the auricle. It is not, then, correct to call it a mitral regurgitant murmur. It is an intraventricular murmur, or it may be called a *mitral systolic murmur*, this term not implying the occurrence of regurgitation.

Positive evidence of endocarditis is not afforded by the existence of the murmur just named. A mitral murmur exists often without denoting existing inflammation. It may proceed from valvular lesions which are to be presently considered. To be evidence of existing endocarditis, the murmur must be developed under observation; in other words, one must be satisfied that the murmur which is discovered did not exist on a previous examination. As it is in cases of rheumatism especially that endocarditis occurs, let it be assumed that on the first examination of a patient affected with rheumatism a mitral murmur is found. The murmur is not proof of endocarditis, for it is not known that the murmur did not exist prior to the rheumatic attack. The existence of endocarditis is only probable, and the probability of the disease is greater if this be the first attack of rheumatism. But if, on a first examination, no murmur be found, and subsequently a mitral murmur become developed, it is evidence of endocarditis. The evidence is strengthened if at the same time præcordial distress and excited action of the heart point to the occurrence of a cardiac complication.

Reference has been made, thus far, to a mitral murmur only as evidence of endocarditis. Vegetations and fibrinous deposits at the aortic orifice may occur in this disease, giving rise to an aortic murmur. An aortic direct murmur, and also a pulmonic direct murmur, however, are not infrequent in cases of rheumatism without endocarditis, more especially in women, being due to the condition of the blood; in other words, being inorganic murmurs. Hence, a positive diagnosis of endocarditis is not to be based on these murmurs, unless the symptoms at the same time denote a cardiac complication and pericarditis is excluded. Irrespective of cases of rheumatism, it is well known that an aortic murmur with the first sound—*i. e.* systolic—is common as an inorganic murmur, and alone is never proof of endocarditis. Insufficiency of the aortic valve as an immediate effect of endocarditis must be rare. Sibson, however, gives some instances in which this effect was shown by the occurrence of an aortic regurgitant murmur developed during the rheumatism, and afterward disappearing.

Endocardial murmurs produced by endocarditis may or may not remain permanently. I have repeatedly known a mitral murmur to disappear after recovery from rheumatism. I have known an aortic and a mitral murmur to continue for several years after recovery, and at length to disappear entirely. In cases of endo-pericarditis the endocardial exists with an exocardial or friction murmur. In general, the two murmurs may be readily distinguished, and each referred to its source.

PROGNOSIS.—In the great majority of cases simple endocarditis involves no immediate danger. The danger is for the most part remote, relating to the valvular lesions which often follow. (Vide Chapter III.) The persistence of inflammation in a chronic form is not determinable. The continuance of an endocardial murmur is not proof that inflammation persists; and roughening of the membrane from either vegetations or thrombi which remain will suffice to produce a murmur. The chief source of important morbid effects, exclusive of valvular lesions, is embolism. Vegetations or masses of fibrin of greater or less size are liable to be detached, and, forming emboli, occasion obstruction of arteries in different organs. Passing, of course, into the systemic circulation, they are most likely to become lodged in the spleen and in the kidney, where they give rise to infarctions. They may be carried into the central artery of the retina and give rise to impairment of vision. Again, cerebral embolism may occur, and there have been cases in which gangrene of the lower limbs has resulted from embolic obstruction of their distributing arteries. In the latter case the arteries are generally atheromatous or the circulation is much enfeebled. (For a general account of embolism the reader is referred to Part I. of this work. Vide p. 30 *et seq.*) These are accidents incident to endocarditis. In general, as occurring in its usual pathological connection—namely, in the course of acute articular rheumatism—and disconnected from pericarditis, endocarditis involves no immediate danger and gives rise to no symptoms denoting gravity of disease; but it is frequently latent, and is an important complication, chiefly because it may be the foundation for valvular lesions which, after the lapse of many months, or it may be many years, occasion serious consequences.

TREATMENT.—Simple endocarditis, being confined, generally, to the left side of the heart, and limited chiefly to the valvular portion of the endocardium, does not occasion great constitutional disturbance. Præcordial pain and disturbed action of the heart indicate anodyne remedies and soothing applications to the chest. Blisters with reference to these symptoms are of doubtful propriety. Sinapisms and stimulating liniments will secure all the advantages to be derived from counter-irritation.

Measures addressed to the supposed causative condition of the blood are rationally indicated. Occurring generally in connection with rheumatism, if the local manifestations, this affection of course included, depend on the presence of a morbid material in the blood, it is plainly an object either to neutralize or eliminate this material. The means to be employed for this object will be hereafter considered in connection with the subject of rheumatism;

but it may be here stated that the most important measure is the administration of alkaline remedies in doses sufficient to produce and maintain alkalinity of the urine. The importance of this measure in order to prevent the occurrence of both endocarditis and pericarditis in cases of rheumatism will enter into the consideration of the treatment of the latter disease.

The remote evil consequences of endocarditis proceed from thickening, morbid growths, and the deposit of fibrin upon the valvular portion of the endocardium. It is therefore undoubtedly desirable to limit and remove these immediate effects of the inflammation. The condition of hyperinosis is more marked in rheumatism than in any other disease, and perhaps increases the tendency to fibrinous deposits. With this view, measures addressed to that condition are rationally indicated. Alkaline remedies, there is reason to believe, lessen the liability to fibrinous deposits. Mercury has been supposed to have this effect. That it possesses this power, however, has been inferred rather than demonstrated, and the correctness of the inference admits of doubt. It may be doubted whether the fibrin deposited upon the membrane be ever absorbed; it either remains or is washed away, gradually or in mass, by the current of blood. The importance of absolute rest during the endocardial inflammation and for some time afterward has been enjoined by Sibson, and more especially by Fothergill.[1] The object is to secure a normal minimum of the heart's action in order to prevent changes in the valves leading to permanent valvular lesions. It is reasonable to suppose that these are promoted by an increased strain upon the valves weakened by existing or recent inflammation; hence, everything is to be avoided which will increase the strain beyond that necessarily involved in the tranquil action of the heart. This precaution embraces avoidance of not only physical exertion, but also mental excitement, a stimulating diet, and the use of alcoholics. It is to be added, however, that a good nutrition is to a certain extent protection against these lesions. A diet so reduced as to occasion impoverished blood is injurious.

The practitioner should bear in mind that the persistence of an endocardial murmur is not sufficient ground for persistent medication. The persistence of the murmur is not evidence that the inflammation continues. The medicinal treatment of endocarditis should cease when the local and general symptoms of the disease are no longer present.

Acute Ulcerative Endocarditis.

Among the synonyms for this affection may be mentioned diphtheritic, mycotic or bacteritic, malignant, puerperal, septic, and pyæmic endocarditis. The name acute ulcerative endocarditis is perhaps not the best, as simple endocarditis may be attended with the formation of ulcers, but it is the name commonly employed.

Acute ulcerative endocarditis is distinguished by the malignancy of its course. It may be primary, but it is usually secondary to acute infectious diseases, and in either case it is accompanied by symptoms of severe blood-poisoning, and usually by the development of multiple abscesses. Acute ulcerative endocarditis is due to infection with bacteria, which are always to be found in the valvular lesions.

ANATOMICAL CHARACTERS.—The endocardium presents, in this disease, little necrotic patches of an opaque, yellowish-white appearance. By the breaking down and separation of these patches of dead tissue ulcers are formed. Both the ulcers and the patches of necrosis are usually covered by reddish-gray, soft thrombi, composed of bacteria, blood-plates, leucocytes, fibrin, and red blood-corpuscles. In the margins of the ulcers, and around and subsequently within the necrotic foci, are accumulated small, round cells, probably emigrated white blood-corpuscles. The substance of the affected valves to a greater or less extent is swollen, softened, and lustreless.[1]

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[1] *The Heart and its Diseases*, by J. Milner Fothergill, M. D., etc., 2d ed., 1879.

Valvular aneurisms may be formed by bulging out of the thinned and softened endocardium at the base of the ulcers in consequence of the pressure of blood. These aneurisms, when on the mitral valve, project toward the auricle; when on the aortic valve, toward the ventricle. Of grave import are the perforations and rupture of the valves which may result from the ulcerative process. In a similar way, rupture of the chordæ tendineæ may occur. Fragments of the valves or portions of the thrombotic deposits may be carried into the circulation as emboli.

Microscopical examination reveals constantly, both in the thrombotic deposits and in the substance of the valves, masses of bacteria, which are nearly always micrococci. Although the micrococci have been found in small blood-vessels in the valves, it is probable that they usually invade the endocardium from its free surface, being deposited from the blood within the heart. The immediate effect of the invasion of the endocardium by the micrococci is to cause the foci of necrosis which have already been mentioned. Outside of the necrotic patches, which are mostly or wholly devoid of nuclei, there appear emigrated white blood-corpuscles, which wander into the necrosed tissue.

The bacteria which are present in acute ulcerative endocarditis are not always of the same nature. Both staphylococci and streptococci have been observed, and in most cases these organisms seem to be identical with those found in pus. It is possible to produce artificially lesions identical with those of acute ulcerative endocarditis, by injecting into the blood of animals certain of the micrococci which have been cultivated from pus, after having first mechanically injured the endocardium.[2]

Acute ulcerative endocarditis is particularly prone to affect valves which are already diseased, but it may occur, apparently without previous lesion of the endocardium.

Inasmuch as the emboli which are swept off from the endocardium in this disease contain infectious bacteria, they produce necrosis, and usually abscesses, wherever they lodge. The emboli of simple endocarditis, on the other hand, produce only mechanical effects, being non-infectious or bland emboli. Among the most frequent complications of acute ulcerative endocarditis, therefore, are multiple abscesses in the kidneys, the spleen, and other parts of the body.

Petechiæ are often found in the skin in this disease. Sometimes, but not always, it is possible to demonstrate colonies of micrococci in the capillaries in or near the hemorrhagic tissue.

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[1] According to statistics of malignant endocarditis collected and analyzed by Osler (*The Lancet*, March 7, 1885), the mitral valve alone was affected in 77 cases, the aortic valve alone in 53 cases, the aortic and mitral valves together in 41 cases, the heart-wall in 33 cases, the tricuspid valve in 19 cases, and the pulmonary valve in 15 cases. In 9 cases the affection was confined to the right heart, the tricuspid valve being involved 5 times and the pulmonic valve 4 times.

[2] In several instances of acute ulcerative endocarditis the *Staphylococcus pyogenes aureus* has been found and cultivated. Wyssokowitsch produced artificially acute ulcerative endocarditis by injecting, after lesion of the valves, various forms of bacteria, the most important being *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes* (p. 91). Ribbert was able to produce, experimentally, mycotic endocarditis and myocarditis, without preliminary injury of the endocardium, by injecting cultures of *Staphylococcus pyogenes* grown on potato. The material injected contained particles of the potato, which formed emboli.

It is less easy than might be supposed to draw a sharp line of distinction between acute simple and acute ulcerative endocarditis. The macroscopical appearances may be the same in both diseases. Some pathologists hold that all forms of acute endocarditis are of mycotic origin. The weight of evidence, however, is in favor of the view that acute endocarditis may occur without the presence of bacteria.

Acute ulcerative endocarditis may occur as a primary affection. How, in this case, the bacteria enter the blood is not known. More frequently the disease is secondary to some infectious process, particularly puerperal fever, pyæmia, and acute articular rheumatism, less often pneumonia, dysentery, diphtheria, variola, scarlet fever, and typhus and typhoid fevers. It is not necessary to suppose that the specific organisms of all these infectious diseases are capable of causing malignant endocarditis, but it is more probable that the endocarditis is due in many cases to secondary infection with some form of micrococcus. We know that such mixed infections are not rare in the class of diseases which here come into consideration.

In acute ulcerative endocarditis occurring as a secondary affection in connection with acute rheumatism, puerperal fever, and pyæmia, the symptoms referable to the endocardial affection are commingled with those of the associated diseases, and the affection is liable to be overlooked. If endocardial murmurs be ascertained, they may be supposed to denote only simple endocarditis. Subjective phenomena referable to the heart are not marked and are often wanting. The prominent symptoms are attributable to infective matter contained in the blood. Following an initial chill there is high fever, with notable remissions, the latter occurring irregularly or sometimes simulating those of periodical fever. There may be repeated chills; vomiting and diarrhœa are frequent symptoms; jaundice occurs not infrequently; purpuric spots on the skin are of frequent occurrence; prostration is great; and death is preceded by delirium and coma. Local symptoms in other organs than the heart arise from infective emboli. The affection being generally seated in the left side of the heart, emboli are carried to the spleen and kidneys. The spleen becomes enlarged and tender on pressure. Albuminuria may be present with or without renal embolism, and hæmaturia sometimes occurs. In exceptional cases the right side of the heart is the seat of the affection, and then embolic abscesses may be produced in the lungs. The symptomatology in some cases is suggestive of typhoid fever, and in other cases it accords with that of pyæmia, so that the disease has been considered as presenting two types, denominated typhoid and pyæmic.

When the disease is secondary to pyæmia it is not easy to determine whether the pyæmic symptoms be referable in any measure to the heart. Practically, this question is not one of much moment. The symptoms which have been enumerated, occurring in cases of acute rheumatism and connected with the physical signs of endocarditis, point to this endocardial affection. Further evidence of its existence is afforded by the rapid production of regurgitant lesions of the aortic or the mitral valve, provided it be known that these lesions were not present prior to the occurrence of the attack of rheumatism. The diagnosis, when the disease is primary, can be made with more positiveness. It is to be based on the symptoms and signs of an endocarditis followed by the general and local symptoms which denote pyæmia. Pre-existing valvular lesions of the heart, however, render it difficult to decide as to a recently developed endocarditis, so that under these circumstances the diagnosis is not easy.

It is not known that recovery from acute ulcerative endocarditis ever takes place. With a prognosis based on this fact, all that can be done in the way of treatment, with our present knowledge, is to be guided by symptomatic indications and to endeavor to obviate the tendency to death.

Myocarditis—Diseases of the Coronary Arteries.

Inflammation of the muscular structure of the heart is called myocarditis. Various forms of myocarditis are distinguished, but that form which is of the greatest clinical importance is the variety called *chronic interstitial*, or *fibrous myocarditis*, and fibroid degeneration of the heart.

In the most important class of cases of fibrous myocarditis the primary lesion is not inflammation, but is atrophy or necrosis of muscular fibres of the heart in consequence of obstruction to the circulation in the coronary arteries. Lesions of the coronary arteries causing obstruction are of great clinical and pathological importance. The most common of these lesions is atheroma with calcification. Another lesion is obliterating endarteritis, which is not rare in syphilis and may also occur in other conditions. In consequence of disease of the arterial coats thrombi may form in the affected vessels, and, as the anastomoses of the coronary arteries are few, infarctions follow this complete obstruction. Less frequently, emboli enter the coronary arteries and lead to similar results. Infarctions in the heart may be either hemorrhagic or anæmic necrotic infarctions. If the emboli be infectious, as in acute ulcerative endocarditis, they cause abscesses in the myocardium. If life be sufficiently prolonged, the infarctions are replaced by cicatricial fibrous tissue. Some of the fibrous plaques found in the cardiac muscle are the result of such infarctions. More frequently the obstructions resulting from disease of the coronary arteries are not sufficient to cause typical infarctions, but they cause simple atrophy of the muscular fibres in circumscribed, scattered patches. This atrophy is followed by a new growth of fibrous tissue. According to a view which has much in its support, most of the chronic interstitial inflammations of organs are due to a primary atrophy of the parenchymatous elements.

In the form of fibrous myocarditis, here considered, which is due to disease of the coronary arteries, grayish-white, glistening streaks and patches of fibrous tissue are found scattered through the substance of the myocardium. These streaks and patches may be in any part of the muscular wall of the heart, but they are most frequently found in the anterior wall and apex of the left ventricle and in the papillary muscles. They affect by preference the outer layers of the myocardium, but they may be most abundant near the endocardium.

Another variety of fibrous myocarditis is that which is secondary to fibrous thickening of the endocardium or of the pericardium. Here new-formed connective tissue grows from the thickened endocardium or pericardium into the subjacent muscular tissue, leading to compression and atrophy of the muscular fibres.

In both forms of fibrous myocarditis the heart-wall is weakened, often sufficiently to yield to the blood-pressure and to cause cardiac dilatation. A spot in the heart-wall thus weakened by fibrous growth may bulge out in a sacculated manner and form an aneurism. This is the usual method of development of the so-called chronic partial aneurism of the heart. Such aneurisms are most frequently seated near the apex of the left ventricle, more rarely at the base of the ventricle, where they occupy by preference the situation of the pars membranacea septi, in the latter case bulging into the right ventricle. They may lead to rupture of the heart.

Thrombi are often found in these aneurisms as well as upon portions of the endocardium overlying infarctions and fibrous plaques.

When hypertrophy of the heart is present in cases of fibrous myocarditis, the cause of the hypertrophy is to be sought in concomitant valvular disease or widespread arterio-sclerosis or chronic Bright's disease. Hypertrophy of the right ventricle may be due to insufficiency of the left, in consequence of myocarditis.

In syphilis, patches of fibrous tissue may be found in the heart-muscle with or without the presence of gummata. Whether in such cases the fibrous myocarditis be always dependent upon obliterating arteritis or other arterial disease is uncertain.

Other forms of myocarditis are of less clinical importance. *Acute interstitial myocarditis*, rarely diffuse, more commonly circumscribed, characterized by an accumulation of lymphoid cells, may occur in diphtheria and other acute infectious diseases.

Better understood is *suppurative myocarditis*, or abscess of the heart. The abscesses are usually small and multiple, but a single large abscess may be formed. The most frequent cause of cardiac abscesses are infectious emboli, especially those derived from acute ulcerative endocarditis. The pus may be discharged into the pericardial sac, giving rise to purulent pericarditis, or into one of the cavities of the heart. In the latter case the blood makes its way into the abscess-cavity, thus leading to the formation of acute cardiac aneurism. Acute aneurism may also be produced by deep ulcerations in cases of acute ulcerative endocarditis. Rupture of the heart may be a result of suppurative myocarditis. Micrococci are present in the pus of cardiac as well as of other abscesses, and are to be regarded as the cause of the suppuration.

Some authors assume the existence of a parenchymatous myocarditis; but the changes which are described as belonging to parenchymatous myocarditis are in reality those of albuminous or fatty degeneration of the muscle, and will be described subsequently.

It should be mentioned that extensive disease, particularly atheroma, of the coronary arteries may exist without causing changes in the cardiac muscle. In such cases it is probable that the circulation is not sufficiently obstructed to lead to the changes which have been described, and to which, as will subsequently appear, fatty degeneration of the heart-muscle is to be added.

In all cases of sudden death without other explanation careful examination should be made of the condition of the coronary arteries.

It may be inferred that the myocardium is the seat of lesion of some kind when cardiac symptoms denote something more than functional disorder, and when enlargement from other causes, pericarditis, endocarditis, and valvular lesions can be excluded.

The effect of obstruction of the coronary arteries and fibroid degeneration of the heart is impairment of its functional power. If this effect exceed a certain degree, the symptoms denote persistent weakness of the heart's action. The pulse is feeble, compressible, and often irregular, and in some cases is notably infrequent. Dyspnœa, at first felt only on exercise, may become constant, ending at length in orthopnœa. With notable disturbance of respiration is associated lividity of the prolabia and face. General dropsy may occur, and death may take place slowly, as in cases of dilatation.

The physical signs denote weakness of the heart's action, usually with more or less enlargement of the organ. Cardiac impulse is feeble or wanting. The murmurs indicative of valvular lesions are not present, and these lesions may be thereby excluded. The sounds of the heart are weak. The sound of impulsion over the apex especially is weak, and may not be appreciable, the systolic sound being chiefly or entirely valvular in quality.

The differential diagnosis lies between myocarditis, with obstruction of the coronary arteries, fatty degeneration, and a purely functional disorder. The last-named affection is excluded by the persistence of the symptoms pointing to heart-failure. The symptoms of fatty degeneration are essentially identical, and this affection is not therefore easily excluded. Practically, this is not important. The fact that the patient has had syphilis is in favor of myocarditis. Calcification of arteries which can be felt is suggestive of a similar alteration of the coronary arteries, but is not conclusive evidence.

Sudden death is liable to occur in cases of myocarditis associated with obstruction of the coronary arteries. This may follow violent physical exertion, although not preceded by symptoms denoting any important cardiac affection.

Angina pectoris occurs in connection with obstruction of the coronary arteries, either with or without fibroid degeneration of the heart. Sudden death is liable to occur without as well as with an attack of angina.

The TREATMENT in cases of myocarditis is essentially the same as in cases of dilatation of the heart. (Vide Chapter III.) The objects of treatment relate to symptomatic indications and tolerance. Avoidance of active physical exertion and mental excitement is to be enjoined. Assimilation and nutrition are to be promoted by a nutritious diet and tonic remedies. The iodides and arsenic have been supposed to have a favorable influence. The former are indicated if there be evidence of constitutional syphilis. Impending danger from heart-failure is to be warded off by alcoholic or ethereal stimulants.

CHAPTER III.

VALVULAR LESIONS WITH ENLARGEMENT OF THE HEART.

Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.

THE structural lesions of the heart relate, in the first place, to the valves and orifices of the heart. These are known commonly as *valvular lesions*. Other lesions relate to the walls and cavities of the heart. Enlargement of the heart often proceeds from valvular lesions, and the latter sooner or later, in the great majority of cases, give rise to enlargement. In considering, therefore, valvular lesions, I shall also consider enlargement as dependent thereon, and afterward notice enlargement occurring without valvular lesions.

ANATOMICAL CHARACTERS.—Valvular lesions, in the great majority of cases, are due to chronic endocarditis. When developed after birth, they are nearly always situated in the left side of the heart; that is, at the mitral or the aortic orifice. Tricuspid and pulmonic lesions are comparatively rare in extra-uterine life. Rheumatic endocarditis most frequently attacks the mitral valve. With chronic inflammation of the aortic valve an atheromatous condition of the neighboring portion of the aorta is usually present, the processes in the valves and in the inner membrane of the aorta being essentially similar. Aortic lesions therefore, like atheroma, are more frequent in advanced life than in youth. Chronic endocarditis of the aortic valve is often associated with a similar change in the mitral, particularly in its aortic, segment. The changes in the endocardium produced by chronic inflammation have already been referred to in treating of endocarditis. The morbid process in the valves may begin either acutely or subacutely, or it may develop slowly and insidiously. The essential change is the formation, in the substance of the valve, of new fibrous tissue of firm consistence and with a marked tendency to retract. The thickening is usually most marked along the edges of the valves, where warty outgrowths of fibrous tissue are frequently observed. Calcification and fatty degeneration of the new connective tissue are often extensive. The anomalies in the valves resulting from these changes are—1, thickening and rigidity due to the formation of new connective tissue and the deposit of the salts of lime; 2, shortening in consequence of retraction of the newly-formed tissue; 3, adhesion of the valvular segments to each other or to the heart- or vessel-wall; 4, valvular aneurisms and rupture of the valves. Shortening, rigidity, and sometimes rupture of the chordæ tendineæ may coexist. Deposits of thrombi may be present on the thickened and calcified valves, and serve as a source of emboli. Emboli may also be formed by ruptured fragments of the valves or by the breaking off of calcific incrustations. The thickened valvular segments are sometimes united at their sides to such an extent as to leave only a buttonhole-like slit sufficient for the admission of the end of the little finger, or sometimes, in the case of the aortic valves, for the passage only of a crow's quill.

The importance of these various changes lies in the fact that they constitute the vast majority of those structural alterations which interfere with the function of the valves. From a clinical point of view all the diverse alterations of structure embraced under the name valvular lesions may be arranged, according to their effects upon the circulation, into the following groups: *First*, obstructive lesions—that is, lesions which impede the flow of blood by producing contraction or stenosis of the orifices; *second*, regurgitant lesions—that is, lesions which render the valves incompetent or insufficient, consequently allowing backward or regurgitant currents; *third*, lesions which involve both obstruction and regurgitation; and *fourth*, lesions which involve neither obstruction nor regurgitation, but which give rise to morbid sounds by roughening the surface over which the blood flows. The lesions belonging to the last group are of little or no immediate importance, and, although giving rise to abnormal sounds known as endocardial or bellows murmurs, they are innocuous. The most frequent form of lesion is that which occasions both obstruction and insufficiency at the same time, one or the other of these effects frequently preponderating. Obstruction unaccompanied by any regurgitation is rare, while regurgitation without obstruction is not infrequent. In certain instances we must recognize the existence of so-called *relative insufficiency* of the valves. This is incident to cases of marked dilatation of the heart-cavities, the ostia becoming so wide that the unchanged valvular segments are incapable of completely closing the dilated orifices. This condition is met with most frequently at the tricuspid orifice in cases of extreme dilatation of the right ventricle as a consequence of mitral disease.

The lesions which involve obstruction or regurgitation lead primarily to certain changes in the heart itself, and secondarily to alterations in various other organs of the body. These changes in the heart are at first of a nature to compensate more or less completely the impairment of function of the valves. The mechanical effects of obstruction or of regurgitation at any of the valvular orifices, when not compensated, are a diminution in the amount of blood supplied to the aorta and general arterial system and a corresponding over-repletion of the systemic veins. These effects are produced by stenosis and by insufficiency from valvular disease of either the left or of the right side of the heart. The blood-pressure in the systemic arteries is therefore lessened, while that in the veins is increased; and the result is retardation of the general circulation. These effects of impaired function of the valves are at first more or less completely compensated by increase in the capacity and the strength of certain portions of the heart. Certain cavities dilate and their walls become hypertrophied, so that, notwithstanding the valvular defects, the arteries of the body receive their proper amount of blood. During what may be termed the period of compensation the hypertrophy bears a harmonious relation to the dilatation; when the compensation becomes disturbed or destroyed, the dilatation exceeds in proportion the hypertrophy. During the stage of compensation, which may last for years, the individual suffers but little inconvenience from the valvular lesions. Sooner or later, in the majority of cases, the compensation is disturbed, and various characteristic symptoms and morbid changes ensue.

In order to understand the changes in the heart it is well to consider individually the different valvular lesions as to their effects. In cases of insufficiency of the mitral valve (the most frequent of all valvular lesions), during the systole of the left ventricle a portion of the blood is sent back into the auricle, and the aorta receives a proportionately smaller amount of blood. During the diastole an abnormally large amount of blood flows into the left ventricle in consequence of the over-accumulation in the left auricle and the pulmonary veins. The left ventricle is thereby over-filled, and as a consequence its wall becomes hypertrophied. At the same time the left auricle becomes dilated and hypertrophied; but the hypertrophy of the thin-walled auricle cannot compensate the over-accumulation of blood. The blood is therefore dammed back upon the pulmonary veins, whence the increase of tension is transmitted through the pulmonary vessels to the right ventricle. This becomes over-distended with blood, and in turn it becomes hypertrophied. Mitral insufficiency is thus compensated by the hypertrophy of the two ventricles, so that notwithstanding the regurgitation at the mitral valves a normal amount of blood is transmitted into the aorta. The muscular coat of the auricles is so thin that they can accomplish but little in the way of compensating valvular lesions. It may be that the compensation for mitral insufficiency is so complete that the patient suffers for a considerable period no inconvenience. When the compensation is destroyed the dilatation becomes excessive, the right auricle becomes over-distended, and the blood is dammed back upon the general venous system. The effects of this venous congestion of the various organs and parts of the body will be presently described.

In mitral stenosis less blood than normal flows into the left ventricle through the contracted orifice during the diastole. The blood accumulates in the left auricle, which dilates and becomes hypertrophied. The blood-pressure, as in mitral regurgitation, increases in the pulmonary vessels, and consequently the right ventricle becomes over-filled and hypertrophied. Inasmuch as the left auricle and right ventricle are rarely able to completely compensate the obstruction, the left ventricle receives less than the normal amount of blood, and, being called upon to do less work, it becomes atrophied. Mitral stenosis unaccompanied by regurgitation and consequent hypertrophy of the left ventricle, however, is rare. It will be observed that while mitral regurgitation is compensated by hypertrophy of both ventricles, mitral stenosis is compensated essentially by hypertrophy of the right ventricle, and is less fully counteracted.

The immediate result of insufficiency of the aortic valves is that during the diastole a portion of the blood which had been driven into the aorta by the systole flows back into the left ventricle. The ventricular cavity is thereby over-filled and its wall becomes hypertrophied, so that by its increased power of contraction the excessive amount of blood which accumulates in the left ventricle during the diastole can be forced into the aorta.

In this way the arteries can receive their normal quantum of blood notwithstanding the regurgitation. No valvular lesion, as a rule, is more completely compensated than aortic insufficiency. In consequence of the backward flow of blood from the aorta the left auricle cannot empty itself as readily as usual, and it therefore dilates and becomes hypertrophied. The blood is then dammed back upon the lungs, and, as in the previous instances, the right ventricle becomes hypertrophied and dilated. The tension in the right ventricle may be still further increased by simultaneous involvement of the mitral valve in the endocarditic process, or rarely by relative insufficiency of the mitral valve.

In aortic stenosis the left ventricle, being unable to completely empty itself, becomes hypertrophied and dilated. The degree of dilatation, however, is usually less than in cases of aortic insufficiency. Dilatation and hypertrophy of the left auricle and of the right side of the heart are produced in the same way as in the valvular lesions already described. The most extensive enlargements of the left ventricle met with are those consecutive to aortic insufficiency accompanied with moderate stenosis. Aortic lesions are situated more favorably as regards completeness of compensation than mitral, because the former defects are counteracted by hypertrophy of the ventricle immediately behind the valves, while the latter require hypertrophy of the opposite ventricle, to which the increase of tension must be transmitted through the pulmonary vessels. When, however, the compensation of aortic lesions becomes disturbed—in other words, when the left ventricle is no longer able to meet the increased demands upon its strength—the grave symptoms of venous stagnation follow with greater rapidity than after disturbance of the balance in mitral lesions.

After birth, relative insufficiency of the tricuspid valve is more frequent than insufficiency due to chronic endocarditis of the valve. In either case the affection is usually secondary to disease of the left ventricle. As the result of tricuspid regurgitation, less blood than normal is sent to the pulmonary vessels, and therefore the pressure in the pulmonary veins and that in the aorta are abnormally diminished. On the other hand, in consequence of the over-accumulation of blood in the right auricle the right ventricle is filled under increased pressure. Hypertrophy and dilatation of the right auricle and of the right ventricle ensue in the same way as enlargement of the opposite cavities in mitral regurgitation. In uncomplicated tricuspid regurgitation the left ventricle does not enlarge, because the pressure in the pulmonary veins is not increased, and the aortic tension is not elevated, notwithstanding the regurgitation of blood into the general venous system. Tricuspid regurgitation is therefore imperfectly compensated.

Stenosis of the tricuspid valve, unaccompanied by insufficiency, has been observed only as a result of fœtal endocarditis. Uncomplicated tricuspid stenosis belongs among the curiosities of medical experience. It is the most unfavorable of all valvular lesions as regards compensation. It can be only very imperfectly compensated by dilatation and hypertrophy of the right auricle, all of the other cavities of the heart receiving less than the normal amount of blood, and therefore becoming atrophied. Tricuspid stenosis, as a rule, is accompanied by other valvular lesions.

Stenosis and insufficiency of the pulmonic valves are well-recognized forms of congenital valvular disease. Still, they are very rare. When of fœtal origin they are often complicated with other anomalies, such as stenosis of the pulmonary artery or of the conus arteriosus, perforation of the septum ventriculorum, and non-closure of the foramen ovale or of the ductus Botalli. When uncomplicated they are compensated, often completely, by dilatation and hypertrophy of the right auricle and ventricle. The left side of the heart does not enlarge, but, on the other hand, it has been found atrophied. These lesions may develop in very rare instances as a sequence or concomitant of chronic endocarditis of the left side.

We have now considered the mechanism of the production of the primary changes in the heart following valvular lesions. It has been shown that the different lesions may be compensated to a greater or less extent by hypertrophy and dilatation of certain cavities of the heart. The dilatation is mechanically produced by over-accumulation of blood; the hypertrophy is the result of augmented nutrition of the muscular wall consequent upon the demand for increased activity of the heart. In favorable cases the hypertrophy bears a certain definite relation to the dilatation and to the degree of obstruction or regurgitation, so that the functional defects of the valves are as nearly counteracted as may be. In unfavorable cases, even very early, the dilatation may so predominate over the hypertrophy that the valvular lesion may never be as fully compensated as is possible. This is likely to be the case in persons very much enfeebled, especially when the valvular defects are acutely developed.

In nearly all valvular lesions, either early, or, more frequently, after a variable period of compensation, the dilatation comes to bear an excessive ratio to the degree of hypertrophy, and the blood is dammed back upon the general venous system. Changes ensue referable to chronic venous congestion or passive congestion of various parts of the body. A knowledge of these changes is necessary to a proper understanding of the symptoms common to uncompensated valvular lesions. They can only be referred to briefly here, as their description belongs to other sections of this work.

The compensation is sometimes disturbed by extension of the morbid processes in the valves, and in many cases it is impaired by fatty degeneration of the hypertrophied muscular tissue. Depression of the general vitality of the patient and the occurrence of intercurrent disease likewise enfeeble the heart. In some cases, however, the cause of the failure of the heart is not evident.

In the lungs, as a result of chronic venous congestion, the condition already described as brown or pigment induration is developed (p. 181). In this state the pulmonary capillaries are distended and encroach upon the air-cells. Hemorrhagic pulmonary infarctions are frequent, produced by emboli in branches of the pulmonary artery (p. 266). These emboli are derived usually from thrombi formed in the right side of the heart. It is not rare in the later stages of valvular disease for thrombi to form, especially in the apices of the ventricles and in the auricular appendices, in consequence of the retardation of the blood-current. Chronic bronchitis usually attends valvular lesions of the left side of the heart. The condition of the heart during the stage of non-compensation is particularly favorable for the production of œdema of the lungs.

In consequence of chronic passive congestion the condition called nutmeg liver is developed. The blood being dammed back upon the inferior vena cava and hepatic veins, the central veins of the hepatic lobules become distended, and the lobules present a red centre and light periphery. In the peripheral zone the hepatic cells often contain fat. This swollen state of the liver may be followed by a so-called red atrophy of the organ—a condition to be hereafter described in Section IV.

Chronic congestion of the spleen is evident after death from its enlargement, dark color, and firm consistence. The kidneys are enlarged, and in typical cases of extremly hard consistence and of a dark-red color. This condition was formerly mistaken for chronic interstitial nephritis. The albuminuria of cardiac disease is explained by increase of pressure in the renal veins and consequent slowing of the circulation, and the diminution in the amount of urine excreted, by the diminished tension in the renal arteries. Chronic congestion of the alimentary tract may induce chronic inflammation of the stomach and intestine. Cyanosis and anasarca, with transudation of serum into the various serous sacs of the body, result from the general venous congestion. In consequence of the retardation of the general circulation the blood may coagulate in the peripheral veins and venous sinuses of the body. Thrombi so formed are called marantic (p. 28). They form an additional source of origin for emboli which are carried to the pulmonary artery. Venous hyperæmia and arterial anæmia of the brain are among the consequences of uncompensated valvular lesions. The lodgment of emboli in the cerebral arteries, particularly in the middle cerebral or its branches, and the consequent softening of the anæmic portion of the brain, will be considered in Section V.

CLINICAL HISTORY.—Valvular lesions, so long as they involve neither obstruction nor regurgitation, give rise to no symptoms of disease, and their existence is only ascertained by means of physical signs. Lesions involving either obstruction or regurgitation, or both, as a rule do not occasion inconvenience until they have led to enlargement of the heart. Moreover, lesions occasion suffering and are attended with danger in proportion as the dilatation predominates over the hypertrophy.

The first symptoms proceeding from *mitral obstructive or regurgitant lesions* pertain to the respiratory system. Deficiency of breath on exercise is for some time the chief inconvenience. This progressively increases in proportion as the obstruction to the pulmonary circulation increases. The patient is at length obliged to discontinue any active muscular exertion, but may be comfortable while remaining quiet. Dyspnœa finally becomes habitual, and is more or less prominent as a symptom after dilatation of the right ventricle has taken place. A still further development of this symptom renders the patient unable to lie down, then constituting orthopnœa. This may be due, in a measure, to coexisting pulmonary conditions. Cough and more or less muco-serous expectoration are incidental to congestion or a low grade of inflammation of the bronchial mucous membrane. An abundant serous expectoration sometimes occurs, constituting bronchorrhæa. Hæmoptysis is not infrequent, the hemorrhage usually being small. This event generally denotes simply bronchorrhagia, but occasionally blood is extravasated into the air-cells, constituting circumscribed pulmonary apoplexy or hemorrhagic infarction. Pulmonary œdema is another intercurrent event which is liable to occur, increasing the suffering from dyspnœa. Hydrothorax is still another source of embarrassment of respiration. Pigment induration of the lungs dependent on persistent congestion, caused by regurgitant and obstructive mitral lesions, enters into the rationale of dyspnœa in many cases.

After enlargement of the heart takes place, its action, so long as the hypertrophy predominates, is abnormally strong. This may be perceived by the hand placed over the præcordia. The patient, however, rarely complains much of palpitation, having become accustomed to the gradual increase of the power of the heart's action. The pulse is small and weak in proportion to the amount of obstruction or regurgitation, each of these immediate effects of the lesions lessening the quantity of blood propelled into the aorta and its branches by the systole of the left ventricle. The quantity of blood contained in this ventricle at the time of a systole may sometimes be so much lessened that it fails to produce a radial pulse; hence one source of intermission of the pulse. Another source is an actual intermission of the ventricular systole. Inequality of the successive beats of the pulse, as regards volume and force, represents unequal supplies of blood from the left auricle to the left ventricle. Irregularity in the action of the heart is not infrequent. Intermittency of the pulse and inequality of successive beats, due to deficiency of the supply of blood to the left ventricle, are characters which denote mitral contraction rather than regurgitation.

The characters pertaining to the form of the pulse, as ascertained by the sphygmograph, in cases of mitral regurgitation, are—notable variations in the length of the line of ascent in different pulsations, shortness of this line in a greater or less number of pulsations, its vertical direction, and marked dicrotism in the line of descent. In cases of mitral obstruction the line of ascent is longer and the variations in different pulsations are less, the direction of this line being also vertical, and the line of descent showing dicrotism. These characters denote quickness of the ventricular systole, feeble arterial tension, and want of uniformity as regards the quantity of blood sent into the aorta in different beats.

Pain is rarely a prominent symptom during the progress of mitral lesions, but uneasiness and an indefinite sense of distress, referred to the præcordia, may be complained of. Nor are there any notable symptoms referable to the nervous system. There is a marked contrast, as regards mental anxiety and apprehension, between the cases in which the action of the heart is disturbed by lesions and those in which the disturbance is purely functional. In the former mental depression is proportionate to the physical suffering, and patients are likely to be apathetic with respect to danger; in the latter the mind is depressed out of proportion to the actual ailment and patients are harassed by imaginary fears. If dyspnœa be a prominent symptom, the loss of sleep adds to the sufferings of the patient. The sleep which is obtained is imperfect and disturbed by frightful dreams. It is not uncommon for patients to be unable to lie down for many weeks before death, short periods of sleep being obtained by inclining the body forward and resting the elbows on the knees or on some solid support.

Sooner or later, if life be not cut off by some intercurrent affection, dropsical effusion takes place into the areolar tissue and serous cavities. The lower limbs generally first become œdematous, afterward the face and body, and more or less liquid accumulates within the peritoneal and within the pleural sac. The limbs and body sometimes become greatly swollen and the scrotum acquires an enormous size. The skin on the lower limbs under these circumstances may become erythematous; ulcerations or fissures are liable to occur, with abundant draining away of serum. Other circumstances, in addition to dilatation of the right side of the heart, may contribute to the occurrence of dropsy; for example, anæmia. The face is congested, and not infrequently the lips are livid. The dusky hue, due to congestion or cyanosis, combined with œdema, gives to the face an appearance which contrasts with the pallid aspect characterizing general dropsy from renal disease. General dropsy arising from mitral lesions occurs after enlargement by dilatation of the right side of the heart has taken place, and is dependent mainly on the venous obstruction which the latter condition involves. Tricuspid regurgitation, which may exist to some extent normally, is increased in this condition, and adds to the venous obstruction. This condition of the right cavities gives rise to turgescence of the cervical veins, especially when the patient is sitting or standing, and this turgescence is sometimes very great. Under these circumstances pulsation of the superficial veins of the neck, occurring synchronously with the auricular or ventricular systole, or with both, is not uncommon. Jugular pulsation is, however, observed in some cases without venous turgescence, and when dropsy has not taken place venous pulsation of the veins of the extremities is sometimes observed.

The organs composing the digestive system suffer from the congestion arising from dilatation of the right side of the heart. Enlargement of the liver is sometimes observed. Pulsation over the liver, apparent to the eye and touch, is an occasional effect of tricuspid regurgitation with hypertrophy of the right ventricle. The pulsation is sometimes so strong as to simulate aneurism. The appetite and digestion become impaired. Prior to the occurrence of dilatation, however, the appetite and digestion may be but little disturbed. Nutrition may be but little affected even after the lesions have led to considerable suffering. Emaciation is not an early, nor at any time a marked, effect of valvular lesions of the heart. The kidneys participate in the congestion, and the secretion of urine is scanty. Albumen may be present in the urine as an effect of renal congestion without denoting degenerative or inflammatory disease of the kidneys. Hemorrhage from the stomach or intestines is an occasional effect of the congestion of the mucous membrane in these situations. Hemorrhoids and epistaxis belong in the same category.

Aortic lesions involving obstruction or regurgitation usually give rise, as the first symptom, to inconvenience from the increased power of the heart's action consequent upon hypertrophy of the left ventricle. The patient complains of palpitation, especially on exercise or under the influence of mental emotions. The action of the heart is felt, by the hand over the præcordia, to be inordinately strong, and the dress or portions of the body may show visible movements with each ventricular systole. Pain is more likely to be present than in cases of mitral lesions, and, irrespective of the painful affection called *angina pectoris*, it is not infrequently a prominent symptom. Cough, expectoration, dyspnœa, and hæmoptysis, dependent on pulmonary congestion and the accidents incident thereto, occur less frequently in connection with aortic than with mitral lesions. Pulmonary congestion, dependent on dilatation of the left auricle, does not occur until the dilatation of the left ventricle becomes predominant, and the disease may end fatally without this result taking place.

If the lesions produce contraction with little or no regurgitation, the pulse is simply weakened, but not in a notable degree even when the contraction is considerable. Under these circumstances, however, the pulse is frequently weak in proportion to the power of the heart's action; and the force of the stroke felt by the finger over the artery is in striking contrast to the impulse felt by the hand over the præcordia. If the lesions impair the aortic valve so as to involve considerable regurgitation, the pulse is somewhat distinctive of this fact. The artery strikes against the finger with quickness, and appears instantly to recede; the stroke is not sustained, and the pulse is said to be *jerking* or *collapsing*, giving a sensation as if a "ball of blood were shot through the artery." This is due to the fact that directly after the ventricular systole the aorta is emptied by the regurgitant current, and this current and the direct current come into collision when the ventricle contracts. Another symptom pertaining to the arteries in some cases is distinctive of free aortic regurgitation—namely, visible movements of the arterial trunks which are superficially situated, such as the carotid, temporal, subclavian, brachial, etc. The appearance caused by the movements of these vessels is sometimes very striking, and it is so distinctive that aortic regurgitation is rendered highly probable by this symptom alone.

The tracings by the sphygmograph in cases of aortic obstructive lesions give an oblique and curved line of ascent, denoting the slowness with which the blood is driven into the aorta, and in the line of descent dicrotism is either slight or wanting. In cases of aortic regurgitation the line of ascent is vertical, and dicrotism in the line of descent is marked. If, as is not infrequent, senile induration of the arteries coexist, the summit presents a plane of greater or less length. If this condition of the arteries do not exist, the upper part of the diagram representing a pulsation is pointed, and the line of ascent may be notched near its upper extremity.

General dropsy occurs in only a small proportion of cases of aortic lesions; that is, dropsy arising from the cardiac affection. This is owing to the fact that life is generally destroyed before the lesions have led to predominant dilatation of the right cavities of the heart. Hence, turgescence of the cervical veins and jugular pulsation do not belong to the clinical history of aortic lesions. This statement also holds good with respect to congestion of the abdominal viscera and the kidneys.

Aortic regurgitant lesions give rise to symptoms proceeding from a sudden increase of the accumulation of blood within the left ventricle sufficient to embarrass the action of this ventricle by distension. This ventricle becoming overloaded by the regurgitant current in addition to the direct mitral current, the ventricular walls are distended so as to weaken their power of contraction. This is a rational explanation of paroxysms of distress to which these lesions give rise. The action of the heart is irregular and spasmodic, and the patient experiences a sense of great oppression at the præcordia, with a feeling of impending death. These attacks are excited at first by muscular exercise or mental emotions, but after a time they may occur without any obvious exciting cause. They occasion great distress, and sudden death may take place in an attack of unusual violence, the left ventricle being paralyzed by over-distension. They are more frequent and serious when dilatation of the left ventricle predominates over hypertrophy, yet they may be more or less frequent and violent, and may prove fatal while hypertrophy is predominant. The occurrence of these attacks renders patients affected with aortic lesions more anxious and apprehensive than those affected with mitral lesions. The danger from over-distension of the ventricle is perhaps greater without than with mitral insufficiency. The latter, as regards this danger, may be said to be compensatory.

Aortic and mitral lesions, involving either obstruction or regurgitation, or both, in each situation, are not infrequently associated in the same case. Certain of the symptomatic phenomena arising from lesions at the mitral and aortic orifice are then combined. Mitral obstruction as well as regurgitation, however, may afford some protection against the paroxysms of distress and the liability to sudden death incident to aortic regurgitation, by preventing over-distension and paralysis of the left ventricle.

Tricuspid and pulmonic lesions, as already stated, are comparatively rare. In the majority of cases in which they exist they are congenital. They are therefore most liable to be met with in young subjects. Tricuspid lesions, in proportion as they involve obstruction or regurgitation, lead primarily to dilatation of the right auricle, and thence to systemic congestion. General dropsy under these circumstances is likely to occur early, without having been preceded by the symptoms denoting pulmonary congestion. Turgescence of the cervical veins and venous pulsation are also early symptoms in such cases. Lesions at the pulmonic orifice, involving contraction or regurgitation, in like manner soon lead to dilatation of the right auricle and the symptomatic phenomena dependent thereon.

PATHOLOGICAL CHARACTER.—The primary evils of valvular lesions are mechanical. They do harm by obstructing the direct or normal currents of blood, and by allowing abnormal or regurgitant currents to take place. They induce hypertrophy or morbid growth of the walls of the heart by increasing the force of the heart's action, and thereby inducing hypernutrition. This result, it is to be borne in mind, is not an evil. The hypertrophy resulting from valvular lesions is conservative. The augmented power of action which the heart acquires with its increased muscular growth enables it better to carry on the circulation despite the obstacles afforded by obstructive and regurgitant lesions. The hypertrophy is compensatory for the obstacles which these lesions afford. This fact has an important practical bearing on the management. Dilatation of the cavities is another result of valvular lesions. This result, when it predominates over hypertrophy, is an evil. The heart is weakened in proportion as it becomes dilated. The circulation then suffers, not only from the obstacles afforded by the valvular lesions, but from the inadequate power of the heart's action. As a rule, it is not until dilatation predominates over the hypertrophy that distressing and grave symptoms of disease of the heart are developed; and the suffering and danger increase as the dilatation progresses.

It is important to bear in mind that valvular lesions do not necessarily involve existing inflammation. The atrophy and calcareous degeneration which lead to contraction of the valves, rigidity, rupture, etc. are not inflammatory processes, although they may be, and in a large proportion of cases probably are, the after-effects of inflammation.

CAUSATION.—In the majority of cases valvular lesions originate in an endocarditis occurring in acute articular rheumatism. The degenerative changes which follow endocarditis lead to obstruction or regurgitation after a period more or less remote. When the evils or inconvenience resulting from valvular lesions are sufficient to lead the patient to seek for medical aid, it may be found that one or more attacks of rheumatism were experienced five, ten, fifteen, or twenty years before; and when cases first come under observation more or less enlargement of the heart has already taken place. Enlargement, indeed, has probably existed for a long time, and it may not be until the heart becomes weakened by dilatation that the patient applies to the physician. Moreover, rupture of the valves or tendinous cords sometimes results from the violence of the heart's action, without any previous structural change. Rheumatic endocarditis affects by preference the mitral valves. The congenital lesions generally affect the valves of the right side. On the other hand, chronic inflammation of the aorta, leading to an atheromatous condition, is likely to involve the aortic valves. These lesions are more frequent in advanced than in early life, whereas the mitral lesions consequent on rheumatic endocarditis are oftener manifested in the latter period. Chronic non-rheumatic endocarditis, however, often affects both the aortic and the mitral valves, more especially the aortic segment of the latter. Valvular lesions occur not infrequently as a remote effect of syphilis.

DIAGNOSIS.—The existence of valvular lesions can be determined with positiveness only by means of auscultatory signs. By means of signs the existence of lesions, their situation, and their character as regards being obstructive or regurgitant may generally be ascertained. Lesions situated at the different valves and orifices are represented by endocardial murmurs, and the presence of these is evidence of the existence and situation of the lesions. It will suffice here to enumerate the several organic murmurs produced within the heart, together with the distinctive characters and significance of each. They are naturally arranged into mitral, aortic, tricuspid, and pulmonic murmurs.

Mitral Murmurs.—A murmur heard with the first sound of the heart—*i. e.* systolic, having its maximum of intensity at or near the situation of the apex-beat, or perhaps limited to that situation—represents mitral lesions. These lesions may or may not involve insufficiency of the valve and consequent regurgitation. If a regurgitant current exist, the murmur is properly called a *mitral regurgitant murmur;* but as mere roughness of the valve probably may give rise to a murmur with the characters just mentioned, without regurgitation, the name just given is not always strictly correct. A better name is *mitral systolic murmur*, with or without regurgitation. If the murmur be diffused beyond the apex, around the left lateral surface of the chest, and heard on the back near the lower angle of the scapula on the left, and sometimes on the right side, mitral regurgitation may be inferred. If the murmur be heard only around the apex and over the body of the heart, it does not afford proof of regurgitation; it may be a mitral systolic, non-regurgitant, or an intraventricular murmur. A mitral systolic murmur, regurgitant or non-regurgitant, is not transmitted above the heart, and is not heard over the carotid arteries. This murmur may be soft (bellows-like) or rough, and sometimes it is musical. It is to be distinguished from a murmur sometimes produced by the impulse of the heart against the portion of lung covering the apex. This exocardial murmur is called a *cardiac pulmonary murmur.* It is usually limited to the inspiratory act and to a space over or a little to the left of the apex-beat.

A murmur heard after the second and just before the first sound of the heart—*i. e.* presystolic, continuing up to the first sound and instantly arrested when this sound occurs—also represents, generally, mitral lesions. This murmur is heard at or near the apex, and is usually confined within a circumscribed area around the apex. It is almost always rough, resembling a sound produced by throwing the lips or tongue into vibration with the expired breath. The letters *rrrp*, whispered, the tongue vibrating with the first three letters, represent the murmur, the letter *p* representing the first sound of the heart. This murmur is produced by contraction of the mitral orifice in consequence of the union at their sides of the mitral curtains, forming a buttonhole-like slit. The murmur represents mitral contraction with occasional exceptions. In some cases in which free aortic regurgitation exists, the left ventricle becoming filled before the auricles contract, the mitral curtains are floated out, and the valve is closed when the mitral current takes place. Under these circumstances the murmur may be produced, although no mitral lesion exists. I have reported cases in which this murmur was marked, the autopsy showing no mitral lesions, but aortic lesions permitting free regurgitation.[1] This murmur is called the *mitral direct* or *presystolic murmur.*

A mitral murmur, following the second sound of the heart and ending before the first sound, is produced by the current of blood from the auricle to the ventricle prior to the auricular contraction. This murmur may have the quality which usually characterizes the presystolic murmur, or it may be a soft blowing sound. It is heard at or a little above the apex. This murmur may be called the *mitral diastolic murmur.* It may be associated with the presystolic murmur, and the two murmurs will then occupy the whole of the long pause of the heart.

The murmurs produced by the direct and the regurgitant streams of blood not infrequently coexist. Either may exist without the others, the mitral regurgitant being much the more frequent.

Aortic Murmurs.—A murmur with the first sound, or a systolic murmur, heard with its maximum of intensity at or above the base of the heart and propagated into the carotids, is called an *aortic direct murmur.* This murmur may represent aortic lesions, and it is then called organic, or it may be due to a morbid condition of the blood, without aortic lesions, and it is then called an inorganic, an anæmic, or a hæmic murmur. The following circumstances render it probable that the murmur is inorganic or anæmic: The evident existence of anæmia; the absence of other signs of cardiac lesions; the variableness of the murmur, being sometimes present and sometimes wanting, and differing much at different times in intensity; the presence of murmur in the larger arteries, such as the carotid, subclavian, etc., and a continuous murmur in the veins of the neck, commonly known as the venous hum. Attention to these points will generally suffice for determining whether an aortic direct murmur be organic or inorganic. The organic murmur denotes lesion of some kind, but not necessarily obstructive; that is, the lesion may simply render the membrane at or near the aortic orifice rough, without involving obstruction to the current. The murmur may be soft or rough, and it is sometimes musical.

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[1] Vide article on cardiac murmurs by the author in the *American Journal of the Medical Sciences*, July, 1862. A case in which a mitral presystolic murmur existed without mitral lesions, free aortic regurgitation existing, is reported by Dr. John Guiteras in the *Philadelphia Medical News*, Nov. 14, 1885.

An aortic murmur with the second sound of the heart—*i. e.* diastolic, heard usually at the base, but in most cases having its maximum a little below the base, on or near the left margin of the sternum—is called an *aortic regurgitant murmur.* It represents insufficiency of the aortic valve, and consequent regurgitation from the aorta into the left ventricle. This murmur may be more or less loud and prolonged; it is generally soft, but sometimes rough, and it may be musical. According to Dr. Balthazar Foster, if the murmur be heard at the apex of the heart, it denotes that the insufficiency is due to a lesion of the posterior segment of the aortic valve.

A murmur is sometimes produced by the backward current of blood in the aorta, the aortic valves being sufficient; that is, regurgitation into the ventricle not taking place. This is a very short murmur, preceding and ending with the second sound. It is heard over the site of the aorta in the second right intercostal space, close to the sternum. It may be called an *aortic prediastolic murmur.*

The aortic direct and regurgitant murmurs not infrequently exist in combination, but either may be present without the other. Aortic murmurs coexist with mitral murmurs in the cases in which mitral and aortic lesions are associated.

Tricuspid Murmurs.—Tricuspid lesions are comparatively rare, and hence a murmur referable to this orifice is infrequent. A *tricuspid regurgitant murmur* occasionally exists, representing regurgitation from the right ventricle to the right auricle, with, perhaps, hypertrophy of the ventricle. It occurs with the first sound, and is to be distinguished from a *mitral regurgitant murmur* by its being either limited to, or having its maximum of intensity at, the right inferior border of the heart—namely, near the ensiform cartilage. It is liable, without care, to be confounded with a mitral regurgitant murmur. A tricuspid direct murmur is exceedingly rare, owing to the fact that lesions analogous to those which give rise to a mitral direct murmur are infrequent at the tricuspid orifice.

Pulmonic Murmurs.—A *pulmonic direct murmur* is not of very infrequent occurrence. It is heard with the first sound of the heart, at or above the base, over the pulmonary artery—namely, in the second intercostal space, near the left margin of the sternum. It is to be distinguished from an aortic direct murmur by its situation, as just stated, coupled with its non-transmission into the carotids. It represents pulmonic lesions when it is not inorganic or anæmic, as it not infrequently is. The circumstances which render it probable that it is inorganic are the same as when the question relates to the organic or inorganic character of an aortic direct murmur.

By means of the foregoing murmurs the valvular lesions are recognized and localized, but it is important to bear in mind that neither the extent nor gravity of lesions is determined by the murmurs. Whether the direct murmurs be produced by lesions which involve obstruction or not cannot be determined by the study of the murmurs alone; and whether the regurgitant murmurs proceed from much or little regurgitation must be ascertained by other evidence than that which the murmurs afford. The intensity of the murmurs is no proof of the gravity of the lesions; slight or innocuous lesions may give rise to loud murmurs, and grave lesions may be represented by feeble murmurs. Nor does the character of the sound as regards softness, roughness, or a musical intonation furnish any definite information respecting the extent or gravity of the lesions. With reference to this important point of investigation, therefore, evidence is to be sought for elsewhere. Symptoms relating to the circulation, which have been considered under the head of the Clinical History, shed light upon this point, but in addition other physical signs are to be taken into account.

A comparison of the sounds of the heart, as produced separately at the aortic and pulmonic orifice, is one source of information. This comparison is made by listening successively to the sound as heard in the right and in the left second intercostal space near the margin of the sternum, the sound in the right side emanating from the aortic, and the sound of the left side from the pulmonic, orifice. If aortic lesions be indicated by the presence of the aortic direct or regurgitant murmur, and the lesions be of such a character that the aortic valve is impaired, the second sound, as produced by this valve, will be more or less weakened, or perhaps extinguished, and the alteration is ascertained by a comparison with the pulmonic second sound. If, on the contrary, the aortic sound be unimpaired, it may be inferred that the aortic valve is intact. A comparison of the aortic and the pulmonic sound is not less useful in the cases in which mitral lesions are shown to exist by the mitral direct or the mitral regurgitant murmur. If there be much obstruction or regurgitation, or both, at the mitral orifice, the aortic sound will be weakened by the diminished amount of blood propelled into the aorta with each ventricular systole; and if the mitral obstruction or regurgitation, or both, have led to hypertrophy of the right ventricle, the pulmonic sound will be intensified by the greater force with which the blood is propelled into the pulmonary artery by the systole of the right ventricle. Thus, by means of this application of auscultation useful information is obtained respecting the extent or gravity of the lesions, the existence and situation of which are revealed by the endocardial murmurs.

Further evidence of the extent or gravity of valvular lesions relates to enlargement of the heart. Enlargement of the heart is an effect of valvular lesions, and is proportionate to the amount and duration of the obstruction and regurgitation which the valvular lesions occasion. Hence, the degree of enlargement is a criterion of the extent and gravity of the valvular lesions. The question then arises, How are the existence and degree of enlargement of the heart to be ascertained? The altered situation of the apex, if not attributable to extrinsic causes, is evidence both of the existence and the degree of enlargement. The first effect of enlargement is to carry the apex to the left of its normal situation within the linea mammillaris. The next effect is to lower its situation to the sixth, seventh, or eighth intercostal space. In proportion as the apex is removed without the left nipple and lowered, is the amount of enlargement. The situation of the apex is to be determined by the eye or touch, or, if it can neither be seen nor felt, by finding the point where the systolic sound has its maximum of intensity. Other evidence of enlargement is obtained by percussion. The dulness within the area known as the superficial cardiac space, or the space in which the heart is uncovered of lung, is increased in degree and in extent in proportion as the heart is enlarged. The left border of the heart is found by percussion to fall one, two, or three inches without the left nipple, the situation of the right border and the base of the heart being in most cases but little changed.

Having determined the existence of enlargement and its extent, it remains to ascertain the kind of enlargement; that is, whether hypertrophy or dilatation predominates. The most important signs in this discrimination are furnished by palpation. If either the apex-beat or additional impulses in the intercostal spaces above the apex be felt by the hand to be abnormally strong, and especially if there be a heaving elevation of the præcordia with the ventricular systole, hypertrophy predominates. The apex-beat under these circumstances may be diffused, owing to the globular form which the heart may assume when enlarged; the augmented power of the heart's action is then to be appreciated by the impulses above the apex or by the præcordial heaving. On the other hand, if dilatation predominate the cardiac impulses, if felt at all, are feeble. Auscultation also furnishes signs of importance in this discrimination. The systolic sound of the heart over the apex is loud, prolonged, and booming in proportion as hypertrophy predominates, and on the contrary the sound is short and valvular in quality if dilatation predominate. Attention to these points, in connection with the symptoms, will enable the physician to judge of the kind of enlargement to which the valvular lesions have given rise.

PROGNOSIS.—Valvular lesions not involving either obstruction or regurgitation may remain innocuous for an indefinite period. The physician should be careful not to attach undue importance to the presence of one or more of the organic murmurs. These are frequently discovered in examinations of the chest when patients complain of no symptoms referable to the heart, and in persons who suppose themselves to be in perfect health. If the lesions be accompanied by enlargement of the heart, obstruction or regurgitation, or both, may be inferred, and the lesions are not innocuous; yet so long as the enlargement is exclusively or mainly hypertrophic, serious evils directly attributable to the cardiac lesions rarely occur. The patient under these circumstances, as a rule, simply suffers more or less inconvenience. The suffering and danger, as already stated, depend chiefly on the weakness arising from predominant dilatation of one or more of the cavities of the heart. The progress of enlargement is generally slow, and it is not uncommon for patients affected with valvular lesions, together with more or less hypertrophy, to live many years, and even to old age.

The SYMPTOMS which denote danger, immediate or not remote, differ according to the seat of the valvular lesions. In connection with mitral obstructive and regurgitant lesions habitual and considerable dyspnœa, if referable to the cardiac affection—that is, not dependent on a coexisting affection, such as asthma—is evidence that the fatal termination is not very far distant. The supervention of general dropsy generally shows that the end is near at hand. Yet sometimes, under judicious management, the dropsy diminishes or disappears, and life is prolonged for a considerable period. Death takes place suddenly in some cases in which the valvular lesions are exclusively mitral;[1] but, as a rule, if life be not cut off by some intercurrent affection, the patient dies after a period of suffering more or less prolonged. In cases of aortic lesions involving obstruction and regurgitation, especially the latter, danger is indicated by the occurrence of paroxysms characterized by great præcordial distress and by a sense of impending death, the action of the heart being irregular or tumultuous. Sudden death is liable to occur in paroxysms of this kind, the heart being paralyzed by over-distension of the left ventricle. Sudden death sometimes occurs in cases of aortic lesions when little or no habitual inconvenience from the cardiac affection had been experienced and the existence of any affection of the heart was perhaps unknown. It is rare for lesions exclusively aortic to lead to general dropsy. The immediate or proximate danger is to be determined by the amount of obstruction or regurgitation, especially the latter, in connection with the degree of enlargement, particularly enlargement by dilatation.

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[1] My colleague, Prof. Janeway, has met with a case in which, a thrombus having formed in the left auricle, a portion, becoming detached, plugged the mitral orifice and caused sudden death. This was diagnosticated before death.

In cases of tricuspid and pulmonic lesions general dropsy is an event denoting a degree of weakness of the right side of the heart from dilatation, which will be likely to lead before long to a fatal result.

In all cases of valvular lesions associated with much enlargement, especially if the enlargement be by dilatation, the prognosis is unfavorable. The patient will die sooner or later with the cardiac affection if life be not destroyed by some other disease, the occurrence of a fatal result being only a question of time and the duration varying greatly in different cases.

Death may occur in consequence of certain local affections or accidents incident to the cardiac affection. Thus, pulmonary œdema and pneumorrhagia, incident to mitral lesions, may be the immediate cause of a fatal result. Cerebral hemorrhage may be favored by dilatation of the right side of the heart and by hypertrophy of the left ventricle. The loss of blood by hemorrhage from mucous surfaces may in like manner lead to a fatal termination. The effects of emboli derived from cavities of the heart are to be included in this category.

TREATMENT.—The treatment in cases of valvular lesions has reference, *first*, to the condition of the heart as respects these lesions and coexisting enlargement; *second*, to symptoms or events incidental to the cardiac affection; and *third*, to the general condition of the patient, the condition of the blood especially claiming attention.

Valvular lesions, so long as they are either innocuous or have not led to enlargement of the heart, claim attention only with regard to preventing or retarding their progress; and for this end the strain upon the valves occasioned by the excessive action of the heart is, as far as possible, to be avoided. Very active muscular exertions, and the excitement of the circulation produced by the abuse of alcoholic stimulants and by violent mental emotions, will be likely to hasten or increase the damaging effects of the lesions. It is, however, by no means necessary to enjoin great restriction in the manner of living in these cases; it is sufficient if the habits of life be governed in all respects by prudence and moderation. It is not advisable, under these circumstances, for persons to consider themselves as invalids. If, as is frequently the case, the existence of the lesions be discovered accidentally or casually, it may not be always judicious to communicate the fact to the patient. If a medical opinion be not requested, and proper hygienic management can be secured without directing the patient's attention to the heart, much needless anxiety is sometimes spared. If, however, information be desired, it should not be withheld, but explanations may be made which will tend to prevent an over-estimate of immediate danger.

If cardiac symptoms have led the patient to apply to the physician, more or less enlargement will probably be found associated with the lesions. Let it be supposed that the enlargement which exists is mainly due to hypertrophy; it is not desirable to endeavor to diminish this hypertrophy. Inasmuch as whatever obstruction or regurgitation the lesions occasion must continue, the abnormal growth of the walls of the heart is conservative; the comfort and safety of the patient depend upon it, and measures which tend to weaken the heart will do positive harm. On the contrary, measures to maintain the muscular tone and vigor of the heart are indicated. The diet should be nutritious. The appetite and digestion, if impaired, should be improved by tonic remedies. Moderate open-air exercise is to be encouraged, very active exertions being interdicted. In short, the object is in every way to invigorate and strengthen the system.

If dilatation predominate, the measures indicated are still those which tend to give as far as possible, tone and vigor to the heart. The labor of the circulation may be in some degree diminished by restricting the quantity of liquids ingested, redundancy of the mass of blood being in this way prevented. Especial care, however, is to be taken not to impoverish the blood. The diet is to be highly nutritious, although as dry as is compatible with comfort; tonic remedies are to be employed, and moderate exercise out of doors is still advisable. In proportion as the powers of the system are lowered the heart is weakened and dilatation favored. The object of management is, by strengthening and invigorating the system, to retard the progress of dilatation.

A recent writer upon this subject, Oertel, advocates systematic exercise, and especially mountain-climbing, as a means of increasing the muscular power of the heart.[1] There is foundation for this in both reason and experience. Quietude carried too far is perhaps likely to do more harm than over-exertion. I have known of instances in which pretty active exercise afforded notable relief from symptoms referable to valvular lesions and enlargement of the heart. In a case under my observation exercise by means of the "health-lift" was signally useful. Patients exchanging habits of activity for complete rest are likely to become rapidly worse. Extremes in either direction are to be avoided. This may be done by enjoining such an amount of exercise as can be taken without any sense of discomfort. Within that limit exercise is not only allowable but beneficial.

Thus far, the treatment has reference to the condition of the heart. Of the symptoms incident to the cardiac affection, irregularity, undue frequency, and feebleness of the heart's action call for treatment. The appropriate remedies are those which diminish the frequency, restore the rhythm, and strengthen the action of the heart. Digitalis is an invaluable remedy, regulating the heart's action without diminishing, but, on the contrary, increasing, the power of the ventricular contractions. This remedy is therefore particularly suited to cases in which the action of the heart is rendered irregular and feeble by dilatation, and it is not suited to cases in which the symptoms and signs denote hypertrophy. The fact that the diminished frequency of the heart's action is due to a lengthening of the diastole renders important a cautious use of the remedy in cases of free aortic regurgitation; yet a considerable increase of the power of the ventricular contraction may be a gain overbalancing any risk of an over-filling of the ventricular cavity from an increased diastolic rest. The pulse, the apex-beat, and the intensity of the systolic sounds of the heart, together with the subjective symptoms, enable the physician to judge of the safety and utility of the remedy in these cases. There is little ground for the traditional notion that digitalis is peculiarly a cumulative remedy, the patient being liable to a sudden manifestation of its toxic effects. This remedy is specially useful in cases of enlargement dependent on mitral regurgitant lesions. Digitalis in moderate doses (a grain or two of the leaves, two or three drachms of the infusion, from ten to fifteen minims of the tincture, or one-sixtieth of a grain of digitalin twice or thrice daily) is often well tolerated, and may be continued with advantage for a long period.

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[1] Vide *Handbuch der Allgemeinen Therapie der Kreislaufs-Störungen*, Leipsic, 1884.

Not infrequently, however, after a time it causes gastric disturbance, which is sometimes avoided by changing the form of the remedy or by the addition of a carminative. Generally, it is advisable to discontinue it when its good effects have been obtained, resuming it according to circumstances.

Aconite is a useful remedy when the action of the heart is rapid, and it may be substituted for digitalis if the latter be not tolerated. It is not, however, a substitute for digitalis in respect of a tonic effect on the heart. It is specially applicable to cases in which disturbance of the heart's action is due to functional disorder superadded to lesions, and when the action is not notably weakened by dilatation.

Belladonna, as a neurotic sedative, is sometimes of service, in this respect belonging in the category of other sedatives—hyoscyamus, hydrocyanic acid, gelsemium, etc. A belladonna plaster over the præcordia has a good effect. I have known it to dilate the pupil. Nauseant sedatives, as veratrum viride, are not appropriate. The latter lessens the frequency, but also the strength, of the heart's action. Nux vomica or strychnia appears, either directly or indirectly, to have a tonic influence upon the heart. Caffeine is a remedy of much utility. It answers well in some cases as a substitute for digitalis. Convallaria and the cactus grandiflorus are remedies which within late years have been introduced as cardiac tonics, increasing the force and diminishing the frequency of the heart's action. They are to be regarded as succedanea of digitalis, and trial of them may be made, especially if digitalis fail or be not well tolerated. There is considerable discrepancy in the testimony of different observers respecting the value of these remedies. They are inferior to digitalis when this remedy is well tolerated. The expectations attending their introduction have not been sustained, but they are valuable in some cases, and they should not be allowed to fall into disuse.

As palliatives for cardiac dyspnœa, ammonia, the ethers, and dry cups applied to the chest are serviceable. Notable relief is obtained often by a brisk hydragogue purgative from time to time. So far from inducing weakness of the heart's action or general prostration, the effect of this measure is in these cases the reverse. Bronchorrhœa is not to be checked by opium, but small doses, administered hypodermically or otherwise, not infrequently afford relief, and are not hurtful if the patient do not suffer from the after-effects of this drug. The objections entertained by many physicians to the use of opium for the relief of cardiac dyspnœa is unfounded. If well tolerated, the effect of opium is that of a heart-tonic.

General dropsy is an event requiring treatment. Diuretic remedies should be first tried, and if these prove inoperative hydragogue cathartics are to be resorted to. Of these, elaterium is the most efficient. Given in small doses, this remedy may often be continued for a long period without producing depression. The bitartrate of potassa with jalap and podophyllin are valuable hydragogues. The Epsom salts answer exceedingly well in many cases; they are reliable, in general well tolerated, and the amount of hydragogue effect is easily regulated. If only a mild hydragogue effect be desired, the bitter waters—Pulna, Friedrichshall, and Hunyadi Janos—may suffice. In the majority of cases only partial and temporary relief of the dropsy is procured, but in some cases it is completely removed, and it may not return for a considerable period. Diuretic remedies sometimes act very efficiently. I have known the infusion of parsley-root to act with wonderful power as a diuretic remedy. If the anasarca be so extreme that the limbs become enormously distended, a great number of punctures may be made with the point of a pin, not very sharp, the punctures being so slight as not to draw blood, and through them a large amount of liquid will sometimes escape, affording for a time marked relief. Vesications or cracks in the integument of the lower limbs in some cases occur as a consequence of the distension, giving exit to an abundant flow of serum, with relief. Incisions or deep punctures are attended with a risk of gangrene. In the treatment of the dropsy, restriction of the amount of ingested liquid, as far as is compatible with comfort, is important.

A highly important part of the treatment relates to the general condition of the patient, especially to coexisting anæmia. Anæmia is not infrequently associated with the lesions under consideration, and as a consequence functional disorder of the heart is superadded to the disturbance caused by the lesions. Irregular action of the heart or excessive action, dyspnœa, and even dropsy, may be due to the superadded functional disorder. The error is not infrequently committed of attributing the symptoms in such cases exclusively to the lesions, and forming a prognosis more unfavorable than the latter actually warrant. By removing the anæmia the symptoms are relieved, and the patient may even seem to recover completely from the cardiac affection. Anæmia existing in connection with valvular lesions claims appropriate treatment—namely, removal of the cause if it be apparent; as, for example, lactation, the employment of chalybeate tonics, with nutritious diet and other hygienic measures. Exclusive of anæmia, the general condition of the patient should be improved and maintained at the highest possible point of strength and vigor. As a rule, in proportion as alimentation, digestion, assimilation, and nutrition are approximately normal, the grave effects of heart lesions are less likely to occur; in other words, the lesions are best tolerated. The importance of a nutritious diet, remedies to promote digestion if it be defective, and hygienic agencies conducive to the welfare of the body cannot be too strongly enforced as promoting tolerance and prolonging life.

CHAPTER IV.

HYPERTROPHY AND DILATATION OF THE HEART.—ATROPHY OF THE HEART.—OBESITY AND FATTY DEGENERATION OF THE HEART.—PARENCHYMATOUS DEGENERATION OF THE HEART.—NEW GROWTHS AND PARASITES IN THE HEART.—RUPTURE OF THE HEART.—THROMBOSIS OF THE HEART.—EMBOLISM OF THE PULMONARY ARTERY.

Hypertrophy and Dilatation of the Heart.

THESE conditions will be considered together, inasmuch as they are usually combined with each other. They may affect the whole heart or certain sections of it, and are therefore classified as total and partial hypertrophies and dilatations. The most important classification, however, is that based on the capacity of the heart-cavities. In *simple hypertrophy* the cavity is of normal size, in *eccentric hypertrophy* it is dilated. In *simple dilatation* the muscular wall is of normal thickness, in *hypertrophic dilatation* the wall is hypertrophied, in *atrophic dilatation* it is thinned. Hypertrophic dilatation is often employed in the same sense as eccentric hypertrophy. Rokitansky, however, proposed to apply the term eccentric hypertrophy to enlarged hearts in which the hypertrophy predominates over the dilatation, and hypertrophic dilatation—or, as he called it, active dilatation—to cases in which the dilatation is decidedly in excess of the hypertrophy. Of the different varieties, eccentric hypertrophy is by far the most frequently observed. Some writers have assumed the existence of a concentric hypertrophy, a condition in which the muscular wall is thickened and the cavity diminished in size. Great caution, however, is required in estimating the capacity of the ventricles after death. If the heart be found firmly contracted, its cavities seem smaller and its walls thicker than normal. It is probable that cases of supposed concentric hypertrophy are only those of firmly contracted normal or hypertrophic hearts. It is also necessary not to mistake a relaxed and flabby heart, such as is often found after death from acute infectious diseases, for a heart affected with dilatation. In so-called simple dilatation the heart is evidently hypertrophied, as the normal thickness of the muscular walls could not otherwise be preserved. In fact, hearts affected with dilatation and thinning of the walls—that is, with so-called atrophic dilatation—usually weigh more than normal, and are therefore hypertrophied. Simple hypertrophy is infrequent. It may be found in connection with certain forms of chronic Bright's disease.

In a given case it is important to determine the degree of dilatation in proportion to the hypertrophy, for while hypertrophy strengthens the muscular power of the heart, dilatation enfeebles it; the latter is therefore much the graver of the two conditions, giving rise to nearly all the symptoms.

If the enlargement be confined to the right side of the heart, the organ is increased chiefly in breadth and has a more rounded and less conical shape than normal. The rounded apex is formed by the right ventricle. When the left ventricle is hypertrophied the heart is elongated, the apex being lower down and farther to the left than normal, and if hypertrophy be combined with dilatation the septum ventriculorum is pushed over to the right side, encroaching upon the cavity of the right ventricle. If the whole heart be enlarged its shape is triangular with rounded angles. The name *cor bovinum* has, from ancient times, been applied to a heart enormously enlarged. The degree of hypertrophy varies much in different cases. The weight in the majority of hypertrophied hearts is between twelve and twenty ounces. Hope mentions one weighing forty-four ounces, and Stokes one which yielded the enormous weight of four pounds two ounces, avoirdupois. In determining a small amount of hypertrophy, it is necessary to take into consideration the variations in weight within physiological limits, as well as the age and general physique of the individual.

Hypertrophic heart-muscle is brownish-red in color, and firm, almost board-like, in consistence. The wall of an hypertrophied right ventricle has a peculiarly tough, leathery consistence. After incision the walls remain open and rigid. Both color and consistence will be modified if the hypertrophic muscle have undergone fatty degeneration, which is not infrequent.

There has been much discussion as to whether, in hypertrophy of the heart, new muscle-fibres are produced or the pre-existing muscle-fibres simply increased in volume. The formation of new muscular fibres and the proliferation of the muscular nuclei seem to be established. It is probable that enlargement of the muscular fibres also occurs, as was thought by the older writers.

Hypertrophy and dilatation of the heart are secondary affections, and consecutive in the majority of cases to some mechanical obstacle to the circulation. The obstacle may be seated in the heart itself or in the pulmonic or the systemic circulation.

The causes, seated in the heart, of hypertrophy and dilatation are—

1. Stenosis and insufficiency of the valves in front.
2. Insufficiency of the valves behind.

3. Chronic pericarditis and pericardial adhesions.
4. Myocarditis with or without partial cardiac aneurism.
5. New growths in the heart.

Weakening of the cardiac muscle from parenchymatous and from fatty degeneration leads to dilatation of its cavities.

The obstruction may be in the pulmonic circulation, in which case only the right side of the heart enlarges. The most important of these causes are—

1. Pulmonary emphysema.
2. Long-continued and large pleuritic effusions.
3. Stenosis of the pulmonary artery, either as a primary change in the artery itself or by pressure from without, as by aneurisms of the aorta, tumors, etc.
4. Curvatures of the spine, causing compression of the lungs.

Obstructions in the aortic system causing hypertrophy and dilatation are—

1. Atheroma of the aorta and larger arteries. Many cases of senile hypertrophy are referable to this cause.
2. Narrowing of the calibre of the aorta, either as a congenital condition or by pressure from without, as by tumors, etc.

It is important to notice that, contrary to former belief, aneurism of the aorta does not lead to hypertrophy of the left ventricle. This has been demonstrated by the investigations of Axel Key.[1] Aneurism of the aorta alone does not tend to increase the tension in the left ventricle. Very often, however, an atheromatous condition of the aortic valves or of the general arterial system is associated with aneurism, and this may be the cause of ventricular hypertrophy.

A well-recognized form of cardiac hypertrophy is that which is secondary to certain forms of chronic Bright's disease, particularly to the small, granular kidneys. The explanation of this is not thoroughly understood. It will be referred to under the head of Bright's Disease. Suffice it to say here that it is probably due to some resistance to the blood-current through the small vessels. This form may be simple hypertrophy, but it is usually eccentric. All of the other forms of cardiac hypertrophy are eccentric. An index to the degree of distension to which the ventricular cavity is subjected is afforded by the condition of the papillary muscles. Thus, in aortic insufficiency, which is attended with great increase of pressure in the left ventricle, the papillary muscles of this ventricle are found elongated, flattened, and in part fibrous, especially at the origin of the chordæ tendineæ; in the simple hypertrophy of Bright's disease, on the other hand, the papillary muscles are enlarged and rounded.

The name primary or idiopathic hypertrophy of the heart has been given to cases in which the disease has been attributed to habitual over-exertion of the muscular system and to repeated nervous excitation of the heart. DaCosta, Peacock, Seitz, Fraentzel, Allbutt, and others have called special attention of late years to cardiac hypertrophy and dilatation referable to over-exertion, especially in the case of soldiers who have done heavy marching in a campaign. Their observations render it probable that long-continued severe muscular strain is an important element in the production of hypertrophy and dilatation of the heart, but the infrequency with which these affections occur under the conditions named leads to the inference that an additional factor, not yet understood, enters into their causation.

The hypertrophy without valvular lesion which has been sometimes observed in cases of Graves's or Basedow's disease (exophthalmic goitre) may be referred to the prolonged frequency of the heart's action.

Hypertrophy and dilatation of the heart are sometimes found as apparently idiopathic conditions when they cannot be referred to any cause. These cases are sometimes described under the name weakened heart.

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[1] Key, *Nordiskt med. Arkiv*, Bd. 1, 1870.

The SYMPTOMS of hypertrophy are referable to the increased power of the heart's action. Hypertrophic enlargement of the left ventricle leads to an abnormal force of the current in the systemic arteries; the pulse is strong and full, the face flushed, active cerebral congestion is favored, and certain symptoms, such as pain, vertigo, tinnitus aurium, are attributable to the latter. The patient feels the powerful action of the heart, but he becomes accustomed to it, and it may occasion but little annoyance.

If the hypertrophy have been induced by an impediment to the circulation, it is conservative, as when it proceeds from valvular lesions, and is not, therefore, to be regarded as an evil. It may, however, lead or contribute to serious accidents. It is intelligible that hypertrophy of the left ventricle without valvular lesions (these being, in a measure, protective) may favor cerebral hemorrhage in cases in which the arteries of the brain have become weakened from degenerative changes. In like manner hypertrophy of the right ventricle may contribute to pulmonary hemorrhage and give rise to œdema of the lungs.

The DIAGNOSIS of hypertrophy must rest on physical signs, which are the same as when the hypertrophy is associated with valvular lesions. The fact of enlargement and its degree are determined by the altered situation of the apex-beat, together with the enlarged area of the superficial and the deep cardiac space. That the enlargement is due, either exclusively or mainly, to hypertrophy is determined by the abnormal force of the impulses of the heart, and by the prolongation, intensity, and booming character of the first sound.[1] The absence of endocardial murmurs is the basis of the conclusion that the enlargement is not connected with valvular lesions.

Hypertrophy, when conservative or compensatory, of course does not claim treatment. If it be congenital or there be sufficient grounds for considering it otherwise than conservative, measures to prevent further growth and to obviate accidents are called for. Bloodletting and other measures which impoverish the blood are not indicated. Anemia, by rendering the heart unduly excitable, involves additional inconvenience and danger. Excessive action of the heart may be moderated by tranquillizing remedies, such as aconite, hydrocyanic acid, and belladonna. In the cases of hypertrophy disconnected from valvular lesions observed by DaCosta among soldiers, marked benefit was obtained by the use of aconite, one or two drops of the tincture being given twice or thrice daily, and continued, without any increase of the dose, for months. The diet should be sufficiently nutritious, but unstimulating. Alcoholics are to be interdicted. Liquids should be taken sparingly. Active muscular exercise and emotional excitement are to be avoided.

Dilatation of the cavities of the heart may accompany hypertrophy without, as well as with, valvular lesions. If there be causes giving rise to hypertrophy which continue after the morbid growth has reached its limit, dilatation of necessity follows, and in time will become predominant; but in most cases excessive dilatation proceeds from weakness of the muscular walls, arising from fatty degeneration and other causes. The muscular walls may yield to distension and dilatation may take place when there is no abnormal impediment to the circulation; and dilatation, however induced, tends progressively to increase.

The SYMPTOMS of predominant dilatation are referable to weakness of the heart's action. This weakness is shown by feebleness of the pulse, coolness and cyanosis of the surface, and if the right cavities become considerably dilated general dropsy ensues. Fatal syncope may occur from an over-accumulation of blood in the cavities in cases in which the organ is greatly enfeebled by dilatation. The physical signs show enlargement of the heart; and that the enlargement is due to predominant dilatation is shown by the feebleness of the cardiac impulses and by the weakness, shortness, and valvular quality of the first sound as heard over the apex. The absence of valvular lesions is inferred from the absence of endocardial murmur.

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[1] Vide preceding chapter.

The indications for TREATMENT are the same as when the dilatation is in association with valvular lesions, the object, in general terms, being to endeavor to improve the tone and vigor of the muscular walls.

Atrophy of the Heart.

Simple wasting or atrophy of the heart is found in emaciated persons as part of the general marasmus. Diseases attended with prolonged cachexia, notably carcinoma and chronic pulmonary tuberculosis, are likely to lead to atrophy of the heart. An abnormally small size of the heart has been observed as a congenital condition in connection with imperfect development of the whole circulatory system, frequently combined with arrested sexual development. Senile marasmus may be accompanied with cardiac atrophy, but not infrequently it is associated with hypertrophy in consequence of atheroma of the large arteries. Pressure upon the heart from without, as by tumors, pericardial and pleuritic effusions, has also been adduced as a cause of atrophy. Atrophy of the left ventricle results from uncomplicated stenosis of the mitral orifice, as has been already stated. The heart may be reduced to four ounces in weight. The coronary vessels, taking no part in the atrophy, appear tortuous and prominent upon its surface. Atrophied hearts are sometimes covered with a considerable amount of adipose tissue, which may obscure the actual reduction in size of the myocardium.

Atrophy of the heart usually presents itself in the form of so-called brown atrophy. The wasted muscle has a brownish color, and under the microscrope the muscular fibres are found to contain little granules of yellowish-brown pigment, accumulated especially about the nuclei. A similar pigmentation, but in less degree, is not uncommon in normal hearts.

In a clinical point of view this lesion is unimportant. It occurs under circumstances which divest it of evil consequences, if, indeed, it be not a conservative condition.

Fatty Degeneration—Obesity of the Heart.

By *obesity* of the heart (called also fatty infiltration, deposition, or growth) is understood the accumulation of adipose tissue upon the surface of the organ and between the muscular fibres. This condition is met with in corpulent subjects, and sometimes without general obesity, especially in the bodies of old persons and drunkards. The fat accumulates first in the sulci in the course of the coronary vessels, and it may eventually cover the entire right and most of the left ventricle. A moderate accumulation of fat upon the outside of the heart cannot be considered of any importance; but when the fat is excessive in amount, and especially when it penetrates deeply into the myocardium, the muscular fibres become atrophied from compression and the heart is enfeebled. It will be observed that in obesity of the heart the muscular fibres suffer only in so far as they are compressed.

In *fatty degeneration* the muscular substance is metamorphosed into fat, which appears in a granular form in the muscular fibres. By Quain's fatty degeneration is understood this form. Fatty degeneration may attack a heart previously diseased, or the organ may have been previously healthy. In the former case the degeneration may be localized in the affected portion of the heart; in the latter instance the whole heart becomes fatty, but not of necessity in an equal degree in all parts. The fatty heart is of a pale yellowish color and is soft in consistence. The yellowish color is usually more marked in certain parts than in others. It is usually most noticeable in the inner layers of the myocardium, which are particularly liable to fatty degeneration. These layers, as seen through the endocardium, especially in the papillary muscles of the left ventricle, present a peculiar mottled appearance which is quite characteristic. The fatty degeneration may be confined to the inner part of the myocardium.

Examined under the microscope, the muscular fibres are found clouded by the presence of large numbers of granules and small globules of fat, frequently arranged in longitudinal rows within the muscular substance. The transverse striation is obscured or entirely obliterated. Some fibres are more affected than others. A small number of fatty granules is not uncommon in the cardiac muscle. When of clinical importance, the degeneration is in such amount that it can usually be recognized with the naked eye. The fatty material is undoubtedly a product of the metamorphosis of the albuminous substance of the muscle.

The parenchymatous or granular degeneration to be presently described, which accompanies many acute infectious diseases, such as typhoid, remittent, and puerperal fevers, acute yellow atrophy of the liver, etc., may be followed by a more or less extensive fatty degeneration.

Fatty degeneration of the heart, as already mentioned, is common in hypertrophied and dilated hearts, particularly during the stage of non-compensation. It may be associated with fibrous myocarditis and diseases of the coronary arteries. It is frequently present in parts adjacent to an inflamed pericardium or endocardium. General causes are important in its etiology, such as poisoning with phosphorus, arsenic, or mineral acids, and in acute infectious diseases. A most intense fatty degeneration of the heart attends certain profound anæmias, particularly pernicious anæmia and leucocythæmia.

The SYMPTOMS of fatty degeneration of the heart are due to its inability to carry on effectively the circulation. Degeneration of the left ventricle occasions feebleness of the pulse. The pulse may be irregular. It has been observed to be notably infrequent, the number of beats sometimes falling as low as 20 per minute. The infrequency of the pulse may be owing to many of the ventricular systoles being too feeble to propel the blood with sufficient momentum to be felt at the radial artery. Dyspnœa, especially on exercise, is more or less prominent as a symptom according to the feebleness with which the circulation through the pulmonary circuit takes place; and this symptom is more marked when the right ventricle is affected. Paroxysms of syncope are liable to occur, accompanied by a sense of præcordial oppression and distress. These denote distension of the cavities from an accumulation of blood. Accompanying these, paroxysms of pain referred to the præcordia, and sometimes extending to one or both upper extremities, more especially on the left side (vide Angina Pectoris), are not uncommon. The remarkable rhythmical irregularity of breathing known as the "Cheyne-Stokes respiration" is sometimes observed, but it is by no means diagnostic of this lesion.[1]

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[1] This is described by Stokes "as a form of respiratory distress consisting of a period of apparently perfect apnœa (*i. e.* absence of breathing), succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the beginning of another apnœal period."

It occurs in uræmia, in certain cerebral affections, and in connection with other cardiac lesions. Seizures resembling apoplexy, characterized by temporary loss of consciousness without paralysis, the surface being pallid and cool and the circulation extremely feeble, have been observed in persons who have subsequently died of this affection. These pseudo-apoplectic seizures in some cases have been of frequent recurrence. These symptoms are also present in cases of fibroid degeneration or myocarditis.

The signs which accompany fatty degeneration are—feebleness or absence of cardiac impulse, and weakness of the heart-sounds, more especially the systolic sound, this sound sometimes suppressed over the apex, and if heard short and valvular like the second sound. These signs are equally present as denoting weakness of the heart from dilatation, fibroid degeneration (myocarditis), and obstruction of the coronary arteries. Sex is of some weight in the diagnosis, men being the subjects much oftener than women. The age of the patient is to be considered. Fatty degeneration rarely occurs in the male under fifty, and in the female under forty years of age. The tendency to obesity has not great weight in the diagnosis, inasmuch as fatty degeneration of the heart occurs in lean subjects as well as in those prone to corpulency. As regards the arcus senilis, clinical observation has abundantly shown that this fatty change in the cornea occurs without degeneration of the heart, and *vice versâ.*

To give rise to symptoms which point to the heart as the seat of disease the fatty degeneration must be considerable. Even if considerable, it may not occasion sufficient weakness to give rise to any notable symptoms. More or less degenerative change is not infrequently found after death in cases in which it had not been suspected during life.

Causal indications are often not determinable with precision in individual cases. Clinical observation appears to show that defective nutrition is concerned in the causation. The lesion is found in those who are underfed as well as those who are overfed. General anæmia, especially pernicious anæmia, is causative. The lesion is not of frequent occurrence in connection with simple or benign anæmia. Defective nutrition of the heart from local causes in not a small proportion of cases is, intelligibly, an etiological condition. It is incident to obstruction of the coronary arteries by calcification or embolism, and to defective circulation as a consequence of impaired recoil of the aorta from atheroma or aortic insufficiency. It participates in the acute degeneration of other organs in poisoning by phosphorus, and, it is said, also by mineral acids. The pathological character of this lesion has been considered in Part I. (Vide p. 54.)

The objects of treatment are to develop the muscular vigor of the heart to the extent of its capability for improvement in this respect. The diet should be nutritious, consisting of animal food in as large proportion as is compatible with the appetite and digestion. Fatty food is to be interdicted. Farinaceous and saccharine articles should form a moderate proportion of the diet. Alcohol should be taken in moderation, if at all. Moderate exercise out of doors is important; it should not be carried to the extent of overtasking the heart, but may be taken within the limits of comfort; that is, so as not to occasion hurried circulation, præcordial oppression, or dyspnœa.

The hygienic is far more important than the medicinal treatment; yet remedies may be useful. Tonic remedies are generally indicated. The appetite and digestion, if impaired, are to be improved as much as possible. If the patient be anæmic, this condition claims appropriate treatment. Coexisting disorders of any kind are to be relieved. The symptomatic effects of weakness of the heart will claim palliative measures. These measures are the same as when the weakness proceeds from dilatation.

Parenchymatous Degeneration of the Heart.

In parenchymatous as in fatty degeneration the muscular substance is clouded and the striæ are more or less obscured by the presence of a granular material; but in the former the granules are albuminous, in the latter, fatty. Granular degeneration, albuminous degeneration, and cloudy swelling are synonymous terms for this affection. The name parenchymatous inflammation is sometimes used improperly in the same sense. Many of the cases formerly described as simple softening of the heart belong in this category. The degeneration affects the whole muscular substance of the heart. The heart is paler and of softer consistence than normal. The granules of parenchymatous degeneration are paler, less glistening, and usually smaller than those of fatty degeneration, from which they can be distinguished by their solubility in acetic acid and insolubility in ether. The granules are formed by some modification of the pre-existing albuminous substance, but it is not known in what this modification consists.

Parenchymatous degeneration is found chiefly after death from acute infectious diseases, such as typhus and typhoid fevers, puerperal fever, the exanthematous fevers, erysipelas, cerebro-spinal meningitis, pyæmia, and septicæmia, also in consequence of extensive burns and of poisoning by phosphorus, arsenic, etc. It can be produced artificially in animals by exposure to high temperatures, but the influence of this cause in man has been exaggerated. Severe blood-poisoning is the most efficient cause. The degeneration may be developed with great rapidity. Proof of this is afforded by a case observed by Wagner,[1] who found this change in the heart and kidneys of a previously healthy girl who died six hours after extensive burns. Fatty degeneration is a frequent accompaniment and sequence of parenchymatous degeneration, but there is no necessary connection between the two processes. Without doubt, parenchymatous degeneration occurs in the course of many diseases which end in recovery (Part I. p. 53).

This lesion explains the weakness of the circulation out of proportion to the adynamia or general debility in certain diseases. The symptoms and signs are those which represent notable feebleness of the heart's action. As regards the signs, diminished intensity of both sounds, but especially of the first sound, characterizes the feebleness from this as well as from other lesions. These signs contraindicate depletory or debilitating measures of treatment, and, on the contrary, furnish a reason for supporting treatment in addition to the indications derived from the general condition of the system.

New Growths and Parasites in the Heart.

These have no distinctive clinical history, and therefore will receive only the briefest mention here. The most common new formations in the heart are tubercles and gummata. Miliary tubercles are frequently found in the heart in cases of acute general tuberculosis. They should be sought for especially in the endocardium of the conus arteriosus of the right ventricle. Tubercles in the myocardium may give rise to myocarditis (tuberculous myocarditis). Further than this it is not known that they give rise to any symptoms. Cardiac gummata and syphilitic myocarditis have already been referred to under the head of Myocarditis. Carcinoma, sarcoma, lipoma, fibroma, myxoma, myoma strio-cellulare, have been observed in the heart. Echinococcus cysts, and more rarely cysticercus cellulosæ, have been found in the heart.

The interesting subject of congenital malformations and diseases of the heart does not come within the scope of this work. The reader is therefore referred to works on pathological anatomy and to special treatises on the subject, including those devoted to diseases of infants, for information concerning the morbus ceruleus or blue disease, non-closure of the foramen ovale, of the ductus Botalli, of the septum ventriculorum, stenosis of the pulmonary orifice and artery, etc.

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[1] *Handbuch des Allgem. Pathol.*, Leipzig, 1876, p. 413.

Rupture of the Heart.

The most frequent cause of spontaneous rupture of the heart is necrosis with softening of a circumscribed part of the wall. This condition, which is sometimes called myomalacia, is due to complete obstruction, either by a thrombus or an embolus, of a branch of a coronary artery. Other causes of rupture of the heart are the bursting of a cardiac aneurism or of an abscess in the heart, or the extravasation of blood resulting from rupture of a branch of a coronary artery. An ulcer of the stomach has been known to perforate the heart. Rupture of the heart very rarely happens in persons less than sixty years of age. Usually it causes instant or speedy death; but if the perforation be small it may be temporarily plugged by a clot and life continue for hours and even days. I have known death to be delayed for six hours after the symptoms denoted the occurrence of the rupture. The symptoms are those which sudden compression of the heart by the accumulation of blood in the pericardium would be expected to produce, together with the loss of blood. In most cases, however, there is no time before death for the observation of symptoms. Rupture has been repeatedly observed to occur in connection with a paroxysm of præcordial pain resembling angina pectoris.[1] In a case which came under my observation, the patient, a middle-aged man, had had repeated attacks of severe angina, and the heart gave no evidence, by physical signs, of disease. Death occurred suddenly, and a rupture was found of the left ventricle. The rupture took place within a circumscribed space in which there was advanced necrosis, the muscular wall being much softened. A branch of the left coronary artery leading to the necrosed space was completely occluded by a thrombus, and there was partial atheromatous occlusion of both coronary arteries. Except in the necrosed space the heart had not undergone fatty degeneration.

Thrombosis of the Heart.—Embolism of the Pulmonary Artery.

It is important to distinguish ante-mortem thrombi from the coagula formed after death. At autopsies the right ventricle and auricle are usually found to contain coagulated blood. These coagula are often in great part devoid of blood-coloring matter, and they constitute the so-called decolorized post-mortem clots. The blood in these cases coagulates so slowly after death that the red blood-corpuscles have time to sink; hence the upper layers of the clot consist mostly of fibrin and white blood-corpuscles, the latter being of lighter specific gravity than the red corpuscles. These post-mortem clots are yellowish-white, succulent, loosely attached to the heart-walls, and they send offshoots into the pulmonary artery and its branches. They receive the imprint of the valves and other irregularities with which they lie in contact. They have no clinical significance.

Fibrinous deposits may form in the heart during life. The usual causes of their formation are the same as those of the production of thrombi in general—namely, slowing of the circulation and alterations of the inner layer of the vessel. The presence of fibrinous deposits on the inflamed and necrotic endocardium, especially of the valves, in acute and chronic endocarditis, has already been mentioned (p. 321). Cardiac thrombi are liable to form in consequence of the slow circulation attending excessive dilatation of the heart and great weakness of the heart. These are called marantic thrombi. They form especially in the recesses of the heart, where there is a natural obstacle to the propulsion of the blood. They are to be sought, therefore, particularly in the appendices auricularum and between the columnæ carneæ, especially on the right side of the heart. They also form in cardiac aneurisms. When these thrombi attain a considerable size and project into the heart-cavities, they are often called cardiac polypi. These ante-mortem thrombi are of a whitish, yellowish, or reddish-gray color, dull in appearance, adherent to the heart-wall, often friable in consistence, laminated in structure, and with the free surface usually rounded (*végétations globuleuses* of Laennec). The central portion of the older thrombi is often broken down into a creamy mass which was formerly mistaken for pus, and such thrombi were called puriform. The creamy mass consists chiefly of granular matter. The thrombus itself is composed mostly of fibrin, blood-plates, and white blood-corpuscles, and belongs to the so-called white thrombi. Cardiac thrombi are usually small in size, but they may take up the greater part of one of the heart-cavities, and may even extend from one cavity into another. Organization of cardiac thrombi has been observed, but it is very rare.

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[1] For an analysis of 24 cases of spontaneous rupture of the heart by Barth, vide *Archives générales de Médecine*, Paris, 1871. For an analysis of over 100 cases by Quain, vide *The Lancet*, 1870.

The formation of thrombi of considerable size within the right ventricle (heart-clot) has been referred to especially in connection with acute lobar pneumonia or pneumonic fever, and also as occurring in connection with other diseases. They produce symptoms attributable to obstruction at the right side of the heart—namely, dyspnœa and cyanosis, with notable smallness and feebleness of the pulse. These symptoms develop suddenly or more or less rapidly, and are generally the forerunners of speedy death. The heart-sounds are weakened, and it may be practicable to determine that the first sound is due to the tension of the mitral valve, without the participation of the tricuspid valve. A tricuspid systolic murmur may be found which had not existed previously. Excluding pulmonary conditions which would account directly for the foregoing symptoms, and taking the latter in connection with physical signs, the diagnosis may sometimes be made with much positiveness.

Absolute rest, sustaining measures of treatment, and saturation of the blood with ammonia constitute, with our present knowledge, the measures of treatment to be pursued. The history of some cases appears to show recovery, but there is always room for doubt in these cases respecting the diagnosis.

The fibrinous deposits upon vegetations or roughened spots, occurring generally within the left ventricle, are chiefly important from their liability to become detached and to give rise to emboli. Heart-clots of large size may form ante-mortem in the left auricle when largely dilated as a result of valvular lesions. They may also form in the left auricle when weakened by dilatation or lesion of the muscular walls.

Embolism of the main trunk of the pulmonary artery may be here noticed as a cause of sudden or rapid death. The embolus may be derived from a thrombus formed within the right ventricle or auricle, or from a thrombus formed in a large vein, most frequently the femoral vein. The symptoms are sudden, urgent dyspnœa, not explicable by physical signs, cyanosis, and coldness of the extremities, the pulse becoming rapidly small and feeble and soon extinct.

Sudden and complete obstruction of one of the primary divisions of the pulmonary artery may take place without causing death or giving rise to permanent notable inconvenience, as has been demonstrated in cases under my observation.[1]

CHAPTER V.

FUNCTIONAL DISORDER OF THE HEART.—EXOPHTHALMIC GOITRE.—ANGINA PECTORIS.—THORACIC ANEURISM.

Functional Disorder.

BY the term functional disorder is meant disturbed action of the heart not dependent on either inflammation or structural lesions. The beating of the heart is sometimes abnormally intense. The patient is conscious of its violent action, as when it is temporarily excited by fear or some other strong mental emotion. This is commonly known as palpitation. The sense of violence may be a subjective symptom only, the force of the heart's action not being actually increased; but generally the action is at the same time accelerated and irregular. The irregularity is represented by the pulse, and is felt when the hand is placed over the præcordia. The patient is also painfully conscious of it; the organ appears sometimes to perform a rolling or tumbling movement, and a sensation is described as if the heart were in the throat. Intermittency of the heart's action is another feature in certain cases. The action of the heart is for an instant arrested; one, two, or three beats are lost, and generally irregularity of action precedes and follows the intermissions. Of these the patient is vividly sensible, and the feeling is that fatal suspension of the heart's action may be liable to take place. These rhythmical aberrations may be accompanied with either violent or feeble action of the heart. The varieties of functional disorder occur in paroxysms which are very variable as regards severity, frequency of occurrence, and duration.

In contrast to paroxysmal disorders, there is an affection in which the heart beats regularly, but with great increase in frequency. The number of beats per minute may be increased to 150 or even 200. The increased frequency may persist for days, weeks, and even months. This form of disorder has been named *tachycardia* (*ταχύς καρδία*). I have met with an instance of such an increase in the frequency of the heart's action, together with feebleness, that it was difficult to count the pulse, the disorder persisting for several days, accompanied by anorexia and a degree of pallor and prostration which seemed to denote impending death, recovery taking place, and examinations prior and subsequent to the attack showing no cardiac lesions.

An occasional form of disorder is characterized by every alternate ventricular systole being so feeble as not to be appreciable by a radial pulse, the action of the heart being more or less rapid. On auscultation, the heart-sounds are found alternately loud and feeble. I have known this form of disorder to persist for several weeks. This is liable to be mistaken for reduplication of the heart-sounds, there being four sounds to each radial pulse. The error is avoided by comparing the radial with the carotid pulse. The alternate feeble systoles are always strong enough to cause an appreciable pulsation of the carotids.

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[1] Vide *Clinical Medicine*, by the author, p. 248.

Reduplication of the heart-sounds denotes a variety of functional disorder, giving rise, however, to no definite subjective symptoms and occurring in various pathological connections. Reduplication of both sounds may be appreciable, or it may be limited to one sound, which is usually the second. The reduplication is recognized easily by one accustomed to auscultation, and the proof is the occurrence of three or four sounds for each carotid pulse. A slight deviation from synchronism in the contraction of the right and the left ventricle accounts satisfactorily for the reduplication of both sounds, but not so satisfactorily for a reduplication limited to the second sound. The latter is best explained by supposing that under certain conditions of arterial pressure the recoil of the aorta and of the pulmonary artery after the ventricular systole is not in unison. This will account for a reduplication of the second sound, although the systoles of the two ventricles occur synchronously.

A variety of functional disorder to which the attention of clinical observers has not been much directed is diminished frequency of the heart's action. In a paper on this topic I have reported several cases in which the number of ventricular systoles was diminished to from 40 to 26 per minute.[1] Physical exploration in these cases showed that the disorder was not associated with any organic affection of the heart. The disorder was associated with more or less cerebral disturbance, but the evidence of intracranial inflammation, embolism, and hemorrhage was wanting, and in every instance the disorder was temporary. I have met with other examples since the publication of that paper. The cardiac disorder is evidently connected with some centric nervous condition which affects the heart through the inhibitory action of the pneumogastrics. To recognize the disorder as a functional one is important; otherwise it is regarded as a symptom pointing to some grave cerebral affection. It was so regarded in the cases which I have reported. Before deciding that the disorder exists, two points are to be ascertained—namely, *first*, that the radial beats represent accurately the ventricular systoles—that is, to distinguish this from another variety of functional disorder which has been noticed, in which every alternate systole is too feeble to produce an appreciable pulse at the wrist; and *second*, that the patient has not normally an infrequent pulse. The exclusion of grave cerebral affections by the absence of evidences of their existence other than the infrequent pulse is of course important. The cerebral affections which diminish the frequency of the heart's action rarely, if ever, occasion so great an infrequency as occurs in some instances in which it is purely functional. As great or greater infrequency is observed in some cases of fatty degeneration of the heart; hence this lesion, as well as other organic affections, is to be excluded.

Infrequency of the heart's action, reduplication of the heart-sounds, and the disorder characterized by alternate systoles too feeble to be represented by a radial pulse are rare varieties of functional disorder. The most frequent varieties are the forms of disturbance first mentioned—namely, rapid action either violent or fluttering, irregularity, and intermittency occurring in paroxysms. They are often met with in practice. They usually give rise to great mental apprehension. The patient has a firm conviction of the existence of organic disease and is in fear of sudden death. It is sometimes difficult to remove this conviction by the most positive assurances to the contrary. Cases of functional disorder are distinguished, as a rule, by much mental anxiety and depression, in this respect differing from cases in which disturbed action is due to structural lesions.

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[1] Vide *The American Practitioner*, January, 1876.

The causes of functional disorder are various. It sometimes occurs in so-called full-blooded persons addicted to the pleasures of the table, digesting and assimilating readily, and taking but little active exercise. It is much oftener, however, associated with anæmia. Anæmic persons are rarely exempt from more or less disturbance of the heart's action. It may be produced by long-continued mental anxiety or depression. It frequently occurs in persons of an anxious, worrying disposition, and in those who are constitutionally disposed to melancholy. The effect which it produces on the mind reacts on the disorder and tends to perpetuate it. It appears in some cases to be dependent on indigestion or dyspepsia, and paroxysms are often excited by dietetic errors. The immoderate use of tobacco or of strong tea and coffee gives rise to it in some persons. It is incidental to the gouty diathesis. The exhaustion following prolonged mental excitement and physical exertions may be accompanied by it. It prevailed to a great extent among soldiers during the late civil war, excessive and persistent palpitation often leading to discharge on the ground of disability from supposed organic disease of the heart. Excessive venery and habits of self-abuse are to be included among the causes. The associated symptoms will of course vary according to the different circumstances under which it occurs. The different causes of course act upon the nervous system; but it is not certain whether the particular nerves acted upon be the pneumogastrics, the sympathetic, or the nerves and ganglion-cells in the substance of the heart. A predisposition seems to enter into the etiology. Some persons apparently have, as an idiosyncrasy, an irritable heart, and they generally have other evidences of what is called a nervous temperament.

It is extremely desirable, in view of the comfort and welfare of the patient, to determine with positiveness, in cases of functional disorder, that structural lesions do not exist. Several points connected with the history and symptoms have a bearing on the diagnosis. The occurrence of the disturbance in paroxysms, the action at other times being regular; the paroxysms occurring at night rather than in the daytime, and frequently not being occasioned by any obvious cause, such as muscular exertion or mental excitement; the ability of the patient to take active exercise without palpitation or dyspnœa when not suffering from the disorder; and the intensity of mental anxiety and apprehension,—are points which render it probable that the difficulty is purely functional. These points, however, are not conclusive. A positive diagnosis is to be based on the exclusion of lesions of structure, by the absence of the physical signs of the latter. If, on a careful examination of the chest, the heart be found not to be enlarged; if there be no murmur present or if an existing murmur be inorganic; if the heart-sounds be normal; and if, from the absence of persistent weakness or irregularity of the heart's action, fibroid or fatty degeneration of the heart and obstruction of the coronary arteries can be excluded,—the affection may be confidently pronounced functional. Without the negative proof afforded by physical exploration the mind of the practitioner must be in doubt as to the diagnosis. If he give a decided opinion, it is a guess which may prove to be either right or wrong. If he avoid giving a decided opinion, the inference which the patient usually draws is that organic disease exists and that the physician is reluctant to tell the truth. I could cite, from cases which have come under my observation, not a few in which patients were for many years rendered unhappy and deterred from engaging in the active duties of life, either by an erroneous medical opinion that they had organic disease of the heart or by a fixed belief that such was the fact, based on the indecision of the physician.

The curative treatment of functional disorder must have reference to the circumstances with which it is connected. Occurring in connection with what is known as a "full habit," the measures indicated are—restriction of diet, exercise in the open air, with depletion by saline laxatives and perhaps by small bloodlettings. Occurring in connection with anæmia, measures of precisely an opposite character are indicated—namely, those designed to restore the normal condition of the blood. An investigation with reference to the source of the disorder is to be made in all cases, and the causes, if apparent, are to be removed. If connected with dyspeptic ailments, these are to be remedied by appropriate treatment; and if dependent on the gouty diathesis, this will claim suitable remedies. In the great majority of cases the proper treatment consists of tonic remedies, a nutritious diet, and an avoidance of excesses of all kinds; a proper amount of sleep; the interdiction of tobacco, strong coffee, and tea; and out-door life with mental recreation; in short, an invigorating system of hygiene. Positive assurances of the absence of organic disease, such as may be given if based on the absence of all the physical signs of lesions, will often go far toward effecting a cure, the disorder being frequently kept up by mental disquietude occasioned by the fear of sudden death.

During the paroxysms palliative measures are called for. These consist of ethereal stimulants, antispasmodics, and occasionally opiates, with sinapisms or stimulating liniments to the chest. If paroxysms occur frequently a belladonna plaster may be constantly worn over the præcordia. Blisters or other severe counter-irritants are never requisite. Small doses of aconite are useful if the action of the heart be violent, and, on the other hand, if the action be feeble and irregular digitalis may prove of use. Kunze and other German authors recommend as a palliative measure wearing an ice-bag over the præcordia. A mouthful of brandy or some other spirit, swallowed undiluted, will sometimes promptly arrest a paroxysm.

Persons who are strongly predisposed to disturbed action of the heart are liable to suffer more or less from paroxysms of functional disorder for many years or during their whole lives. The mental anxiety in such cases after a time ceases, and patients become reconciled to this as to other physical evils. The frequent recurrence of functional disorder for an indefinite period does not lead to the development of organic disease. The physician is fully warranted in giving positive assurance to the patient on this point.

In the foregoing remarks on functional disorder it has been assumed that structural lesions are not present; but functional disorder and lesions may be associated when the former is not dependent on the latter; in other words, the causes which give rise to functional disorder when the heart is free from lesions will produce the same effect when cardiac lesions exist. It is highly important that the practitioner appreciate this fact. Not infrequently the disturbance of the heart's action in cases in which organic murmurs are found is mainly or exclusively functional. It is not uncommon for practitioners to err in considering all the symptoms referable to the heart as arising from organic lesions, although these may be entirely innocuous. Anæmia coexisting with lesions may occasion greatly disturbed action of the heart, with dyspnœa and even general dropsy, so that the patient may seem to be in the last stage of cardiac disease, and if the anæmia be cured the recovery sometimes appears to be complete. It should always, therefore, be a question in cases of organic lesions whether more or less of the symptomatic phenomena may not be due to functional disorder arising from anæmia, deranged digestion, or a morbid condition of the nervous system produced by tobacco, coffee or tea, mental depression, excessive venery, or gout. This question is to be decided in the affirmative when causes of functional disorder are discoverable, and when the organic affection, as determined by the physical signs, appears to be insufficient to account for the symptomatic phenomena. In proportion as the latter are fairly attributable to functional disorder, the prognosis is of course less grave, and improvement may be expected from appropriate treatment.

Enlargement of the Thyroid Body and Prominence of the Eyeballs (Exophthalmic Goitre), associated with Functional Disorder of the Heart.

Within recent years the attention of clinical observers has been directed to an affection characterized by enlargement of the thyroid body and prominence of the eyeballs, conjoined with inordinate action of the heart. These three pathological events form a striking combination, giving to the affection a well-marked individuality. In the absence of a name expressive of its character, a German author, Hirsch, applied to it the name *Basedow's disease*, after a German observer who was among the first to describe it fully; and the distinguished clinical teacher, Trousseau, in view of the prior claim of the late Dr. Graves, proposed to call it *Graves's disease*. The three events just named evidently have some pathological connection, the nature of which is not yet fully established.

The prominence of the eyes is the most remarkable of the triple event. Existing in some cases in a marked degree, it gives to the countenance a peculiar expression. The projection of the globes displays more or less of the tunica albuginea, and the patient has a ferocious, staring look, which, existing in both eyes, is at once diagnostic. The projection is sometimes so great as to prevent closure of the lids, and the eyeballs are partially exposed during sleep. Vision is generally not impaired, and the appearance of the eyes, aside from the prominence, is natural. The condition of the eyes does not involve pain, but the patient sometimes feels as if the eyeballs were being squeezed out of the sockets. Accompanying and sometimes preceding the exophthalmus defective co-ordination of the movements of the eyeballs with those of the upper eyelids is observed. If the patient look downward, the eyeballs move without corresponding movements of the lids. This defective co-ordination is not a feature in all cases. The exophthalmus is sometimes more marked on one side, and it may be unilateral. Falling out of the eyebrows and cilia is sometimes observed. The thyroid body is more or less enlarged, the enlargement being usually greater on the right side. The increase does not go on indefinitely, forming the immense tumors which are sometimes seen in cases of ordinary bronchocele, or goitre, but ceases after a moderate or considerable augmentation of bulk has been attained. The swelling does not give rise to pain, but it may occasion some obstruction to respiration from pressure on the trachea, and may lead to embarrassed breathing and modification of the voice by interfering with the function of the recurrent laryngeal nerve. The thyroid arteries are enlarged, and a strong pulsation is felt over these and the carotids, frequently accompanied by marked thrill. Arterial and venous murmurs, frequently loud and sometimes musical, are heard when the stethoscope is placed over and near the enlarged thyroid body. The degree of the thyroid enlargement is often variable at different times.

Increased frequency of the heart's beats (tachycardia) is a constant and notable feature. In some cases this amounts to a persisting palpitation. The pulse varies from 100 to 140 beats per minute. Valvular lesions and enlargement of the heart exist in some, but by no means in all cases. When present, they do not account for the functional disorder. Loud inorganic murmurs at the arterial orifices of the heart are not infrequent. The inordinate action of the heart may continue persistently for months and years.

More or less anæmia coexists generally, but not invariably. This has been by some incorrectly considered as an essential pathological element of the affection. The anæmia, however, is usually marked. Other symptoms are mental depression and irritability, insomnia, amenorrhœa, abnormal increase of the appetite followed by anorexia, hysterical phenomena, and emaciation. The affection is essentially chronic. In the majority of cases it continues for years. It does not tend intrinsically to a fatal result, but patients are liable to be cut off during its continuance by some intercurrent affection. Recovery takes place in a certain proportion of cases, the improvement being slow, and some degree of enlargement of the thyroid body and prominence of the eyes often being permanent. Fothergill quotes from Von Dusch 14 complete recoveries in 56, and great improvement in 26 cases; and of cases observed by Von Graefe, the ratio of recoveries was 20 per cent., and of great improvement, 30.

The pathological character of the affection is not established. That the three pathological events are due exclusively to anæmia is disproved by the occurrence of anæmia so frequently without these events, and by the fact that there is not always coexisting anæmia. The enlargement of the thyroid body is chiefly due to enlarged vessels. The prominence of the eyes is not due to enlargement of the globes. In some autopsical examinations which have been reported an abnormal amount of areolar and adipose tissue has been found at the bottom of the orbit; but that the protrusion is not chiefly due to this cause is shown by the fact that it has been observed to cease after death, and during life moderate pressure suffices to restore the globes to their normal situation. Enlargement of the vessels behind the globes is probably the chief cause. The prominence of the eyes and enlargement of the thyroid body are consecutive to the cardiac disorder. Cases are observed in which enlargement of the thyroid body is conjoined with inordinate functional activity of the heart, without prominence of the eyes, and the two latter may be associated without enlargement of the thyroid body. Examples of each of these variations have fallen under my observation. I have met with cases of rapid action of the heart persisting for a long period without either prominence of the eyes or enlargement of the thyroid bodies, in which I suppose the affection to be the same minus these associated events. Considering that the cardiac disturbance always precedes the two other characteristic events, that either of the latter may be wanting, and that both involve enlargement of vessels, a fundamental morbid condition evidently relates to the circulatory system; but this condition probably involves a prior neuropathic affection which, it is probable, pertains to the sympathetic nerve. In some instances morbid appearances after death in and around the cervical sympathetic nerve have been found, but in other instances nothing was discoverable. The neuropathic condition is supposed by some to be seated in either the cervical portion of the spinal cord or the medulla oblongata. The affection in the great majority of cases occurs in females. Of 50 cases collected by Wilthuisen, a Danish author, only 8 were male subjects. In all the cases which I have seen, save one, the patients were females. The disease sometimes shows an hereditary tendency.

The indications for treatment relate to the functional disorder of the heart, together with the morbid state of the nervous system and the often-coexisting anæmia. Sedative remedies which tranquillize the action of the heart without producing depression or interfering with the appetite and digestion are indicated. For this end, hydrocyanic acid, aconite, belladona, and digitalis may be employed. The anæmia calls for tonic remedies, especially ferruginous tonics; and the latter have seemed to be beneficial in cases which I observed. Arsenic has been found useful, but in some instances it has apparently done harm. Iodine, with reference to the affection of the thyroid body, is of doubtful utility. In two cases under my observation, recovery in the one case and notable improvement in the other case occurred under the use of the tincture of aconite. In the first of these cases the patient took one minim only of the tincture three times daily, but continued it for three years in conjunction with a chalybeate tonic. At the end of that period she was perfectly well and had increased in weight forty pounds. In the other case the dose of the tincture was gradually increased to seven minims three times daily, and was continued steadily for ten months without a chalybeate, anæmia not existing in this case. Several observers bear testimony to the usefulness of galvanization, with the constant current, of the cervical sympathetic nerves. Cases of recovery under this agent have been reported. Ergot is among the remedies which have been found of service. Hygienic measures are of much importance. Freedom from mental depression is to be secured as far as possible, and, whenever practicable, change of scene and other means of mental recreation are advisable. The diet should be nutritious, and out-door life is desirable. Trousseau advocated the employment of cold water after the hydropathic method.

Angina Pectoris.

The affection known as angina pectoris involves, as an essential feature, neuralgia, and might therefore be included among the diseases of the nervous system. It is considered here on account of its frequent connection with cardiac lesions and with notable disorder of the heart's action. The following description will relate to typical cases of angina associated with disease of the heart.

The affection is characterized by pain, usually intense, burning, tearing, or lancinating in character, emanating from the præcordia or beneath the sternum, and radiating thence in different directions—namely, into both sides of the chest, the back, and frequently the left shoulder; extending down the arm and sometimes to the forearm; occasionally felt in the forearm and hand, and not in the shoulder and arm; in some cases extending more or less into both upper extremities, and it has been known to extend into one or both of the lower extremities; and, finally, it may shoot upward into the neck, temples, and jaws. Different cases differ much as regards the intensity of the pain and the situations, among those just named, in which it is felt. The pain in some cases is so intense as to be truly excruciating, and is accompanied by a feeling of constriction of the chest; in other cases it is moderate or even comparatively slight.

The affection is paroxysmal, and the pain is but one of the elements which enter into the paroxysm. The action of the heart is generally more or less disturbed. The beats are irregular, sometimes violent or tumultuous and sometimes feeble. In some cases the pulse is notably slow. An indescribable anguish or "heart-pang" is another element. A vivid sense of impending death is felt. The paroxysm is usually developed suddenly, and the patient feels compelled instantly to remain perfectly still, seizing hold of some firm support in order to maintain a fixed position. The patient is afraid to breathe freely, although the power of doing so is not lost. Dyspnœa does not belong to the paroxysms, save in so far as it may depend on existing cardiac lesions or on the voluntary restraint of the respiratory movements. A sense of numbness is felt in the parts into which the pain extends. The countenance denotes anxiety and suffering. The surface is generally pallid, cool or cold, frequently bathed in perspiration, and sometimes cyanosed or livid.

Cases differ greatly as regards not only the severity of the paroxysms, but their duration and the frequency of their recurrence. They usually last for a few moments only, but in some cases they continue for several hours. They may recur at wide intervals or frequently. As a rule to which there are a few exceptions, if a paroxysm have been once experienced other paroxysms will follow sooner or later. Generally they become progressively more and more frequent. At first and for some time they appear to be excited by some notable cause, such as unusual muscular exertion or mental emotion, but after a time they are liable to occur from trivial causes and without any apparent cause. They are sometimes produced during sleep, apparently by distressing dreams.

The affection as thus described is incident generally to organic disease of the heart, of the aorta, or of the coronary arteries, but, as far as at present known, not to any one form of lesion. It is much oftener associated with lesions at the aortic than at the mitral orifice, yet it occurs in only a very small proportion of the cases of aortic lesions. It occurs in some cases of fatty degeneration of the heart and of myocarditis without valvular lesions. It is sometimes associated with aneurism of the aorta. The affection is rare. Of over 150 cases of organic disease of the heart which I analyzed in 1859, it occurred in only 7, and it occurred in 8 out of 188 additional cases analyzed in 1869. That it has a pathological connection with organic disease of the heart is certain; but upon what particular condition or circumstance common to different forms of organic disease it depends is not ascertained.

Angina pectoris, especially if associated with diseases of the heart, involves liability to sudden death. Death takes place suddenly in a paroxysm. The most reasonable way of accounting for the sudden death, as it seems to me, is to attribute it to over-accumulation of blood within the ventricular cavities, and arrest of the heart's action as a result of paralysis from distension. The explanation, in fact, is the same as of sudden death in cases of free aortic regurgitation and fatty degeneration. The liability to sudden death characterizes cases in which the coexisting organic disease is of a kind to favor sudden over-accumulation of blood in the cavities. Aortic lesions and fibroid or fatty degeneration have this effect. If the lesions do not lead to this effect, patients rarely die suddenly, but linger on for an indefinite period, with the recurrence of the paroxysms more or less frequently. Moreover, the overloading of the cavities of the heart will account for elements of the paroxysms other than the pain—namely, the disturbed action of the heart, the indescribable anguish, and the sense of impending death. If the lesions to which the angina is incident be not of a character to lead to overloading and consequent distension of the ventricular walls, the suffering is mainly from the pain. Some cases of angina are of this description, very little disturbance of the heart's action accompanying the paroxysm. On the other hand, all the elements of a paroxysm of angina, except the pain, are observed not infrequently in cases of aortic lesions with regurgitation or with fibroid or fatty degeneration; and sudden death is liable to occur in the paroxysms of distress to which patients affected with these lesions are subject, the paroxysm lacking only pain to constitute angina pectoris. The prognosis in cases of angina is always grave if physical signs show the existence of serious cardiac lesions; and sudden death is to be expected if there be much aortic regurgitation, or the habitual weakness of the heart render probable impaired nutrition of the organ from obstruction of the coronary arteries or degeneration of the walls. The danger, it is to be observed, is not to be measured by the intensity of the pain; it is not the pain which kills, but the coexisting organic disease.

Angina pectoris, as far as the neuralgic element is concerned, may occur without any evidence of coexisting disease of the heart or the aorta. Several examples have fallen under my observation. In these cases the paroxysms generally lack those elements which are derived from cardiac lesions. The necessity of keeping a fixed posture while the paroxysm lasts is not felt in the same degree; and the patient may writhe with pain and try frequent changes of posture with the hope of obtaining relief. The sense of impending death is less or may be wanting. There is less danger of sudden death, and the prognosis as regards recovery is favorable. These points are to be considered in the differentiation of these cases from those in which the angina is connected with disease of the heart; but the absence of cardiac lesions is determined by the negative result of a careful physical exploration of the chest. It is, however, to be considered that there are anatomical changes not represented by physical signs—*e. g.* defective nutrition from obstruction of the coronary arteries. It may be stated that the danger in a given paroxysm is in proportion to the feebleness and irregularity of the heart's action, together with a feeling of impending dissolution. In the endeavor to estimate the danger, the nature and extent of lesions, as evidenced by signs, are to be taken into account. Much damage of the aortic valvès, dilatation of the left ventricle, and weakness of the heart from fatty or fibroid degeneration are the conditions which especially render paroxysms dangerous. Yet there are cases in which the paroxysms recur for a long time without proving fatal, although heart lesions exist, and if the latter do not exist it is probably true that there is little danger of sudden death. I have reported several instances of recovery in the second edition of my work on *Diseases of the Heart* (1869). Such instances should not be lost sight of in the prognosis.

Angina is to be discriminated from intercostal neuralgia and gastralgia. Each of these affections I have known to be mistaken for it. Paroxysms of intercostal neuralgia are sometimes so severe as to rival, in the intensity of pain, attacks of angina. In other respects the points of distinction are sufficiently marked. Both intercostal neuralgia and gastralgia are distinguished by their own diagnostic traits, aside from the absence of those which belong to angina. This statement applies also to paroxysms of pain caused by the passage of gall-stones. The most rational explanation of the immediate cause of the paroxysms is that they are due to deficiency of arterial blood in the walls of the heart (ischæmia).

Pertaining to the pathological character and relations of the different elements in angina are discussions which cannot be here entered into. The neuralgic element probably has its primary seat in the cardiac plexuses, and the radiation of pain to the neck and upper extremities, as well as to other parts of the chest, may be explained by anatomical connections with branches of the cervical and dorsal nerves. As remarked by Eulenburg, the pain closely resembles that referable to the sympathetic nerve in other situations; for example, hepatic colic, enteralgia, etc. The disturbed action of the heart, irrespective of cardiac lesions, is as unintelligible as when it occurs in the various forms of functional disorder. To institute several varieties of the disease according to the different nerves supposed to be involved seems to me to be, in the present state of our knowledge, premature and confusing. Practically, however, the presence or absence of cardiac lesions and the degree of disturbance of the heart's action in the paroxysms are important points, especially in reference to prognosis and the liability to sudden death.

The TREATMENT embraces measures to afford relief in the paroxysms and those indicated in the intervals. The palliative measures embrace opium, given promptly and sufficiently to relieve the pain. It should be given in a form to act as speedily as possible. The hypodermic method, if the physician happen to be at hand, is to be preferred. Alcoholic, ethereal, and other stimulants are indicated in proportion as the action of the heart is irregular and feeble. Revulsive applications to the chest and extremities are appropriate. Swallowing pieces of ice has been found effective. The nitrite of amyl is a valuable remedy. Five drops inhaled from a handkerchief held over the nostrils will often almost instantly arrest a paroxysm. It may prevent sudden death, and trial of it should therefore always be made, observing, of course, proper precautions in its use. The remedy is efficacious in only a certain proportion of cases. Naturally, its efficacy is most marked when there is much arterial tension, but it may relieve, although this element be wanting, by diminishing the accumulation of blood in the heart-cavities. The inhalation of ether or of chloroform is to be mentioned among the resources for arresting the pain or mitigating its severity. The inhalation of chloroform should not be resorted to if the action of the heart be feeble.

In the intervals the patient should avoid, as far as possible, everything which will be likely to provoke a paroxysm, such as active exercise, mental excitement, over-ingestion of food, and the abuse of alcohol. Aside from prophylaxis, the measures indicated are those which have reference to the causes of neuralgia, to the general health, and in the majority of cases to coexisting organic disease of the heart or the aorta. Anstie found arsenic, in small doses long continued, extremely useful in preventing the paroxysms. Others have recommended this remedy, and, again, others have seen no benefit from it. A fair inference is that it is sometimes useful and sometimes not, as in other neuralgic affections. Trousseau, who regarded the affection as analogous to epileptic seizures and called it *cardiac epilepsy*, advised the long-continued employment of belladonna. Electrization of the surface within the præcordia has been found useful both in arresting the paroxysms and postponing their occurrence. The cases most likely to be amenable to treatment are those in which the angina is purely neuralgic; that is, not associated with heart lesions.

The name angina is singularly inappropriate as applied to this disease, inasmuch as neither the symptoms nor the danger have aught to do with strangulation. The term stenocardia has significance only as denoting a sensation accompanying the pain. Of the other various names, most imply erroneous views of the nature of the malady. Cardialgia would be an appropriate name had it not been applied to a gastric affection.

Thoracic Aneurism.

An atheromatous condition of the arteries has been usually assigned as the leading cause of spontaneous aneurisms. This atheromatous process is at the onset inflammatory; it is an endarteritis chronica deformans (Virchow). The intima at first is swollen and of a whitish or gelatinous appearance in patches. In these swollen patches are round, fusiform, and stellate cells in increased number. The intercellular substance is imperfectly fibrillated. The endothelial lining of the vessel is preserved. Subsequently, necrosis with fatty and calcareous degenerations occurs in the cells and intercellular substance; and to the resulting pulpy mass, often containing cholesterin, the name atheromatous abscess has been inappropriately applied. This mass may be discharged by rupture of the endothelial covering, and an atheromatous ulcer is the result.

Recent investigations have rendered it probable that undue importance has been assigned to atheroma as a cause of aneurism. Atheroma is most frequent in old age, a period of life in which aneurisms are by no means most common. The view is now held that the most frequent cause of spontaneous sacculated aneurisms is to be sought in changes in the middle or muscular coat of an artery. It is this coat which normally gives the greatest strength to the arterial wall. The changes in the middle coat which lead to aneurism are various. A rupture of the elastic and of the muscular fibres in a previously healthy, or more frequently in a diseased, middle coat may be the result of traumatism or of some severe strain. A frequent change in the media, especially in cases of atheroma, is a localized inflammation, characterized at first by an accumulation of lymphoid cells, and later by a growth of fibrous tissue. This mesarteritis, as the inflammation is called, leads to a weakening of the middle coat and consequent production of aneurism.

The ascending aorta is the most frequent seat of large aneurisms. If, however, small or miliary aneurisms be reckoned, then the cerebral and the pulmonary arteries are most frequently affected.

Thoracic aneurism occurs either in the form of a diffuse dilatation, most frequently of the ascending aorta, or in the sacculated form. It is the sacculated aneurism which is specially considered in the present article.

According to Rindfleisch, aneurisms of the ascending portion and of the arch of the aorta develop most frequently in the course of a spiral line which corresponds to the places against which the blood impinges with greatest pressure. This line begins on the anterior aspect of the bulb of the aorta, gradually passes on the front of the ascending aorta toward the right, crosses the convexity of the artery just before the innominate trunk is given off, and from here passes very gradually to the posterior and internal aspect of the descending aorta.

Referring the reader to other works for a fuller account of aneurisms as regards their pathology and morbid anatomy, I shall consider in this article aneurismal tumors of the thoracic aorta with reference to their symptoms, physical signs, prognosis, and treatment. As bearing on the diagnosis, it may be here stated that aortic aneurism is a rare event under forty years of age, and that syphilis is often a remote cause.

Aneurismal tumors may spring from the thoracic aorta at different points. Not infrequently they originate within the pericardium, in the sinuses of Valsalva, and in this situation rupture occurs before the tumor attains to great size, the hemorrhage taking place into the pericardial sac, and of course causing instant or very speedy death. A fatal result from aneurism in this situation may occur without having been preceded by any symptoms of importance; death takes place unexpectedly, perhaps when the health was apparently perfect. The diagnosis is difficult and perhaps impossible. Of 703 cases analyzed by Sibson,[1] 87 were within the pericardium. The ascending portion of the arch beyond the pericardium is the most frequent site. In 193 of the 703 cases analyzed by Sibson the situation was in this portion. The ascending and transverse portions are not infrequently both involved in the dilatation. This was the case in 140 of 703 cases. The transverse portion was affected alone in 120 cases, and conjointly with the descending aorta in 20 cases. The descending portion of the arch was the seat in 72, and the aorta below the arch in 71 cases. The aneurismal tumors in the larger proportion of cases spring from the outer aspect of the artery, but they may spring from either the inner, posterior, or anterior aspect.

Aneurismal tumors, according to their situation and size, give rise to certain symptoms and signs by pressing upon the different surrounding parts. Unless referred to their true source, these symptoms may mislead the physician by appearing to indicate other affections; and by means of these symptoms and signs not only may aneurism be suspected, but, assuming its existence, its situation may be inferred.

Interruption of the circulation through the innominata and the left carotid and subclavian artery may arise either from the pressure of the tumor on these vessels or obstruction by fibrinous plugs. Feebleness or extinction of the pulse in the radial, brachial, or carotid artery on one side is one of the symptoms of aneurism, and indicates its situation to be at the right or left extremity of the transverse portion of the arch. When the radial pulse is not abolished, but more or less weakened, the contrast between the pulse of the two arms is well shown by a comparison of the visible characters as traced by the sphygmograph. A notable retardation of arterial pulsations beyond the seat of the aneurism is sometimes observed. Thus, when the aneurism is seated between the points of origin of the great vessels of the arch of the aorta, the radial pulse is felt later on one side than on the other, and if the aneurism be seated in the descending aorta the pulsations of the arteries below occur later than the pulse at the wrist. An aneurismal tumor springing from the ascending or transverse portion of the arch may press upon the superior vena cava or the venæ innominatæ, and induce venous congestion limited to the head and upper extremities or to one upper extremity, accompanied, perhaps, by more or less œdema. These symptoms point to the existence of aneurism and its situation.

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[1] *Med. Anatomy*, fasc. v.

An aneurism seated in the transverse portion of the arch may press upon the trachea or one of the primary bronchi and interfere with respiration. If the obstruction of the trachea be considerable, the breathing is labored, and may be accompanied with a noise, heard at a distance, called stridor. Auscultation will show feebleness of the respiratory murmur on both sides of the chest if the trachea be pressed upon, but feebleness limited to one side and the murmur exaggerated on the other side if one of the bronchi be obstructed. Embarrassment of respiration may proceed from pressure upon the recurrent laryngeal nerve. If the pressure be sufficient to interrupt the function of the nerve on one side, laryngeal obstruction arises from an arrest of the respiratory movements of the glottis on that side. The embarrassment from this cause, however, is not great. The laryngoscope in these cases shows the vocal cord on one side to be paralyzed, while the respiratory movements of the other cord continue, or after a time the paralysis may become bilateral. Spasm of the glottis may be induced if the relation of the nerve to the tumor be such that it is irritated instead of its function being annulled. I have reported a case in which, the left recurrent nerve being situated between a calcareous deposit and the aneurismal tumor, spasm of the glottis occurred so frequently and to such an extent as to prove fatal.[1] Dysphonia and aphonia are effects of either incomplete or complete paralysis from pressure on the recurrent laryngeal nerve, and the affection of the voice is found to vary at different times according to the varying amount of pressure. These laryngeal symptoms are likely to mislead by directing attention to the larynx as the seat of disease. Tracheotomy has been repeatedly performed under the belief that the obstruction of respiration was due to an affection of the larynx. Pressure of the tumor on the pneumogastric nerve, as ascertained after death, occasioned vomiting and pyrosis in a case reported to me by Prof. McCready.

Pressure on the œsophagus, when the tumor is situated in the transverse or descending aorta, may occasion obstruction to the passage of food. This symptom should excite suspicion of aneurism, and such a suspicion should enforce caution in the use of bougies to explore and dilate the œsophagus. Pressure on the thoracic duct has been known to occur, inducing marked emaciation. Pressure on the sympathetic nerve is another effect of an aneurismal tumor, leading to contraction of the pupil of the eye on the affected side. If the tumor be seated in the descending aorta, it is likely to lead to erosion of the vertebral column, and may thus occasion paralysis by affecting the spinal cord. These cases are characterized by considerable pain, referred to the back. Neuralgic pains and well-marked angina pectoris sometimes accompany aneurism seated in the ascending aorta.

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[1] *American Medical Times*, 1864.

The DIAGNOSIS of thoracic aneurism is to be based on the presence of more or less of the foregoing symptomatic phenomena, taken in connection with the physical signs of a tumor within the chest situated at some point in the tract of the aorta. The signs are very obvious when the aneurism has led to erosion of the ribs and an external tumor, the latter pulsating, and perhaps presenting a thrill, and the heart-sounds being heard with more or less intensity when the stethoscope is placed over it, together with a single or double bellows murmur. The signs are less marked before the tumor makes its way through the thoracic wall, but they may be sufficient to render the diagnosis positive. Abnormal dulness on percussion over a circumscribed space in the tract of the aorta is an important diagnostic point. This space will be on the anterior surface of the chest if the aneurism spring from the arch, and posteriorly if it spring from the descending aorta below the arch. Careful inspection and palpation may disclose pulsation, and perhaps thrill, before any external tumor is apparent. Undue audibleness of the heart-sounds within the space found to be dull on percussion, and a single or double murmur not transmitted from the aortic orifice, are highly significant signs. They are, however, by no means uniformly present, and therefore, while their presence contributes to the diagnosis, their absence is not proof that aneurism does not exist.

The PROGNOSIS in cases of thoracic aneurism relates chiefly to the duration of life. The instances of recovery are few. In the majority of cases death is the result of rupture of the aneurismal sac. The rupture may take place in various directions—namely, externally, or into the œsophagus, trachea, bronchi, pleural cavity, heart, venæ cavæ, spinal canal, pericardial cavity, etc. The rupture may be at first small, so that death takes place more or less gradually, or it may be large enough to cause a sufficient hemorrhage to destroy life within a few moments. I have met with a case in which, after a considerable hemorrhage caused by rupture into a bronchus, the opening closed, and a fatal recurrence of the hemorrhage did not take place until after several weeks, the patient in the mean time taking considerable exercise under the advice of a practitioner who scouted the idea of aneurism. The progress of the aneurism varies much in different cases, the duration of life being in some cases but a few months, and in some cases several years. In a considerable number of cases life is destroyed before rupture takes place. Pressure on the trachea, œsophagus, vena cava, thoracic duct, or irritation of the recurrent laryngeal nerve may lead to a fatal result, or the patient may be cut off by some intercurrent affection. Organic lesions of the heart coexist in a certain proportion of cases. These of course involve distress and danger in proportion to their nature and extent, and may prove the immediate cause of death. Clinical observation shows that patients affected with thoracic aneurism rarely have pulmonary tuberculosis.

Recovery from thoracic aneurism sometimes takes place, but in general the only results to be hoped for from treatment are palliation of suffering and prolongation of life. With reference to these results all active exertions are to be avoided, and, as far as possible, emotional excitement. The body is to be well nourished if possible, but the diet should be unstimulating and alcoholic stimulants should be interdicted. Liquids should be taken sparingly. The action of the heart, if abnormally strong, is to be diminished by sedative remedies, and perhaps, if the patient be full-blooded, in some cases by small abstractions of blood. It is highly important not to impoverish the blood.

and anæmia if it exist calls for the treatment appropriate to that condition. Pain and other incidental symptoms are to be palliated by appropriate remedies. If spasm of the glottis be induced through the recurrent laryngeal nerve to such a degree as to endanger life, laryngotomy is warrantable, if not advisable, with a view to palliation and the prolongation of life. This operation will of course be of no avail if the obstruction be due to pressure of the aneurismal tumor on the trachea.

The progress of the aneurism is retarded by the deposit of successive layers of fibrin within the aneurismal sac. It is in this mode that a cure sometimes takes place. It is desirable, therefore, that the blood should be rich in fibrin, and it is probable that the deposit is more likely to take place in proportion as the action of the heart is slow. Cardiac sedatives—namely, aconite, digitalis, and the veratrum viride—may in this way be useful. Certain remedies have a more direct curative effect in some cases of aneurism. At the present time the iodide of potassium is to be relied upon more than any other known remedy. Cases in which a cure was apparently effected by this remedy have been reported by Roberts, Balfour, Bouillaud, and others. I have witnessed remarkable improvement under the use of this remedy in several cases.[1] The doses should be increased up to a point of comfortable tolerance and continued for a considerable period. The iodide of potassium should be faithfully tried in all cases of thoracic aneurism.

[1] Vide *Clinical Medicine*, 1879.

SECTION THIRD.

DISEASES AFFECTING THE HÆMATOPOIETIC SYSTEM.

CHAPTER I.

DISEASES OF THE HÆMATOPOIETIC SYSTEM.

Introductory Remarks.—Simple or Benign Anæmia.—Chlorosis.

IN this section will be considered certain diseases which are believed to be the result of disturbances in the blood-forming functions. It is, therefore, proper to refer these diseases to the hæmatopoietic[1] (or blood-forming) system. As the constituents of the blood-plasma are derived more or less directly from the ingesta, we understand by the hæmatopoietic system especially those organs and parts in which resides the function of producing the formed constituents of the blood—namely, the red and the white corpuscles. The name cytogenic organs has also been employed in the same sense. Our knowledge as to the development of the blood-corpuscles and as to the situations in which they are formed is very imperfect, notwithstanding the abundant researches devoted to these subjects, especially during the last few years. The parts to which has been assigned, with more or less probability, the function of forming blood-corpuscles after birth are the lymphatic glands, the spleen, the red marrow of the bones, and the blood itself.[2] To these parts, therefore, may be given, at least provisionally, the name hæmatopoietic system.

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[1] From *αἷμα*, *blood*, and *ποιέω*, *to make*.

[2] White blood-corpuscles are derived from the lymphatic tissues of the body, especially from those organs where this tissue is most abundant—viz. the spleen, the lymphatic glands, and the red marrow of the bones. Red marrow, which in embryos and young infants exists in all of the bones, is present in the adult in the bones of the trunk, but is replaced by fatty marrow in the long bones of the extremities. In severe forms of anæmia the yellow or fatty marrow is often changed into red or embryonic marrow.

It is now generally held that the red blood-corpuscles are in some way derived from the nucleated red blood-corpuscles which are constantly present in the red marrow of the bones, but the manner of their development is not understood. Some believe that red blood-corpuscles are formed out of ordinary white blood-corpuscles, the nucleated red corpuscles being a transitional stage in this formation (Neumann). Another view is that certain colorless cells, which are thought to differ from ordinary white blood-corpuscles in the possession of vesicular, reticulated nuclei and in the mode of cell-division, give origin first to nucleated red blood-corpuscles, and these to ordinary red corpuscles (Löwit). Still others consider that the nucleated red blood-corpuscles are independent cells which by indirect cell-division produce red corpuscles (Bizzozero). Karyokinetic figures are abundant in various cells found in the osseous medulla, including the nucleated red blood-corpuscles. Under normal conditions, when the new red blood-corpuscles enter the circulation they are fully formed and devoid of nuclei. There is no proof that red blood-corpuscles are formed after birth elsewhere than in the marrow of the bones. A full consideration of these as well as of other problems in the physiology of the blood-corpuscles will be found in the "Cartwright Lectures on Certain Problems in the Physiology of the Blood-corpuscles," by William Osler, *The Medical News*, April 3 *et seq.*, 1886.

The diseases treated of in this section are grouped together, not so much with reference to the anatomical parts affected as in consequence of certain common characteristics. In the majority of these affections we cannot explain, by known anatomical changes, the alteration which exists in the composition of the blood. This is true in spite of the fact that in many cases pathological changes have been recognized in parts thought to belong to the hæmatopoietic system. It is probable that a relation of cause and effect exists between these changes and the blood-alterations, but the connection is rendered obscure by our ignorance as to the life-history of the blood. The anatomical changes found in some of the diseases considered in this section are to be regarded as secondary rather than as primary elements in the disease. We must content ourselves frequently with establishing the existence of some disturbance in the blood-forming function, without attempting to refer the disturbance to anatomical changes.

A common and distinctive characteristic of the diseases of the hæmatopoietic system is the presence of anæmia, usually of a grave form. This anæmia is called *essential,* in that it is not referable to diseases which interfere secondarily with the composition of the blood, such as Bright's disease, cancer, and phthisis, but is attributable to a direct interference with the blood-forming or blood-destroying functions. In many of the essential anæmias no anatomical change has been found in the body which can, with any probability, be assigned as the cause of the anæmia; and in others, as in leucocythæmia and pseudo-leucocythæmia, there are morbid changes which may be considered as interfering more or less directly with the formation of the blood-corpuscles. In these diseases the normal ratio between the consumption and the renewal of the corpuscular elements of the blood is disturbed. The destruction of the corpuscles may be abnormally increased (hæmophthisis), or the reproduction of corpuscles may be diminished below the normal amount (anæmatosis), or both conditions may be combined. It is usually impossible to determine in a given case which of these causes is at work. The result of this disproportion between consumption and renewal is impoverishment of the blood, or anæmia, which, together with the consequent disturbance of nutrition, forms the dominating factor in all of these diseases. It will be found, therefore, that these affections have much in common in their clinical history. They are for the most part insidious in their onset and chronic in their course. As a rule, their causes are obscure, and, with the exception of simple anæmia and chlorosis, their prognosis is grave.

For certain general considerations concerning anæmia and the changes in anæmic blood the reader is referred to Part I. (p. 60).

Simple or Benign Anæmia.

The terms simple and benign may be used to designate anæmia when not pernicious or an element of the other affections embraced in this section, and when diseases of which the anæmic condition is symptomatic, such as phthisis, empyema, carcinoma, etc., may also be excluded. It may be said that, as thus restricted, anæmia is only a symptom. It occurs, however, under various circumstances, and for convenience it may be considered in the light of an individual disease with as much propriety as certain other events which are symptoms—for example, jaundice. Simple or benign anæmia, as just defined, enters largely into medical practice.

General anæmia, as regards anatomical distinctions (oligæmia, oligocythæmia, etc.), together with its causation and effects, has been considered in

Part I. (Vide p. 60.) The obvious causes are—*first*, loss of blood, or hemorrhages; *second*, defective assimilation; and *third*, expenditure of certain blood-constituents, as in lactation and pregnancy. Hemorrhages occur under a great variety of circumstances. In women menorrhagia and flooding with labor are frequent causes. Defective assimilation may depend on a lack of food in sufficient quantity or of food sufficiently nutritious; and with abundant alimentation the processes of digestion may be so impaired as to prevent proper assimilation. Lactation and pregnancy are frequent causes. To these causes may be added certain toxical agencies which interfere with hæmatopoiesis—namely, lead, malaria, etc. Simple or benign anæmia is a sequel of diseases which have kept in abeyance either alimentation or assimilation. Women become anæmic much oftener than men, irrespective of the causes which are peculiar to the sex—namely, pregnancy, lactation, and menorrhagia.

Anæmia gives rise to a multiplicity of morbid phenomena representing especially disorder of the nervous system. The more frequent and prominent are as follows: mental depression, anxiety respecting health, irritability of mind, want of buoyancy and energy, a feeling of lassitude, and a painful sense of inertia or indolence. The feeling of incapacity for muscular exertion is often greater than the actual loss of muscular power. The physical and mental powers are especially depressed during digestion. Palpitation of the heart is frequent, and organic disease is greatly feared by the patient. Neuralgia in various situations is likely to occur, and in women hyperæsthesia of the abdominal walls, simulating peritonitis. The varied symptoms formerly and recently described as due to spinal irritation are liable to occur in connection with anæmia. The varied phenomena considered as hysterical are often associated with, and more or less dependent upon, anæmia. The special relations between the red corpuscles and the nervous system are shown by the neuropathic phenomena to which anæmia gives rise. These phenomena may be said to constitute the pathological expressions of this morbid condition of the blood. The different organs of the body in cases of anæmia are deficient in functional power in proportion to the quantity of blood which they receive in health. The surface of the body is cool and the extremities are frequently cold. The ability to endure cold is lessened. There is diminished endurance of muscular as well as mental exertion. Generally, the face, especially the prolabia, and the mucous membrane in situations where it can be seen, are pallid. The facies often at once denotes anæmia. In some persons, however, the anæmic condition, as determined by physical signs and its pathological phenomena, exists not only without pallor, but with even a rosy complexion. Pallor of the prolabia may be noticeable in the morning or if the patient be fatigued, and it may disappear under excitement. The action of the heart is feeble and easily disturbed, becoming rapid from slight causes. The respirations are panting on exercise. Digestion is labored. The urine often has a low specific gravity from a deficiency of urea, and is notably limpid from a diminution of pigment. The cerulean eye—that is, a dark-blue tint of the globe, owing to a semi-transparency of the conjunctiva—may be a more marked feature than pallor of the face or the prolabia.

The symptomatic evidence generally renders the recognition of anæmia easy. Certain auscultatory signs confirm the diagnosis. The continuous murmur in the neck, known as the *venous hum*, is rarely wanting, and is not often present if there be not anæmia. A frequent sign is a systolic bellows murmur, referable to the carotid arteries. Not less frequently systolic murmurs are heard at the base of the heart, produced within the aorta and the pulmonary artery. The significance of these murmurs depends on their hæmic origin, and this is rendered evident by the absence of signs denoting lesions of the heart or arteries. These hæmic murmurs are useful not only in the diagnosis of anæmia, but as criteria by which the physician is aided in deciding whether the anæmic condition continue or whether it have been removed by measures of treatment. The diagnosis may be rendered demonstrative by ascertaining the number of red corpuscles and the amount of hæmoglobin.

In order to decide that the anæmia is simple or benign, the pernicious form, leucocythæmia, and pseudo-leucocythæmia—diseases considered in this section—are to be excluded, and also all other important diseases which involve, as a symptom or an effect, the anæmic condition. To exclude these diseases is generally not difficult. Although, as has been seen, anæmia stands in a causative relation to a large number and a great variety of pathological phenomena, there are not present symptoms which denote a grave condition. Pyrexia is wanting, and the general nutrition of the body may be well maintained. With reference to the exclusion of important diseases, it is necessary that the physician be qualified to appreciate the phenomena referable to the anæmia. In most instances the causes of the anæmia are apparent, and their existence is to be taken into account in the diagnosis. Moreover, the diagnosis is often corroborated by the speedy effect of appropriate treatment.

The PROGNOSIS in cases of simple or benign anæmia is generally good. Provided the causes do not continue in operation, recovery may be expected. Different cases differ much as regards the rapidity of progress toward recovery. In the exceptional cases which are treated unsuccessfully there is reason to conclude that the want of success may be due to an innate defect in the hæmatopoietic system.

Anæmia may be associated with other diseases, the latter occurring in persons already anæmic. It is of great importance in practice to take cognizance of this association. The phenomena of the associated diseases are modified by the antecedent and coexisting anæmia. Toleration of diseases and recuperation are thereby impaired. Whatever may be the disease thus coexisting with anæmia, but without involving necessarily any relation of cause or effect, the anæmic condition claims recognition, and is to be considered in connection with the indications for treatment. With the different functional affections of the nervous system anæmia is associated as a rule, standing often to them, measurably, in a causative relation. Clinical observation does not afford proof of its predisposing to phthisis, but rather the reverse.

In the TREATMENT of anæmia causal indications are of primary importance. The causes in individual cases are to be ascertained, and if possible removed. Hemorrhages are to be arrested or restrained. Alimentary supplies should be sufficiently abundant and varied. It may be requisite that lactation be discontinued. It is sometimes advisable to caution against pregnancy. Digestion and assimilation are to be promoted by appropriate medical and hygienic treatment. The patient is to be withdrawn from the operation of toxical causes, lead, malaria, etc., and as far as practicable from all unsanitary influences which indirectly lead to the anæmic condition.

The quantity of wholesome food should be equal to, but not exceed, the digestive powers. Appetite and digestion should be promoted by quinia, salicin, and other tonic remedies. The articles of diet should be made attractive. Dyspeptic ailments are to be treated according to the indications to be hereafter considered. (Vide Section IV., Chapter IV.) Out-of-door life, recreation, change of scene, daily sponging of the body, sea-bathing, and all hygienic measures which have a favorable influence on assimilation and conduce to invigoration are useful.

The preparations of iron have a specially curative agency. Their *modus operandi*, with our present knowledge, cannot be satisfactorily explained. Doubtless the fact that iron is a constituent of the red corpuscles of the blood affords a partial explanation. Nothing in practical medicine is better established than the value of iron as a remedy in simple or benign anæmia. Its value is often attested by its efficacy even when the causes of the anæmia cannot be removed and when other circumstances are unfavorable. If in any case it have no curative effect, there is reason to suspect that the anæmia is not simple or benign. On the other hand, the efficacy of the remedy is evidence that the anæmia is of that character.

Of the different preparations of iron it is difficult to give decided preference to any one over the others. A physician is likely to have his favorite preparation, but that the choice is not based on the comparative merits of a considerable number is shown by the fact that almost every preparation has its advocates. Iron reduced by hydrogen, the citrate, the hypophosphite, the tincture of the chloride, the tartrate, the carbonate, and the compound mixture are all eligible preparations. It is a good plan to change from one preparation to another after a few weeks, giving thus several, or it may be many, of the different preparations in succession.

The chalybeate remedies should be given in moderate doses after meals—that is, during the process of digestion—and in order to secure their full efficacy they should be continued uninterruptedly for a considerable and perhaps for a long period. It is well to state beforehand to a patient that all the good which they may accomplish is not to be expected within a brief period. This statement will be likely to secure patience and perseverance. Patients often think that they do not tolerate iron in any form. In general, they are self-deceived in this regard, as may be demonstrated by their ability to take it with no inconvenience if it be given without their knowledge.

Arsenic is another remedy often of much efficacy in the treatment of anæmia. It should be given in small doses, which should not be increased, and it should be continued for a considerable period. The effect of this remedy is sometimes more marked than that of chalybeates.

Wine, especially Burgundy, is often useful. Here, as in other affections, alcoholics are of use only when they have a cordial effect, promoting assimilation and perhaps diminishing waste of tissues, without any excitant effects. Alcoholic excitation is always to be avoided, and the immediate effects, if in any way uncomfortable or disturbing, are contraindicating. Malt liquors sometimes agree better than wine. Spirits, as a rule, are not appropriate. As to the kind of wine or malt liquor to be preferred, experience in each case is to be the guide. Cod-liver oil, if it do not impair the appetite or disorder digestion, contributes to the reproduction of red corpuscles. If not well borne the extracts of malt may be substituted.

In cases of anæmia assurances of the absence of any grave disease and an encouraging prognosis are often of much weight as a therapeutical factor. Despondency is an effect of the anæmic condition upon the mind, and the physician in many cases has it in his power not only to remove gratuitous apprehensions, but to do not a little toward a cure by holding out proper expectations of recovery.

Chlorosis.

Although the chief element in chlorosis is undoubtedly anæmia, it presents certain peculiarities which render its separate consideration appropriate. The impoverishment of the blood in chlorosis has reference chiefly to the red corpuscles, and in a less degree to the albuminous constituents of the plasma.

There is a reduction in the quantity of hæmoglobin in the red corpuscles, either with or without a diminution in their number. It is especially in chlorosis that a reduction in the percentage of hæmoglobin has been found without a corresponding decrease in the number of red corpuscles. Cases have been observed in which the number of red corpuscles was normal, while less than one-half of the proper amount of hæmoglobin was present. This condition of disproportionate diminution of hæmoglobin is called achroiocythæmia or oligochromæmia. (See Part I. p. 60.) Usually, however, the number of corpuscles is lessened, as well as the percentage of hæmoglobin.

According to Virchow, an almost constant lesion in chlorosis is arrest of development of the vascular system, as indicated by the small size and the thin coats of the aorta and other arteries. The intima of the arteries sometimes contains yellowish spots and streaks in consequence of fatty degeneration. Most observers do not consider these irregularities in the vascular system as a constant and essential change in chlorosis. Arrest in development of the female generative organs has been observed in cases of chlorosis, but, on the other hand, they often present no abnormalities. No especial importance, therefore, can be assigned to anatomical changes in these parts as entering into the causation.

Aside from the foregoing differential points, the term chlorosis is applied to anæmia occurring in girls at or near the period of puberty, usually between the ages of fourteen and twenty-four, the causes often not being apparent. The term denotes a greenish coloration pertaining to the facies, but this is by no means a criterion. By some writers chlorosis is regarded as a neuropathic affection not always associated with anæmia. While anæmia is a constant element, there are doubtless causes which act upon the nervous system primarily and more directly than by inducing impoverishment of the blood. The development of the affection is evidently in some way connected with the evolution of the sexual system. How far the causation may involve, on the one hand, ungratified sexual desire, or, on the other hand, unnatural self-abuse, it is difficult to say. There is ground for the belief that each of these causative agencies is not infrequently operative. Disappointment in love, the loss of relations, and crosses of various kinds may be involved. These less apparent and other secret causes may act in conjunction with those already stated as giving rise to simple or benign anæmia. (Vide p. 367.) Blondes are said to be more liable to become chlorotic than brunettes. The affection, however, is not exclusively confined to girls. It is occasionally, owing probably to similar causative agencies, met with in boys.

The SYMPTOMS which have been enumerated as belonging to the clinical history of simple or benign anæmia are more or less marked in cases of chlorosis. Fantastic perversions of appetite are not uncommon in chlorotic girls, manifested by a craving for chalk, slate, charcoal, and other innutritive substances. These perversions are somewhat distinctive of chlorosis. The other points in the differentiation from simple or benign anæmia are those which have been stated as characteristic of the chlorotic affection—namely, the sex, the age, the absence of an obvious adequate causation, the probable connection with the sexual instinct, and in some instances the reduction in quantity of the hæmoglobin of the blood without diminution in the number of red corpuscles. As in simple or benign anæmia, the diagnosis requires an exclusion of the graver diseases of the hæmatopoietic system and of other diseases of which anæmia is a symptom.

The TREATMENT of chlorosis embraces measures, the same as in simple or benign anæmia, relating to diet, out-of-door life, etc., adapted to the circumstances proper to individual cases; and the preparations of iron, as a rule, are not less indicated and not less efficacious. The rules for their administration are the same. Arsenic is sometimes highly useful. Mental hygiene is of special importance in proportion as causative agencies relate to the mind. Agreeable diversion and the moral effect of change of scene are often important measures of treatment. Occupations and sentiments which will displace sexual thoughts and desires in some instances have a radical efficacy. This statement will include the instances in which self-abuse can be ascertained.

CHAPTER II.

LEUCOCYTHÆMIA.

THERE is a group of diseases characterized by a new growth of lymphatic tissue in various parts of the body. Lymphatic or adenoid tissue consists of reticulated connective tissue containing in its meshes lymphoid cells. This new lympathic growth may be homologous or heterologous. It is homologous when in the form of hyperplasia of pre-existent lymphatic tissue, as of the spleen, lymphatic glands, and marrow of bone. It is heterologous when lymphatic tissue is produced in situations where it does not normally exist, as in the liver, kidney, skin, etc. It has been proposed to call these diseases connected with hyperplasia of the lymphatic structures, lymphatic diseases. Common to all of these diseases is profound anæmia, generally progressive and fatal. The basis of classification usually adopted depends upon the number of white corpuscles present in the blood. When the pathological changes in the lymphatic tissues above mentioned are associated with marked increase in the number of white corpuscles, the name leucocythæmia is given to the disease. When the same pathological changes exist with anæmia, but without excess of white corpuscles, the disease is called pseudo-leucocythæmia. Pseudo-leucocythæmia includes splenic anæmia, lymphatic anæmia, and probably some cases of so-called progressive pernicious anæmia.

Leucocythæmia.

Leucocythæmia or leukæmia is a chronic disease characterized by a persistent and considerable increase in the number of white corpuscles in the blood. A temporary excess of white corpuscles, usually moderate in amount, is called leucocytosis. (See Part I. p. 65.) Alterations in the marrow of the bones, enlargement of the spleen, and hyperplasia of the lymphatic glands constitute, either singly or combined, the leading anatomical changes in leucocythæmia. Leucocythæmia is usually divided into three forms, called splenic, lymphatic, or medullary according as the changes in the spleen, lymphatic glands, or the marrow of the bones seem to be primary and predominant. The medullary form is also called myelogenic leucocythæmia. These forms are usually combined with each other to a greater or less extent, and hence the terms lymphatico-splenic, medullo-splenic, etc. These subdivisions of leucocythæmia are not associated with essential differences in the clinical history, and are of importance chiefly as indicating that the anatomical changes may predominate in certain organs. There has been much difference of opinion as to the part primarily affected in leucocythæmia. The prevailing view has been that the primary alteration is in the spleen, or more rarely in the lymphatic glands. Changes in the marrow of the bones have been found, as a rule, since attention was called to them by Neumann. This pathologist believes that they are constant and form the primary lesion. It seems, however, probable that the changes in the spleen, the lymphatic glands, and the marrow of the bones are co-ordinate, and in proportion to their degree of equal importance; but the changes may be extensive in one part and insignificant in the other parts.

The pathognomonic change in the *blood* may be detected during life in a drop drawn from the finger, or after death by examining the blood in the heart and vessels. The blood is paler than normal, and of a brownish-red, sometimes almost of a chocolate, color. The clots found in the heart and vessels are of a yellowish color in their superficial layers, and of a soft, viscous consistence, so that they bear some resemblance to very thick pus. The proportion of white corpuscles to red in health, although varying within wide limits, is on the average about 1 white to 500 red. In leucocythæmia there is often 1 white to 10 or 8 of the red, and there may be even 1 white to 3 or 2 red. In one case related by Sörensen there were 3 white to 2 red. In the great majority of cases the proportion during the course of the disease exceeds 1 white to 20 red corpuscles. Although in general the white corpuscles in leucocythæmia resemble the ordinary white blood-corpuscles, there are certain differences to be noted. In addition to the ordinary polynuclear white blood-corpuscles, leukæmic blood often contains a large number of mononuclear corpuscles, of which some are small with a narrow rim of protoplasm, like lymph-cells, while others are large with a broad mantle of protoplasm, like cells in the spleen and in the marrow of the bones. According to Virchow, an abundance of small corpuscles is indicative of lymphatic leukæmia, and an abundance of large corpuscles, of splenic leukæmia; but there are doubts as to the correctness of this view. Most of the leucocytes in leukæmia are more sluggish in their amœboid movements than ordinary white blood-corpuscles.[1] Nucleated red blood-corpuscles may be present in leukæmic blood, although not often in large number. The blood-plates are sometimes increased in number, but they may be fewer than normal. The number of white blood-corpuscles may vary considerably within a short time. Eisenlohr describes a case in which the excess of white corpuscles nearly disappeared during a febrile attack, to return again after its subsidence.

The number of red blood-corpuscles is usually considerably diminished, although exceptionally it may be normal. The red corpuscles may be reduced to one or two millions, or even less, per cubic millimetre. The red corpuscles are usually of normal appearance, but they may present the various changes described in the article on Anæmia in Part I. (p. 60).

The fresh blood is always alkaline, but it has a tendency to rapidly assume an acid reaction. The specific gravity of the blood is lessened. The amount of fibrin is said to be increased.

Some degree of splenic enlargement is usually present in all forms of leucocythæmia. In *splenic* leucocythæmia, which is the most frequent variety, the enlargement is often extreme. According to Gowers,[1] the average weight of the spleen in splenic leucocythæmia is a little less than 6 pounds, and its average length, 11¼ inches. Leucocythæmic spleens weighing 16 and 18 pounds have been recorded. The increase in size is usually uniform in all directions. The notches upon the anterior border, the recognition of which may aid in the diagnosis, are often exaggerated. The capsule of the spleen frequently presents thickened white patches. There may be adhesions between the surface of the spleen and surrounding parts. The consistence of the organ is usually increased, but it may be soft, especially in the early stages. The cut surface of the spleen may be uniformly brownish-red in color, or the Malpighian bodies may be prominent as whitish dots, or, when greatly enlarged, they may even appear as tumors in the substance of the spleen. The whitish trabeculæ may be much thickened, especially in the late stages. The spleen sometimes presents on section a mottled appearance from the presence of dark-red or yellowish, wedge-shaped infarctions. These are ascribed to obstruction of certain of the splenic vessels by the accumulation of white blood-corpuscles. Softened spots due to hemorrhage and the disintegration of the splenic pulp or to collections of pus have been met with in leukæmic spleens. Microscopical examination shows the change to consist in a hyperplasia of the organ; that is, in an increase of the normal elements of the spleen. The hyperplasia affects chiefly the splenic pulp, but sometimes to a greater extent the Malpighian bodies. Large cells containing coarse granules either of pigment or of fat are common in leucocythæmic spleens.

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[1] Other peculiarities of the white corpuscles in leukæmia have been noted, especially by Ehrlich. Some of the corpuscles may contain molecules of fat, others, granules of brown pigment. There is an increased number of corpuscles containing granules which stain with eosin (eosinophilic cells). There may be corpuscles containing granules which stain with basic aniline dyes (Mastzellen). These do not occur normally in the blood. The mononuclear cells may contain granules which stain with neutral aniline dyes, like granules normally found in polynuclear cells (neutrophilic granules). It has been suggested that many of the corpuscles are not white blood-corpuscles, but are colorless corpuscles, such as are intended normally for the formation of nucleated red blood-corpuscles in the medulla of bone.

In the splenic and medullary forms of leucocythæmia, the *lymphatic glands* are often more or less enlarged, and in the lymphatic variety an excessive hyperplasia of the glands is an early symptom. The abdominal, inguinal, cervical, and axillary glands are those most frequently affected. The single glands may attain the size of a hen's egg, but they are usually smaller. Their consistence is soft, rarely hard, and their color gray or reddish-gray. The microscope shows an adenoid reticulum infiltrated with lymphoid cells, as in the normal glands. Allied to the affection of the lymphatic glands there is sometimes a similar hyperplasia of the follicles of the tongue and pharynx, of the tonsils, and especially of the solitary follicles and Peyer's patches in the intestine.

The change in the *marrow of the bones* is to be included among the hyperplasias of lymphatic structures. The change consists in the disappearance, to a greater or less extent, of the fatty medulla normally present in the long bones of adults, and its replacement by a tissue rich in lymphatic cells. Alterations have usually been found in the long bones, the ribs, the sternum, and the vertebræ. The altered marrow has either a greenish-yellow, purulent aspect, or a dark-red appearance resembling that present in the bones of embryos and infants and in profound anæmias. Neumann calls the greenish-yellow transformation *pyoid*, and the dark-red, *lymph-adenoid*. There is less difference in the microscopical structure than the gross appearances would suggest. In the pyoid alteration, which is the more common, the yellowish color becomes somewhat reddened by exposure to the air. The marrow is of a soft, pulpy consistence like that of creamy pus, but there is usually no real breaking down into pus, as a net-like reticulum can be demonstrated between the cells. In the lymph-adenoid change the marrow is dark red in color, and of a gelatinous consistence firmer than that of the pyoid marrow. The dark-red and the yellowish colors may be found side by side. The reticulum between the cells is better developed in the dark-red than in the pyoid marrow. In both there is a rich capillary network. In both the cells resemble the normal medullary cells and those present in the blood. Sometimes large granular, at other times smaller and less granular, cells predominate. Nucleated red corpuscles are present, but not usually in large number. The hyperplasia, when excessive, is at the expense of the surrounding bone-substance, which thereby becomes more or less rarefied. The sternum in rare instances may become softened through its entire thickness, and swollen by hyperplasia of the marrow and absorption of bone. This softening may be detected during life. Similar changes have been observed in the ribs and other bones during life. These changes in the marrow have been studied most carefully by Neumann, who regards them as the most constant and important of the pathological changes in leucocythæmia. Cases have been reported in which the changes in the marrow seemed insufficient to account for the alteration in the blood.[1]

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[1] Article "Splenic Leucocythæmia," in *Reynolds's System of Medicine.*

Changes are also observed in leucocythæmia in *non-lymphatic organs.* These changes are either diffuse or circumscribed and nodular. The diffuse alteration consists in the distension of capillaries and the infiltration of the tissues with white blood-corpuscles. The circumscribed changes, which are not common, consist in the nodular accumulation of leucocytes either with or without a new growth of adenoid reticulum between the cells. To these nodular accumulations the names heteroplastic leucocythæmic growths, lymphomata, and lymph-adenomata are applied.

The liver is enlarged in the majority of cases, especially those of the splenic variety. A weight of six or seven pounds is not uncommon. Virchow describes leukæmic livers weighing from eight to fourteen pounds. The enlargement is due to a distension of the capillaries with leucocytes, and to the infiltration of the tissue between and within the lobules with these cells, probably in consequence of their escape from the vessels. Disseminated whitish nodules, usually minute, but sometimes of considerable size, consisting of lymphoid cells, are frequently found in the interlobular tissue. These nodules are called lymph-adenomata when an adenoid reticulum can be demonstrated between the cells.

The kidneys are not infrequently enlarged. The changes are diffuse and circumscribed as in the liver. Parenchymatous alterations may or may not be present. Tizzoni has described infiltration and nodules of lymphoid cells in the testicles. Leucocythæmic growths may furthermore be scattered through different parts of the body, as the alimentary tract, the lungs, the serous membranes, the brain and the retina, the skin, etc. Nuclear figures indicating cell-proliferation have been found in the lymphoid cells in these growths.

Next to these accumulations of lymphoid cells, the frequent occurrence of hemorrhages in leucocythæmia deserves mention. Subserous ecchymoses, hemorrhages into serous cavities and from mucous membranes, and retinal and cerebral hemorrhages may be mentioned as frequent and important. The gums and the mucous membrane of the mouth and pharynx may be swollen and ulcerated. The lymphoid growths in the intestine may ulcerate. Pronounced fatty degeneration, especially of the heart and kidneys, has been repeatedly observed.

Various abnormal substances have been found in leucocythæmic blood. These are bodies analogous to glutin, hypoxanthin, leucin, tyrosin, and acetic and formic acids. These substances have not been uniformly discovered. Some are normally present in the spleen. There are frequently found after death minute colorless, elongated, octahedral crystals called Charcot's crystals. They have been detected in the blood, the spleen, the marrow of the bones, the liver, and even in other parts. They are thought to be of an albuminous composition. Although most abundant in leucocythæmia, they have been found in other conditions, and they may form after death in healthy marrow.

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[1] Fleischer and Penzoldt have reported a case of lymphatic leucocythæmia without change in the marrow of the bones. Leube and Fleischer have reported a case of leukæmia with lymph-adenoid marrow, but without changes in the spleen and lymphatic glands. Neumann also has observed cases in which the medulla of the bones was involved without affection of the spleen or lymphatic glands.

There have been several theories as to the relation in leucocythæmia between the morbid change in the blood and the hyperplasia of the lymphatic structures. Bennett, to whom we owe the first recognition, in 1845, of leucocythæmia as a distinct disease, maintained that the change in the blood was primary; and the same view has had its advocates in recent years. Most modern pathologists, however, have accepted Virchow's doctrine that the increase of white corpuscles in the blood is secondary to the pathological changes in the lymphatic organs to which is assigned a blood-forming function. Some have emphasized chiefly the affection of the spleen, and others, that of the marrow of the bones. According to one theory, there is an insufficient transformation of white into red corpuscles in the blood-forming organs and in the blood; according to another, there is increased production of white corpuscles, an exaggeration of the cytogenic function. The most probable view seems to be that there is increased formation of white corpuscles in the spleen, the lymphatic glands, and in the marrow of the bones, sometimes chiefly in one, and sometimes in all of these organs. From these hyperplastic organs the corpuscles are swept in large numbers into the blood and the lymphatic currents. Either the production of red corpuscles is diminished or their destruction is increased.[1]

The development of leucocythæmia, as determined by SYMPTOMS, is usually slow and imperceptible. Before the character of the disease is determinable general debility, indisposition to exertion, disorders of digestion, and other indefinite ailments have existed for one or two years, and sometimes for a much longer period. In exceptional cases, oftener in children than in adults, it is developed within a few weeks. Early and prominent symptoms are those referable to impoverishment of the blood. Pallor of the face, prolabia, and mucous membranes, and dark coloration of the eyeballs, are more or less marked, as in cases of benign anæmia and chlorosis, for which the disease in its early stages is liable to be mistaken.

Coincident with the anæmic symptoms is enlargement of the spleen. This enlargement, if not great, may not have attracted the attention of the patient. The enlargement, however, is often so great as to form a splenic tumor which occasions a projection obvious to the eye. The tumor may occupy a considerable part of the abdominal space. It has been mistaken for an ovarian tumor. It may cause dislocation of the heart, and, by pressure on the diaphragm, interference with respiration. Pain and tenderness referable to the enlarged spleen are frequent but not constant symptoms.

In the so-called lymphatico-splenic variety there is swelling of the glands of the neck, groin, and axilla, forming soft, movable tumors rarely of much size. The mesenteric glands are sometimes enlarged, so as to be felt through the abdominal walls. The bronchial glands may be enlarged, but rarely to an extent sufficient to occasion notable bronchial obstruction. The glandular swelling may occur either early or late in the progress of the disease. If the disease involve the marrow of bones (medullo-splenic and myelogenic leucocythæmia), there may be found over these, particularly the sternum, tenderness and sometimes swelling, but, as a rule, local symptoms are wanting.

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[1] The idea has been advanced that leucocythæmia and pseudo-leucocythæmia are infectious diseases dependent upon some special organism in the blood. Some pathologists class the lymphomatous growths of these diseases among the infectious tumors. This idea, which is not without some probability, is at present purely an hypothesis.

Dyspnœa is a symptom more or less prominent, attributable to the paucity of red corpuscles. It may be caused, in addition, by enlargement of the spleen and also of the liver, the latter occurring frequently. The dyspnœa is increased, and may be rendered extreme by hydrothorax in connection with general dropsy and in some instances pleurisy with effusion.

General dropsy occurs sooner or later in a large proportion of cases. It may be independent of cardiac or renal disease, but the latter is a complication in some cases. Hydro-peritoneum is sometimes produced by the pressure of enlarged glands on the vena portæ, and the accumulation of liquid may be so large as to call for paracentesis.

The action of the heart is increased in frequency, often considerably, and paroxysms of palpitation, during which the pulse is 150 or more per minute, are not uncommon. Hæmic murmurs are common, as in benign anæmia and chlorosis. The temperature of the body is in most cases raised. Different cases differ in respect of the amount of pyrexia. It is generally moderate (from 100° to 102° Fahr.). Occasionally there is hyperpyrexia. Usually, there are febrile exacerbations in the evening. The temperature is found to fluctuate between wide limits at different times in the same case during the progress of the disease. The course of the disease is rapid in proportion to the degree of persistent pyrexia.

The liability to hemorrhage is such that a hemorrhagic diathesis may be said to constitute a prominent feature of the disease. Mosler emphasizes the importance of recognizing this diathesis as contraindicating surgical operations in cases of leucocythæmia. He relates a case in which the patient nearly died from an insignificant operation, the bleeding having been stopped only by continuous digital compression.[1] Hemorrhages may occur early in the disease. The most frequent seat is the nose. Epistaxis may prove the immediate cause of death. Enterorrhagia, gastrorrhagia, and menorrhagia are not rare. Bronchorrhagia and hæmaturia have been observed. Cerebral hemorrhage is a cause of death in some cases. Cutaneous ecchymoses are among the events incident to the disease, and extravasation beneath the skin, in muscles, inclusive of the heart, and into joints. Eisenlohr has reported a case in which peripheral paralysis of several cranial nerves was caused by hemorrhage into their sheaths.[2] Friedländer[3] has reported a case in which the symptoms of cerebral tumor were caused by leucocythæmic growths in the brain.

Among events pertaining to the digestive system may be mentioned inflammation of the pharynx and mouth, accompanied sometimes with ulcerations and sometimes with bleeding from surfaces not ulcerated. Tumors of the character of lymphoma are sometimes developed in the throat. The appetite varies in different cases, and at different periods in the same case. It may not at first be diminished, and it may even be inordinate; but in the latter part of the disease there is generally anorexia. Vomiting is an infrequent symptom. Diarrhœa is a frequent symptom, and the dejections are sometimes dysenteric. Jaundice is an occasional event.

The urine has an excess of uric acid. Hypoxanthin, formic acid, and lactic acid have been found in small quantities—not, however, constantly. Albumen in any considerable quantity and casts denote the existence of renal disease as a complication.

Salzer was the first to call attention to persistent priapism without seminal emissions or sexual desire as an occasional symptom in leucocythæmia.[1] Others have observed this symptom. Dr. G. L. Peabody has reported a case.[2] Another case has been reported by Dr. G. F. Wetherell.[3]

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[1] "Ueber hämorrhägische Diathese bei Leukämie," *Zeitschrift für klin. Medicin*, Bd. 1, p. 265. He observed a case in which priapism lasted seven weeks, and when this symptom disappeared the power of erection was lost.

[2] *Virchow's Archiv*, Bd. 73.

[3] *Ibid.*, Bd. 77.

The following morbid appearances in the retina have been observed by means of the ophthalmoscope: A pale appearance of the retinal vessels, hemorrhagic spots with white centres from an accumulation of lymphoid cells, whitish streaks, and cloudiness of vessels about the optic papilla. The name leucocythæmic retinitis has been applied to these changes. They may or may not give rise to notable disturbance of vision. The retinal hemorrhage may be sufficient to cause blindness. Headache, vertigo, feebleness of the mental faculties, delirium, and chronic derangement of mind occasionally enter into the clinical history. To these may be added impairment of hearing, furuncles, and pustular eruptions on the skin. In a case of leukæmia with deafness Politzer found leukæmic growths in the internal ear.

The limits of the *duration* of leucocythæmia are far apart. In very rare instances it runs a rapid course, ending within a few weeks. On the other hand, its duration may extend over many years. The average duration is about two years. It is not always easy to ascertain the duration, owing to difficulty of fixing the precise date of the origin of the disease. The termination often depends on events which are incidental to the disease, or on complications, such as hemorrhage, apoplexy from extravasation, pleurisy, renal disease, diarrhœa, etc. If not dependent on any of these events, death is produced by slow asthenia.

The PROGNOSIS is as grave as possible after the disease has made a certain degree of progress. In some instances, early in the course it has ceased to be progressive and recovery has taken place. If, with great blood-changes, the spleen becomes much enlarged, and more or less of the grave events belonging to the clinical history have taken place, there is little or no ground, with our present knowledge, for any expectation of recovery. The most to be hoped for is that the disease will progress slowly, and that events which involve immediate danger may not occur. There are occasionally marked remissions in the progress of the disease.

. The clinical history of leucocythæmia from the beginning to the end of the career of the disease contains diagnostic characters, but these are not sufficiently distinctive in all cases for the differentiation from pseudo-leucocythæmia and pernicious anæmia. In an early period of the disease, when a DIAGNOSIS is desirable, it is liable to be mistaken for other diseases in which anæmia coexists with splenic enlargement. A positive diagnosis, however, is always practicable by means of examinations of the blood. Delay or failure in the recognition of the disease is owing to neglect of the means by which the diagnosis is to be made.

The diagnostic criterion is a persistent and progressive increase of the white corpuscles of the blood beyond the limit of the variations embraced under the name leucocytosis. This name embraces a greater or less increase of the white corpuscles, occurring as a symptom in various diseases, the increase falling short of that which constitutes the criterion of leucocythæmia. (Vide Part I. p. 65.) The rule generally accepted as regards the increase requisite for the diagnosis is a proportion of 1 white corpuscle to 20 red corpuscles. As already stated, the proportion is often much greater than this, the number of white being sometimes equal to that of the red corpuscles.

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[1] *Berl. klin. Wochenschr.*, 1879.

[2] *N. Y. Med. Journ.*, May, 1880.

[3] *N. Y. Med. Record*, Aug. 14, 1880. Two other cases are reported in the *N. Y. Med Journal*, Sept., 1880.

On the other hand, in the early part of the disease the proportion may be considerably less than 1 in 20; and if on repeated examinations the proportion be found to progressively increase, there can hardly be doubt as regards the diagnosis.

The method of examination ordinarily employed is to obtain a drop of blood by pricking the finger of the patient, and, placing it in the field of the microscope, to estimate with the eye the relative proportion of the white and red corpuscles. A more accurate method is to add to a measured quantity of blood a definite quantity of a 5 or 6 per cent. solution of sulphate of soda, in order to dilute the blood and to prevent the red corpuscles from forming rouleaux. The field of the microscope is divided into squares, by the aid of which the corpuscles can be easily counted. The squares are ruled either upon the eye-piece or upon the cell containing the diluted blood.[1] It is to be observed that the relative proportion of the white and red corpuscles does not show the degree of actual increase of the former, inasmuch as the red corpuscles are almost always considerably, and sometimes very greatly, reduced in number. Hence, the most exact method is to ascertain the number both of white and red corpuscles in a given quantity of blood by means of the hæmacytometer.

Leucocythæmia occurs at all periods of life, it having been observed in infants soon after birth and in extreme old age. In the great majority of cases, however, patients are between twenty and fifty years of age. It is less rare in men than in women. Social position has no marked influence on its causation. It occurs in women sufficiently often after parturition to show some causative connection. In a considerable proportion of cases patients with this disease have either had malarial fever or have been exposed to malaria. The disease is less rare in malarial districts than in those in which the periodical fevers do not occur. It is therefore reasonable to attribute to malaria a certain degree of causative agency. The white corpuscles have been found in some cases to contain black pigment-granules, such as are characteristic of malarial disease. Mosler applies to these cases the name *melano-leukæmia.*

The disease in a few instances has been preceded by local injury in the splenic region. There is little or no ground for attributing the disease to syphilis. In most cases the disease is developed in persons who at the time of its development were apparently in good health. These statements express all that is at present known of the etiology.

Diminution of the volume of the spleen has been thought to be an important object of TREATMENT, under the supposition that the leucocythæmia will be diminished in proportion. With reference to this object, various measures have been employed—namely, quinia in full doses, hypodermic injections of ergotin, the iodide of potassium, the cold douche, ice applied over the spleen, and the application of electricity. Each of these measures has appeared in some cases to have produced in a limited degree the desired effect, but in other cases to be useless. It cannot be said that clinical experience has established the therapeutic value of any measure employed for the object just stated. Extirpation of the spleen has been resorted to in at least 16 cases, in all, save one, with a fatal result, death resulting from either hemorrhage or peritonitis.[2] The liability to fatal hemorrhage is enough to render the operation unwarrantable. Were the operation warrantable, or were it practicable to reduce considerably, by any measure of treatment, the size of the spleen, it is by no means certain that the leucocythæmia would be controlled; the supposed pathological connection between the latter and the splenic enlargement being theoretical. Moreover, the gravity of the disease does not depend on the excess of the white corpuscles in the blood. In pseudo-leucocythæmia and pernicious anæmia, kindred diseases in which there is not a considerable excess of white corpuscles, the gravity is not less than in leucocythæmia.

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[1] A convenient apparatus for counting the corpuscles is the hæmacytometer of Gowers. Still more exact is the apparatus of Zeiss.

[2] One successful case of extirpation of the spleen in leucocythæmia has been reported in Italy.

Neither arsenic nor phosphorus nor any remedy has as yet been found to have a curative influence over this disease. The treatment, therefore, resolves itself into meeting symptomatic indications, and adopting means, medicinal and hygienic, which may succeed in arresting the progress of the disease, improving the general condition of the patient, and prolonging life. These objects of treatment will embrace the prolonged use of preparations of iron, the various tonics which are likely to improve appetite and promote digestion, hæmostatic remedies and appliances, an alimentation as abundant and nutritious as possible, cod-liver oil as a nutrient, favorable climatic influences, out-of-door life, mental diversion, and all available sanitary measures with refence to nutrition and general invigoration.

CHAPTER III.

PSEUDO-LEUCOCYTHÆMIA—HODGKIN'S DISEASE.

PSEUDO-LEUCOCYTHÆMIA is the name of an affection in which anatomical changes similar to those in leucocythæmia are present, without an increase of white corpuscles in the blood. In many cases the anatomical changes are apparently identical in both diseases. In such cases it seems unscientific to make one symptom—namely, the presence of an excess of white corpuscles—the basis of classification. In justification of this, however, it may be said that the difference in the state of the blood not improbably depends upon anatomical conditions which have not yet been recognized. Lymphatic, splenic, and perhaps medullary varieties may be distinguished in pseudo-leucocythæmia upon the same grounds as in leucocythæmia. In the majority of cases the lymphatic glands are primarily and most extensively involved. Hence the lymphatic form is the one generally meant when the term pseudo-leucocythæmia is used without qualification. A medullary variety has not been recognized generally, but certain cases of progressive pernicious anæmia probably belong to this subdivision. To these reference will be made in the next article. In splenic pseudo-leucocythæmia the spleen is the part mainly affected. This variety has been called also splenic anæmia and simply hypertrophy or tumor of the spleen. It is not to be confounded with the chronic enlargement of the spleen due to malarial infection. In splenic pseudo-leucocythæmia the lymphatic glands are usually, if not always, involved, but to a less extent than in the lymphatic variety of the disease. As the affection of the spleen and that of the lymphatic glands are usually combined, it is no more possible to draw a sharp line of distinction between the splenic and the lymphatic variety than between the same forms of leucocythæmia. While the splenic variety of leucocythæmia is much more common than the lymphatic, the reverse is true in pseudo-leucocythæmia. The anatomical changes in the spleen are similar to those in leucocythæmia, and need not be again described. A nodular enlargement of the Malpighian follicles is more frequent than in leucocythæmia.

The following names are in use to designate the lymphatic or most frequent variety of pseudo-leucocythæmia: Hodgkin's disease,[1] adenia (Trousseau), lymphatic anæmia (Wilks), malignant lympho-sarcoma (Langhans), malignant lymphoma (Billroth), and lymph-adenosis (Ranvier, Gowers). The name pseudo-leucocythæmia was introduced by Cohnheim. It is probable that a number of different pathological conditions have also been described under these names, especially the term lympho-sarcoma. Scrofulous enlargements of the lymphatic glands, enlargements confined to a single group of glands, and the rare cases of primary sarcoma beginning in one group of glands and forming metastases in various parts of the body, but not by preference in other lymphatic glands, are to be excluded from the disease under consideration. In this affection several groups of glands, sometimes nearly all in the body, are involved. In a large proportion of cases the spleen and the liver are enlarged. Secondary deposits may be present in most parts of the body.

The change in the lymphatic glands consists in an increase of the cells and of the reticulum. Virchow and later writers distinguish two forms of lymph-adenoma, the soft and the hard. In the soft variety the glands are large, soft, almost fluctuating, of a gray or reddish-gray color, and often filled with ecchymoses. The enlargement is due to a new growth of lymphoid cells, between which is a delicate reticulum with wide meshes. In the hard form the consistence of the swollen gland is firm, the color grayish or yellowish-white, and with the hyperplasia of the cells there is a new growth and a thickening of the reticulum or stroma, denser and more marked than in the soft lympho-sarcoma. There are transitional forms between the hard and the soft variety. In many cases the hard is a later stage of the soft enlargement. In the same case the characters are not always uniform. Some glands may be hard and others soft. It is therefore not desirable to make a sharp distinction between hard and soft lymph-adenoma, although there are undoubtedly cases in which the hyperplasia of the reticulum predominates from the onset, the glands in no stage of the affection having been soft. It is the soft variety which corresponds more closely, in the anatomical appearances of the lymphatic glands, with leucocythæmia. The enlarged glands may reach the size of a hen's egg. They may remain distinct or may become compacted together into a nodular mass, in which the individual glands can no longer be distinguished. In the latter case there has been either a periadenitis or an extension of the lymphatic growth into the capsule of the gland and into the surrounding connective tissue. The skin over the swollen glands is, as a rule, freely movable, rarely adherent. Upon section of the glands the normal distinction between cortex and medulla is usually obliterated. In the harder tumors, often shining and more or less hyaline masses of fibroid tissue may be detected. Fatty and caseous degenerations are very rare, a circumstance which distinguishes lymph-adenomatous from scrofulous enlargements. The affection begins oftenest in the cervical glands. It has a tendency to involve especially the glands along the great vessels; hence, the retro-peritoneal glands are more frequently affected than the mesenteric. The starting-point of the disease may be in the mediastinal glands.[1] The affection may remain for a considerable time stationary in one group of glands without impairment of the general health, and then rapidly increase and become generalized, with the development of marked cachexia. The glands may attain a large size without making compression upon surrounding blood-vessels, but in some cases there are severe symptoms resulting from pressure on the vessels, the air-passages, etc.

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[1] Hodgkin published his observations in 1832 (*Medico-Chirurgical Trans.*, vol. xvii. p. 69), before leucocythæmia was known. He first called attention to coincident enlargement of the spleen and of the lymphatic glands as a distinct disease. His description included, undoubtedly, cases both of leucocythæmia and of pseudo-leucocythæmia. Some writers apply the name Hodgkin's disease to lymphatic leucocythæmia as well as to pseudo-leucocythæmia.

The spleen is enlarged, according to Gowers, in four-fifths of the cases. The increase in size may be enormous, but, as a rule, it is much less than in leucocythæmia. In the smaller number of cases the appearance is that of a diffuse hypertrophy of the splenic pulp; in the majority there are disseminated whitish growths in the form of nodules throughout the spleen. These are probably hypertrophied Malpighian follicles and growths in the periarterial sheaths. Infarctions may be observed as in leucocythæmic spleens. In the liver there may be lymphoid nodules or a diffuse infiltration of the interlobular tissue with lymphoid cells. In the latter case grayish streaks surround the acini. The hepatic capillaries may be distended with blood. As pointed out by Schulz, the secondary lymphoid formations in the different parts of the body involve especially the walls of the vessels. These secondary growths have been met with in nearly all parts of the body, as the kidneys, the serous membranes, the testicles, the lungs, the brain, the retina, the skin, etc. The thymus, the solitary follicles, and Peyer's patches may be diseased, as in leucocythæmia. The marrow of the bones may present a reddish appearance, as in pernicious anæmia, and may contain lymphomata. It rarely shows the pyoid alteration found in leukæmia.

We are at present unable to explain the relations between pseudo-leucocythæmia and leucocythæmia. We do not know why apparently identical changes in the lymphatic structures should be attended in one case by increase of white corpuscles in the blood, and not in another case. The theory that in pseudo-leucocythæmia there is some obstacle to the passage of cells from the hyperplastic lymphatic glands into the blood- or lymph-current is plausible, but is not supported by any anatomical facts. Ackermann[2] has succeeded in injecting the afferent and the efferent lymphatic vessels of both the soft and the hard glands. He finds them pervious. The fact that there may be the same changes in the spleen, lymphatic glands, and in other parts in pseudo-leucocythæmia as in leucocythæmia is evidence against the view of those who hold that in leucocythæmia these changes are the result of a primary increase of white corpuscles in the blood. In some cases, especially of hard lymph-adenoma, the origin of the growth appears to be local; but in the majority of cases several groups of glands are affected simultaneously or in rapid succession. In such cases a primary dyscrasia is assumed to exist.

The SYMPTOMATOLOGY of lymphatic pseudo-leucocythæmia embraces the events which belong to the clinical history of leucocythæmia—namely, general debility, feebleness of the circulation, hemorrhages, general dropsy, pleuritis, and other complications, together with the varied phenomena referable to persistent progressive anæmia. A distinctive feature is the occurrence, uniformly earlier and more extensive, of enlargement of lymphatic glands, and often the development of lymphoid tumors where these glands are not normally discoverable. The cervical glands are affected in a great majority of cases, and in most of these cases primarily. The aggregated enlarged glands may attain to a large size, causing much deformity. Next in frequency the axillary glands are affected. The glands in the anterior and posterior mediastinum, the bronchial, the cardiac, the retro-peritoneal, the mesenteric, and the inguinal glands, may be, severally or collectively, the seats of the local affection. Lymphatic growths may be found in the tonsils, the thorax, the soft palate, the posterior part of the tongue, the œsophagus, and beneath the skin. Perhaps the most remarkable case on record as regards the extensive distribution of these growths or tumors was reported by Dr. James H. Hutchinson.[1] In this case, exclusive of enlarged cervical glands, of a tumor of the size of a hazel-nut on the upper eyelid, and of a large tumor involving the sternum and the anterior mediastinum, two hundred and thirty-nine tumors, ranging in size from that of a small pea to half a walnut, were counted on the surface of the body. There were twenty tumors on the surface of the brain. A tumor surrounded the bodies of the sixth, seventh, and eighth dorsal vertebræ, and extended within the spinal cord, causing complete paraplegia. Three hundred small tumors were counted within the lungs. Small tumors were scattered over the peritoneal covering of the intestines. It is noteworthy that in this case the spleen and liver were healthy. An unusual feature was the existence of intracranial lymphatic growths.

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[1] R. Schulz (*Arch. d. Heilkunde*, Jahrg. 15, p. 223, 1874) has analyzed 38 cases with reference to the frequency with which different groups of glands are affected. The cervical glands were affected in 28 cases, the retro-peritoneal in 21, the inguinal in 20, the mediastinal in 18, the bronchial in 14, the mesenteric in 14, the lumbar in 12, the axillary in 9, the cubital in 4, the popliteal in 1.

[2] "Ueber die malignen Lymphome," *Diss. Bern.*, 1879.

The pressure of the tumors in different situations may give rise to important symptoms. Pressure upon the larynx or trachea may obstruct respiration, and death is sometimes due to suffocation. Paralysis of the laryngeal muscles may be caused by pressure upon the recurrent laryngeal nerves. Obstruction of the bronchi may be caused by enlarged bronchial glands. The heart is sometimes dislocated. The œsophagus in rare instances is obstructed. The cervical tumors may diminish the arterial supply to the brain by pressing upon the carotids, or they may induce venous congestion by pressure upon the jugular veins or the vena cava. Deafness is not infrequently produced by growths within the pharynx. The pneumogastric nerve may be involved in a tumor, and the action of the heart be thereby retarded. Pain in the upper or lower extremities may result from pressure upon the nerves in the axillary and inguinal regions. The femoral vein may be compressed and œdema of the lower limb follow. Jaundice is sometimes attributable to pressure upon the bile-duct. Gastric disturbances and diarrhœa may be symptomatic of lymphoid growths in the stomach and intestines.

The *duration* of pseudo-leucocythæmia, like that of leucocythæmia, varies from a few weeks to many years. Of 42 cases analyzed by Dr. Hutchinson, in 29 death took place within two years, and in 34 within three years. The average duration is a little less than that of leucocythæmia.

As regards CAUSATION, as little is known of this disease as of leucocythæmia. In the larger number of cases the age is between ten and thirty years, but the disease sometimes occurs in childhood and in extreme old age. A decided influence pertains to sex, a large majority of patients being males. An etiological agency referable to antecedent diseases or to habits of life or to any appreciable morbid influences has not been established. The disease often occurs in persons who at the time of its development apparently were perfectly well.

The DIAGNOSIS of lymphatic pseudo-leucocythæmia is based upon symptoms common to this disease and to leucocythæmia, taken in connection with the affection of the lymphatic glands and the fact that there is not an increase in the white corpuscles of the blood sufficient to constitute the leucocythæmic condition. The affected lymphatic glands do not undergo the processes of necrosis and suppuration which characterize scrofula; nor are there the gradual invasion of adjacent parts and the ulcerations which characterize the local history of carcinoma.

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[1] Vide *Trans. College of Physicians of Philadelphia*, 3d Series, vol. i., 1875.

The PROGNOSIS is no better than in cases of leucocythæmia. The supposed instances of recovery in an early stage are open to the suspicion of an error in diagnosis. There are probably very few if any exceptions to the rule of a fatal termination when the disease is so far advanced that the diagnosis is positive. The progress of the disease may be either slow or rapid. It is rapid in cases with pyrexia, profuse hemorrhage, and considerable anasarca. The extent of the distribution and the degree of the glandular growths constitute a measure of the tendency to a rapid progress. The immediate cause of death may be an important complication, such as disease of the kidneys, purulent pleuritis, etc. The pressure of the tumors on important parts may lead to the fatal ending. Irrespective of these causes, the disease proves fatal by slow asthenia.

Measures of TREATMENT have been directed especially to the affected glands. The external application of iodine, the employment of cold and heat, frictions, blisters, the injection into the tumors of iodine, nitrate of silver, carbolic acid, arsenic, and other topical remedies, and galvano-puncture, have all been tried, with but little if any satisfactory result. Excision of the affected glands has been repeatedly resorted to, but the efficacy of this measure has not been established. The removal of the tumors or their dispersion by any local treatment would not strike at the root of the disease. An unknown essential pathological condition underlies the affection of the glands. This condition cannot be reached by any therapeutical appliances as yet ascertained. Excision, however, of enlarged glands may be advisable with a view to relieve parts, such as the larynx or trachea, sensory nerves, etc., from pressure. The internal use of arsenic, the doses increased to the utmost limit of toleration, and conjoined with the injection of Fowler's solution into the affected glands, has been advocated by Winiwarter as a useful method of treatment, but the utility which he attributed to it has not been confirmed.

Aside from any treatment instituted with the hope of effecting a cure, the objects and the measures are those stated in connection with leucocythæmia.

CHAPTER IV.

PERNICIOUS ANÆMIA.

THE name pernicious anæmia is given to a severe, often fatal, form of anæmia for which we are unable to assign a sufficient cause either before or after death. The disease has also been called idiopathic or essential anæmia. This form of anæmia was first described by Walter Channing of Boston in 1842. Subsequently, Addison described idiopathic anæmia clearly and concisely. Since Biermer's publication upon progressive pernicious anæmia in 1872 special attention has been directed to this disease.

Pernicious anæmia may develop without any apparent cause, in persons previously healthy and in good hygienic surroundings. More frequently it is secondary to conditions which ordinarily impair the health or produce simple anæmia. Thus, pernicious anæmia has been observed especially among those poorly housed, scantily clad, or imperfectly nourished; and also during or after pregnancy, particularly after repeated pregnancies. Further causes which have been noticed are—exhausting discharges, such as diarrhœa, repeated hemorrhages, mental anxiety, and excessive mental or physical exertion. The disease has been observed to follow typhoid fever. Endemic influences have also been thought to be of some importance in the etiology of this disease. An unusually large number of cases has been reported from Switzerland and certain parts of Germany. In this country it seems to be less common, although it is by no means a rare disease. Excluding cases dependent upon child-bearing, pernicious anæmia is as common among men as among women. The disease is rare before twenty or after sixty years of age.

It is important to note that the causes which have been mentioned, although they may explain a certain amount of anæmia, do not account for the pernicious character of the anæmia. To explain this, it is necessary to assume a profound disturbance of the blood-forming organs—a disturbance which may be either an excessive destruction or a lessened production of the red blood-corpuscles, or both.

An anæmia as intense as pernicious anæmia may develop in connection with certain structural diseases, such as malignant tumors, chronic Bright's disease, phthisis, leucocythæmia, pseudo-leucocythæmia, syphilis, chronic malaria, chronic diarrhœa, and the presence in the intestine of the parasite *Anchylostomum duodenale*. The anæmia in these cases is symptomatic, whereas it belongs to the conception of pernicious or idiopathic anæmia that no adequate cause can be found to explain the anæmia.

It may happen that the exclusion of some of the structural conditions named is not easy or even possible during the life of the patient; and upon post-mortem examination they may escape detection or their recognition may depend in an unusual degree upon the knowledge and skill of the pathologist. Hence it is that a certain amount of uncertainty surrounds the diagnosis in many cases of pernicious anæmia. Thus, it has repeatedly happened that a case supposed during life to be an instance of pernicious anæmia has proven upon post-mortem examination to be one of latent cancer of the stomach. A whole group of cases once supposed to belong to pernicious anæmia, and described as tropical chlorosis and as the anæmia of workers in tunnels, has been found to depend upon the presence of the *Anchylostomum duodenale*. A certain number of cases with all of the symptoms of pernicious anæmia has been proven to depend upon degeneration of the gastric tubules, thus establishing a mode of causation of profound anæmia which was suggested by the author as long ago as 1860.[1] It is probable that certain other obscure forms of profound anæmia, which now receive a provisional shelter under the name of pernicious anæmia, will be relegated by future investigations to other categories.

The most important of the ANATOMICAL CHANGES in pernicious anæmia relate to the blood. It is probable that in many cases there is a diminution in the quantity of blood out of proportion to the reduction in the weight of the body. Such a true oligæmia was proven by Quincke to exist in two of his cases. A diminution in the mass of blood, however, is usually more a matter of inference than susceptible of actual proof. An essential change in this disease is the loss of red blood-corpuscles, or oligocythæmia, which may be accurately estimated by means of the hæmacytometer. The blood appears abnormally pale. In pernicious anæmia the number of red corpuscles may be reduced to one-third or even one-tenth of the normal amount. Instead of 5,000,000 red corpuscles per cubic millimetre, it is common to find no more than 1,500,000. The number is exceptionally reduced to 500,000 or even lower.

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[1] Austin Flint, *American Medical Times*, 1860.

The quantity of hæmoglobin is not always reduced in proportion to the diminution of red blood-corpuscles, so that the actual degree of anæmia may be less intense than is indicated by simply counting the corpuscles. It is considered by Laache as somewhat characteristic of pernicious anæmia, in contrast with chlorosis, that the percentage of hæmoglobin in the red blood-corpuscles is greater than normal. There may, however, be extreme reduction in the quantity of hæmoglobin, as in a fatal case reported by Quinquaud, in which the percentage of hæmoglobin was 2.6 (instead of the normal 12 to 14). More commonly than in any other form of anæmia, the red blood-corpuscles present abnormalities in size and in shape. There may be large corpuscles, 12 to 14 μ in diameter, as well as small corpuscles less than 6 μ in diameter. There are frequently diversities in form described under the name "poikilocytosis" (p. 60), such as pear-shaped, ring-shaped, cup-shaped, kidney-shaped corpuscles, corpuscles with tail-like processes, etc. There may be alterations in the composition of the red blood-corpuscles revealed only by certain staining reagents. Nucleated red blood-corpuscles are sometimes—according to Ehrlich usually—present. Blood-plates are generally fewer than normal. The white blood-corpuscles are usually not increased in number, but there have been a few curious cases observed in which a sudden invasion of the blood with large numbers of leucocytes occurred, so that the pernicious anæmia became an actual leucocythæmia.

The specific gravity of the blood is reduced in consequence of the diminution in solid constituents. In one case Quincke estimated the specific gravity at 1028, instead of the normal 1055.

The body is, as a rule, not emaciated, and there may be decided obesity. The surface of the body and the various organs and tissues are extremely pale. Small extravasations of blood are usually found in different parts. The most frequent seats of ecchymoses are the retina, the inner surface of the dura mater, and the serous membranes. The hemorrhages in the retina are usually small. They are most frequent near the papilla and along the vessels. The ecchymoses in the retina often present a whitish or grayish centre. More or less general œdema, with dropsical accumulations in the serous cavities, is common. There may be a slightly jaundiced tint of this fluid as well as of the sclerotic coat and of the integument.

Almost without exception in the fatal cases of pernicious anæmia there is more or less fatty degeneration of the heart. Often this degeneration is extreme and extends throughout the whole thickness of the heart. The heart-walls are usually flabby. Fatty degeneration of the hepatic and of the renal epithelium has also been observed.

The spleen may be unchanged or it may be moderately swollen. The lymph-glands, which are usually normal in appearance, may be somewhat swollen and of a deep-red color, containing an unusual number of red blood-corpuscles, among which may be nucleated red corpuscles.

An interesting lesion of pernicious anæmia is a large accumulation of iron or some ferric compound in certain organs of the body, especially in the liver, the spleen, the kidneys, and the marrow of the bones. If microscopic sections of these organs be treated with sulphide of ammonium, a great quantity of greenish-black granules of sulphide of iron can be seen in the cells. Thus, the hepatic cells may appear thickly studded with such granules. This accumulation of iron points to an enormous destruction of red blood-corpuscles, for the iron is doubtless derived from this source.

In the great majority of cases of pernicious anæmia, although not invariably, the fatty marrow which is normally present in the long bones of adults is changed into red or lymphoid marrow. Microscopic examination of the altered marrow shows that the fat-cells have in great part disappeared, and have been replaced by large and small, coarsely and finely granular, medullary cells. In addition, as first pointed out by Cohnheim, there are often nucleated red corpuscles in considerable number, which would seem to indicate an increase in the blood-forming function of the marrow. Cells containing red corpuscles or their fragments are often found in large number both in the marrow and in the spleen. In a few cases there has been observed a change in the marrow resembling the pyoid alteration of leucocythæmia. Also in a few cases lymphomatous growths have been found in the marrow of the bones, in the periosteum, and in the kidney.

These changes in the marrow have led to the idea that at least some cases of pernicious anæmia may be regarded as the myelogenic form of pseudo-leucocythæmia. This view is probable, but an objection to it is that similar changes, although generally less intense, are often present in severe symptomatic anæmia.

The anatomical changes found after death from pernicious anæmia are not peculiar to this disease, but may be present in profound anæmia from any cause. Nor do these changes explain the pathogeny of the anæmia. As already mentioned, the high percentage of iron found in different organs is evidence of abnormally great destruction of red blood-corpuscles, although nothing is known as to the cause of this destruction.

The SYMPTOMS of pernicious anæmia, in so far as they represent a profound and persistent anæmic condition, are essentially those belonging to the clinical history of the grave hæmatopoietic diseases already considered—namely, leucocythæmia and pseudo-leucocythæmia. Anorexia, nausea, and vomiting are symptoms more constant and prominent than in the diseases just named. More than twenty-five years ago, I ventured to predict that the disease would be found to involve degenerative changes in the gastro-intestinal tubules. In a certain number of cases this prediction has been found to be correct. (See p. 439.) In respect of the symptomatology it will suffice to state the points embraced in the diagnosis.

The liability to the error of mistaking for pernicious anæmia that which is symptomatic of some obscure, grave local affection, especially carcinoma, has been already mentioned. This error cannot always be avoided. Investigations, however, should always be directed to the discovery of some local affection adequate to explain the anæmia. A positive diagnosis requires the exclusion of any such affection. A diagnostic point bearing on this inquiry relates to the nutrition of the patient. Addison called attention to the fact that in the cases of idiopathic fatal anæmia which he described emaciation was not marked, considerable embonpoint being often maintained. As a rule, it is otherwise when anæmia is symptomatic of a grave affection like carcinoma. In some cases of pernicious anæmia, however, there is extreme emaciation.

Leucocythæmia is excluded by the absence of the characteristic blood-change expressed by the name. The white blood-corpuscles are not in sufficient excess to denote that disease. Pseudo-leucocythæmia is excluded by the absence of the affection of the lymphatic glands which characterizes the disease generally understood by that name, more commonly known as Hodgkin's disease.

The question arises not infrequently in cases of simple anæmia and chlorosis, in which the anæmic condition is unusually intense and rebellious, whether the anæmia be not pernicious. As bearing on this question the following points are to be considered: An adequate discoverable causation renders it probable that the anæmia is benign. Yet an anæmia traceable to causes which seem to be adequate may prove to be pernicious. Intractability of the disease after the removal of supposed causes and the faithful employment of appropriate measures of treatment is proof of a pernicious character. Certain events belonging to the clinical history of pernicious anæmia rarely occur in benign anæmia and chlorosis, however protracted. These events are hemorrhages in different situations, general dropsy, and pyrexia. Pyrexial paroxysms of greater or less intensity, of variable duration, and irregular in their occurrence are frequent in pernicious anæmia, as they are also in leucocythæmia and pseudo-leucocythæmia. The loudness of the hæmic murmurs—cardiac, arterial, and venous—is somewhat diagnostic of pernicious anæmia. Notable feebleness of the heart's action, pointing to fatty degeneration, has diagnostic significance. Age is to be taken into account; in the majority of cases pernicious anæmia affects persons in the middle period of life, whereas chlorosis in a large proportion of cases affects young subjects. It remains to be determined by clinical observations how much can be inferred from the abnormal appearances of the red corpuscles as regards the diagnosis. Impaired vision dependent on retinal ecchymoses is to be included among the diagnostic points, as well as the existence of ecchymoses without an appreciable impairment of vision. The employment of the ophthalmoscope is thus of importance with reference to the diagnosis. According to Quincke, ecchymotic spots in the retina are so frequent in pernicious anæmia, and so rare in other affections involving anæmia, that they have a significance almost pathognomonic. That author has also called attention to the occurrence in some cases of a systolic pulsation of the superficial cervical veins without venous turgescence. Functional insufficiency of the tricuspid valve is the probable explanation of this sign, which occurs sometimes temporarily in other connections.

The PROGNOSIS is affected materially by the latitude of the signification of the term pernicious in this application of it. If a fatal termination be essential for the disease to be considered as pernicious, of course there is no possibility of recovery. This is an extreme view. It suffices for the application of the term pernicious that the anæmia present an *ensemble* of the characters which distinguish it in the cases which do prove fatal. Cases have been reported presenting the requirements for the diagnosis of pernicious anæmia in which recovery has taken place. Taking the view just stated, Biermer and Quincke report 6 recoveries out of 54 cases, and Pye-Smith has collected 20 cases of recovery.

The DURATION of fatal cases varies from a few weeks to several months. The average duration is less than that of leucocythæmia and pseudo-leucocythæmia. Sudden death sometimes occurs from syncope. Cerebral hemorrhage may be the immediate cause of death. If the disease be developed during pregnancy, abortion or miscarriage takes place, the patient speedily dying. If death be not caused by either cerebral hemorrhage or syncope, the mode of dying is by slow asthenia.[1]

The objects and general principles of TREATMENT are sufficiently obvious. The removal of all causes which may have produced the disease or which render it persisting and progressive is an object of prime importance. The substitution for these causes of all agents and influences which affect favorably the functions concerned in assimilation and nutrition is the next important object. These objects are widely comprehensive, but it is not necessary to go into details. The same principles are involved as in the treatment of the anæmia in all the grave diseases affecting the hæmotopoietic system. Of medicinal remedies, arsenic has been found to be the most valuable, and should always be given a trial. The arsenic should be administered in gradually increasing doses. As an extreme measure, the transfusion of blood has been resorted to, but thus far without results affording encouragement to expect from it more than a temporary advantage. Reasoning *a priori*, more than this cannot be hoped for from this measure, inasmuch as it cannot reach the essential morbid conditions on which the anæmia depends.

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[1] For a full report of three cases, and an analysis of the cases published in America (39), vide article by Dr. J. H. Musser in *Proceedings of the Philadelphia County Medical Society*, 1885.

CHAPTER V.

ADDISON'S DISEASE.

IN the year 1855, Dr. Thomas Addison[1] described an affection characterized by disease of the suprarenal capsules, by a peculiar discoloration of the skin and of certain mucous membranes, and by the development of progressive and fatal asthenia. This disease, in honor of its discoverer, is called usually *Addison's disease.* It has also received the names *bronzed-skin disease*, *melasma suprarenale*, and *suprarenal capsular disease.* Since the masterly description given by Addison little has been added to our knowledge of this disease, which still remains one of the most obscure in the nosology. The most important addition since Addison's memoir is that the change in the suprarenal capsules usually presents certain well-defined anatomical characters common to the great majority of cases. It was thought by Addison that any disease of the suprarenal capsules was capable of producing the characteristic group of symptoms; it is now known that, as a rule, the morbid process in the capsules is a definite one, to which the name fibro-caseous metamorphosis is applicable.

Almost without exception both suprarenal capsules are involved. The change may be farther advanced in one than in the other. The capsules are usually found to be enlarged, of firm consistence, and with nodulated surfaces. The size may equal that of a small hen's egg, but it is usually less. Sometimes the size is normal or even less than normal. The latter condition generally corresponds to a late stage of the process. Upon section, usually no trace of the normal parenchyma or of the distinction between cortex and medulla can be detected. The place of the normal tissue is occupied by opaque, yellow, friable, cheesy masses imbedded in firm, grayish, semi-transparent fibrous tissue. The proportion between these two substances varies; sometimes one predominates and sometimes the other. On close inspection gradual transitions from the grayish fibrous to the yellow cheesy material can often be seen. The caseous substance may become dry and calcified, or it may soften and form collections of thick, creamy fluid resembling abscesses. The whole interior of the organ may be converted into a hard, cretaceous mass, or into a cavity containing creamy fluid. In this case the organ may be much diminished in size. In some cases small grayish granulations resembling miliary tubercles can be seen around the cheesy material and in the fibrous stroma. The microscopical examination of the gray fibrous material shows a fibrillated, sometimes a reticulated, stroma containing fusiform cells, lymphoid cells, and not infrequently giant-cells. The caseous substance consists of a granular detritus of albuminous and some fatty particles with remnants of cells. Little nodules composed of lymphoid cells and giant-cells—in other words, of tuberculous tissue—have been occasionally observed. As to the nature of this change in the suprarenal capsules writers are not agreed. Some consider it as a chronic inflammation resulting in the formation of connective tissue and in caseation of the inflammatory products (Wilks). Others regard the alteration as tuberculous in character. This is the most probable view. In not a few cases undoubted tuberculous changes have been found at the same time in the lungs and in other parts of the body, but there have been many cases in which the alteration was confined to the suprarenal capsules. In some cases tubercle bacilli have been found in the lesions of the suprarenal capsules; in other cases the bacilli have been missed. Further observations upon this point are needed, as well as concerning the inoculability in animals of the caseous substance in the capsules.

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[1] *The Constitutional and Local Effects of Diseases of the Suprarenal Capsules*, London, 1855.

The altered capsules are usually found imbedded in a mass of indurated fibrous tissue which binds them firmly to surrounding structures, such as the liver, pancreas, kidneys, stomach, vena cava, and diaphragm. This overgrowth of connective tissue generally invades the important sympathetic nerves in the neighborhood of the suprarenal capsules—namely, the solar and suprarenal plexuses and the semilunar ganglia. Changes have been found, although not constantly, in these nervous structures. These consist in hyperæmia, swelling in consequence of thickening of the nerve-sheaths, fatty degeneration and atrophy of the nerve-fibres and ganglion-cells, and increased pigmentation of the ganglion-cells. Alterations have been described also in the nerve-plexuses in the stomach and in the intestine.

The mesenteric glands, especially those in the neighborhood of the suprarenal capsules, are often enlarged and sometimes cheesy. It is not unusual to find enlargement of the solitary and agminated follicles of the intestine. In some cases the spleen has been found enlarged and softened. Tuberculous deposits in various parts of the body are frequent. Indurated and caseous nodules in the apices of the lungs seem to be the rule. General pulmonary tuberculosis is less frequent. Caseous nodules have been found in the testicles, brain (solitary tubercles), and lymphatic glands. The carious condition of the vertebræ, which has been found several times as a complication with and without lumbar abscesses, probably belongs to the category of tuberculous changes. Acute miliary tuberculosis may result from the invasion of the suprarenal veins by the tuberculous material.

The discoloration of the skin and of certain mucous membranes is due to the deposit of brown or yellowish-brown pigment-granules in the lowest layers of the epithelium. In the skin it is found chiefly in the epithelial cells composing the two or three lowest layers of the rete Malpighii, and is most abundant in the layer next to the corium. Similar pigment-granules are found in the connective-tissue cells beneath the epithelium and about the blood-vessels. The deposition of pigment is in the same anatomical elements as in the negro.

In some, but not the majority, of cases fatty degeneration of the heart has been noted, and also parenchymatous degeneration of the heart, liver, and kidneys. More or less anæmia is usually present, but the anæmia is rarely intense, and it may be absent. Anæmia is not an essential element of the disease.

Before completing the description of the anatomical characters of Addison's disease it is necessary to add that changes in the suprarenal capsules, other than the fibro-caseous metamorphosis, have been reported as associated with bronzing of the skin and the other symptoms of Addison's disease. Most of these changes have been due either to atrophy or to primary or secondary cancer of the capsules. Primary cancer of the suprarenal capsules is very rare, and some of the cases so reported seem to have been in reality fibro-caseous metamorphosis. Secondary cancer is more frequent. In the larger number of cases of carcinoma of the suprarenal capsules no pigmentation of the skin was observed. Greenhow, who has analyzed those cases in which discoloration of the skin was present, comes to the conclusion that the pigmentation did not present the characters typical of Addison's disease. Very positive statements, however, have been made as to the indentity of the pigmentation with that in Addison's disease. There are observations which strongly support the view that a variety of morbid processes in the suprarenal capsules may produce the symptoms of Addison's disease. On the other hand, apparently typical cases of Addison's disease, so far as the pigmentation and other symptoms are concerned, have been reported without any disease of the suprarenal capsules. In a large number of the cases reported as bronzed-skin disease without change in the suprarenal capsules the pigmentation did not present the typical characters, or it was due to some of the many physiological or pathological causes of discoloration of the skin which have nothing in common with Addison's disease. There remains, however, a small number of cases in which the pigmentation and the constitutional symptoms were the same as those in Addison's disease, but in which the suprarenal capsules were found to be normal.

There has been no explanation at all satisfactory of the relation between the three conditions which characterize Addison's disease—namely, the change in the suprarenal capsules, the pigmentation of the skin, and the constitutional affection. It is not within the scope of this work to enter into a discussion of the different theories which have been proposed. A brief mention of the leading views will suffice to bring out the points at issue. According to one theory, disease of the suprarenal capsules is primary and is the cause of the symptoms. In opposition to this theory it is argued that the symptoms cannot be referred to an abolition of the functions of the suprarenal capsules, whatever those functions may be; for the capsules have been extirpated in animals, and have been destroyed in man by cancer and other processes, without bronzing of the skin or other symptoms of Addison's disease. Nothnagel has succeeded in producing in animals caseous inflammation of the suprarenal capsules without the sequence of Addison's disease. Physiological experiments have shown that these organs are not necessary to life. The theory that the symptoms of Addison's disease are due to irritation of supposed secreting cells in the glands, by which their function is increased and deleterious substances are introduced into the blood, was advanced by Klebs, but is not based on established physiological facts, and has not met with favor. Appreciating the difficulty of referring the symptoms to interference with the function of the suprarenal capsules, many, even of those who regard the disease of the suprarenal capsules as primary, attribute the symptoms to involvement of the adjacent sympathetic nerves and ganglia. This theory is advocated by Virchow, Schmidt, Greenhow, Jaccoud, Resel, and many others. The objections raised against this theory are that the alterations in these nervous structures are not constantly present, that similar changes are found in a variety of processes without any of the symptoms of Addison's disease,[1] and that the affection of the sympathetic does not make any clearer the explanation of the characteristic symptoms. Rossbach believes that Addison's disease is a general neurosis due to functional disturbance of the entire nervous system. Another theory is that disease of the suprarenal capsules and the characteristic symptoms are independent of any causative relation to each other, and are all the result of some common antecedent condition which Heschl, Buhl, and others seek in the blood. Buhl suggests the entrance of some morbific agent from without into the blood; in other words, he regards Addison's disease as infectious in its origin. Heschl emphasizes especially the tuberculous diathesis. Pepper ranks Addison's disease with pernicious anæmia, leucocythæmia, and pseudo-leucocythæmia among the anæmatoses; that is, diseases in which the primary change is a profound disturbance of the blood-forming functions.

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[1] Bronzing of the skin has been observed in a few cases of aneurism of the abdominal aorta, and of tuberculous or cancerous disease of the retro-peritoneal glands with involvement of the adjacent sympathetic nerves and ganglia.

The following is a description of the appearances of the skin and of the SYMPTOMATOLOGY, by Dr. Edward Headlam Greenhow, who has devoted special study to this disease and has analyzed a larger number of cases than has been collected by any other writer:[1]

"The peculiar change of color consists in a gradual darkening of the skin of various parts of the body or even of the whole body. The aspect of this discoloration gives to the patient the appearance of belonging to one of the dark races of mankind. Most frequently it has a dusky, smoky, or yellowish-brown hue, but sometimes it is of an olive or green-brown color, and when deepest approaches in parts to the tint of negro skin. The discoloration is never uniform over all parts of the body, but commences earlier and becomes deeper on some or all of the exposed parts—the face, neck, dorsum of hands and fingers; and on those parts which are naturally the seat of more pigment than the general surface—the axillæ, abdomen, groins, genital regions, nipples, and especially the areolæ. The deep discoloration of these last may, in my opinion, be regarded as one of the most decisive external signs of Addison's disease, and the appearance of small, well-defined black specks, like black moles or freckles, on already discolored portions of skin, is in my experience another no less certain. Although sometimes universal, the discoloration is more frequently only distinctly obvious on certain parts of the body. Even in the latter cases, however, it probably affects in some degree the whole cutaneous surface, for on microscopical examination of parts of the skin which by contrast with the dark spots appeared normal, I have found deposited also in them, though in comparatively small quantity, the pigment which imparts the abnormal color to the skin. The transitions from the darker to the paler shades of the discoloration of different parts of the body are never abrupt; the darkest parts are never sharply-defined patches, but merge insensibly into the lighter or apparently normal portions of the surface. There is, however, one marked exception to this rule. When the skin has suffered any superficial abrasion or injury, or when the patient has been blistered at some recent period, the injured parts not only become much darker than the surrounding portions of the surface, but they present defined margins coinciding with the extent of the cutaneous injury. The cicatrices of deeper injuries, on the contrary, usually remain pale, and are surrounded by a distinct border of more or less dark pigment, contrasting strongly with the ivory-like hue of the cicatricial tissue. It does not, however, require actual abrasion of the skin to excite a greater deposit of pigment in certain parts; slight local irritation of the surface appears in not a few cases to have produced the same effect. I have myself seen dark streaks on the skin corresponding with the lines of pressure exerted on particular parts by petticoat-strings and garters, and the same circumstance has been noted by others. In a case recorded by Mr. Nicholson in 1872 it is stated that the patient, a baker's lad, presented on his shoulders dark stripes corresponding to the bands by which the basket he carried was slung at his back. The discoloration in Addison's disease is not entirely restricted to the skin, being also frequently found upon the mucous membrane of the lips, cheeks, and gums, and also upon the tongue. On the lips it takes the form of an irregular bluish-black streak, running lengthwise, near the junction of the mucous membrane and outer skin. It is seen on the gums and buccal mucous membrane in the form of irregular stains and patches, mostly of a brownish color, with ill-defined margins. In two of my own cases the stains on the buccal mucous membrane have certainly corresponded with seats of irritation produced by the pressure of ragged, protruding teeth; but in other cases I have been unable to verify any such cause. The discoloration of the tongue appears usually in sharply-defined stains of a purplish-black or sometimes inky hue; and in my cases these stains have always been situated near the margins of the organ. On the other hand, the conjunctivæ have always remained normal, and the contrast between their pearly whiteness and the dusky hue of the discolored skin is very striking, and involuntarily recalls the similar contrast in the mulatto or negro countenance.

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[1] Vide "Croonian Lectures on Addison's Disease," *The Lancet*, 1875; also, a monograph published in 1866.

"We see, therefore, that the discoloration of the skin in Addison's disease usually begins, and eventually becomes deepest, on those parts which are naturally the most liable to become pigmented either by exposure to sun and air or by the excitement of certain physiological processes; and to these must be added such parts as may accidentally be subjected to the excitement of local irritation. In other words, this peculiar change of color is, like many other pathological processes, merely an exaggerated, and therefore a morbid, development of a natural physiological process."

Greenhow describes the constitutional symptoms as follows: "Gradual progressive asthenia, often originating without any apparent cause and seldom dating from any definite period; great languor and indisposition for exertion, with, in advanced cases, breathlessness and palpitation, frequent sighing or yawning, and generally faintness on making any muscular effort, sometimes even on being raised up in bed. There are almost invariably great weakness of the heart's action and remarkable feebleness of pulse; loss of appetite; irritability of stomach, with nausea; and toward the close of the illness at least occasional, and often persistent, vomiting. The mind is generally clear to the last, but so great is the prostration in the latest stages of the disease that the patient often lies in a drowsy, apparently semi-comatose state, from which, however, he can be roused by questions, and to these he generally gives pertinent, though slow and reluctant, answers. There are in many cases pains in the loins, hypochondria, or epigastrium, and, more rarely, dimness of sight, vertigo, and, near death, a tendency to incoherence or delirium. Death takes place from asthenia, and often rather suddenly." . . . . "There is, in uncomplicated cases, comparatively little or no emaciation. The skin is soft and cool. The tongue is usually clean and moist until the last day of life."

The occasional occurrence of convulsions is mentioned by Greenhow, and also by Jaccoud. The convulsions are oftener partial than general. Pye-Smith and others have observed cases which terminated with severe cerebral symptoms—namely, delirium, coma, etc. In a case reported by Nothnagel the coma was associated with acetonuria (p. 72). Attacks of syncope are not rare, and may end fatally.

All writers dwell upon the frequency and prominence of gastric symptoms—namely, anorexia, nausea with more or less vomiting, eructations, cardialgia, etc.[1] In the early stage of the disease the bowels are generally constipated, but at a later period diarrhœa is not infrequent.

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[1] Merkel, author of the article on this disease in *Ziemssen's Cyclopædia*, in a foot-note mentions, "as a curiosity," that in the *Phil. Med. and Surg. Reporter*, June, 1871, an

As regards the urine, Thudicum found diminution of uric acid and of coloring matter. Rosenstern ascertained that the excretion of urea is diminished, and that there is an increase of indican.

The course of this disease is progressive, but its progress is not always continuous. Greenhow's observations show, in certain cases, paroxysms of progress, exacerbations, and remissions. During a remission the discoloration of the skin is lessened, and there may be more or less general improvement. Exceptionally, the disease runs a rapid course. In these cases bronzing of the skin usually precedes for some time the constitutional symptoms. In the vast majority of cases the progress is slow, the duration, however, being indefinite, extending sometimes over several months and sometimes over many years, the average duration being a year, or, according to Wilks, a year and a half.

The DIAGNOSIS is to be based on the characteristic bronzing of the skin together with the associated symptoms. A discoloration of the skin bearing more or less resemblance to that of Addison's disease may be referable to pityriasis, chloasma, protracted jaundice, cancerous and tuberculous disease, particularly when seated in the abdomen, the internal use of the nitrate of silver, the permanent staining which follows some syphilitic eruptions, and that caused by exposure, dirt, and the irritation of vermin, the latter described by Voght and by Greenhow under the name "vagabonds' disease." The associated symptoms representing the constitutional affection corroborate the diagnosis as based on the appearances of the skin. It is to be borne in mind that the bronzing of the skin may precede as well as follow the constitutional symptoms. Some delay under these circumstances may be requisite for a positive diagnosis. On the other hand, there is no doubt as to the occasional occurrence of cases of Addison's disease, as proved by the constitutional symptoms and by the characteristic changes in the suprarenal capsules, without any pigmentation of the skin. In these cases generally the duration of the disease has been brief or it has been associated with grave complications. Brown patches in the mouth have been observed without bronzing of the skin. It is not very comprehensible that, associated with pyrexia, the disease should be mistaken for typhoid fever. The author of the article in *Ziemssen's Cyclopædia*, however, states that this mistake was made in a case under his observation. Litten, and more recently Krocker, describe pigmentation of the skin as a sequence of typhoid fever.

Addison's disease resembles the other grave diseases of the hæmatopoietic system in the obscurity of its CAUSATION. It occurs after adult age, in early or middle life, but very rarely in childhood and in advanced age. In a large majority of cases patients are of the male sex, and a majority of these belong to the so-called working class. The character of the anatomical changes, and the not infrequent association with phthisis, justify the conclusion that a tuberculous diathesis may favor the development of the disease.

Writers agree that the PROGNOSIS in the present state of knowledge is fatal, but the duration of the disease sometimes extends over many years. It may remain non-progressive for an indefinite period, and in some cases notable improvement takes place. These are important facts to be considered with reference to the prognosis and as affording encouragement in the treatment.

In the absence of any remedies known to have a special curative efficacy,

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attempt is made to "refer the entire complex set of symptoms to degeneration of the glands of the stomach." If the attempt to connect the disease with glandular organs so vast and important as those which secrete the gastric juice be worthy of being mentioned only as a curiosity, how absurd would it have seemed to the author of that article to refer the disease to organs comparatively so insignificant as the suprarenal capsules had he written his article prior to Addison's researches!

symptomatic indications are to be fulfilled, and TREATMENT, medicinal and hygienic, is to be pursued with reference to the same objects as in treating the other diseases of the hæmatopoietic system. In a case which came under my observation the treatment by general faradization and galvanization of the sympathetic nerve in the hands of Dr. A. D. Rockwell was accompanied by marked improvement as regards the muscular strength and endurance; the sexual desire and capability, which had been lost, returned, and the bronzing became perceptibly less. This improvement continued for two years, when the patient suddenly, without any apparent cause, failed, and died within twenty-four hours.[1]

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[1] Vide *Med. and Surg. Electricity*, by Drs. Beard and Rockwell, 2d ed., p. 675. For exhaustive articles in relation to this disease the reader is referred, in addition to the works by Greenhow, to which reference has been already made, to Jaccoud, article "Bronzée maladie," in *Nouveau Dict. de Méd. et de Chirurg. pratique*, tome xi., 1866; and to *Traité de pathologie interne*, tome ii., cinquième edition, 1877; to Averbeck, *Die Addison'sche Krankheit.*, Erlangen, 1869; to article by Pepper, in *Am. Journ. of Med. Sciences*, April, 1877; and by Osler, in *System of Medicine by American Authors*, vol. iii.

SECTION FOURTH.

DISEASES AFFECTING THE DIGESTIVE OR CHYLOPOIETIC SYSTEM.

CHAPTER I.

DISEASES OF THE TONSILS, THE PHARYNX, THE ŒSOPHAGUS, AND THE PAROTID GLANDS.

Introductory Remarks.—Tonsillitis.—Pharyngitis.—Diseases of the Œsophagus.—Parotiditis.—Mumps.

THE important organs composing the digestive system are the mouth, with the parotid, submaxillary, and sublingual glands, the fauces, the pharynx and œsophagus, the stomach and duodenum, the small and the large intestine, with the mesenteric glands, and the solid abdominal viscera—namely, the liver, pancreas, and spleen. Affections of the two outlets of the alimentary canal—namely, the mouth, fauces, pharynx, and œsophagus, with the appended glands, and the rectum and anus—properly belong to the department of surgery, being either open to view or accessible by manual exploration, and often requiring surgical operations. These affections, for the most part, will not be considered in this work. This section will therefore be devoted mainly to the diseases affecting the hollow viscera and the solid organs within the abdomen. The affections of the digestive system, as of other anatomical systems, consist of—*first*, inflammation; *second*, structural lesions; and *third*, functional disorders. The diseases of the different divisions of the alimentary canal will be considered in their natural order, beginning with the affections of the upper part of this canal. The diseases of the mouth and of the tongue, such as the different varieties of stomatitis or of glossitis, belong to surgery. Certain affections of the mouth, prevailing especially in early life, are considered in treatises devoted to diseases of children.

Tonsillitis.

The most important varieties of tonsillitis are acute simple tonsillitis (often designated as catarrhal), follicular tonsillitis, and parenchymatous or suppurative tonsillitis, commonly known as quinsy. When the inflammation involves especially the lacunæ or crypts of the tonsils, the disease is called lacunar or follicular tonsillitis. Pseudo-membranous tonsillitis is usually present in true croup and in diphtheria, and is considered in connection with these diseases.

Acute Simple Tonsillitis is characterized, anatomically, by redness with moderate swelling of the tonsils and an inflammatory exudation from the mucous membrane, composed of mucus mingled with epithelial cells, pus-cells, and serum. This simple tonsillitis is present in most cases of acute pharyngitis, or ordinary sore throat. The tonsillitis contributes its share to the constitutional disturbance, pain, and other symptoms which will be described under

Acute Pharyngitis; but the affection of the tonsils is not usually so severe as to require a separate consideration.

Acute Follicular or *Lacunar Tonsillitis* is of considerable clinical importance. In this affection the inflammation involves not only the mucous membrane covering the surface of the gland, but especially that lining the crypts. The tonsils present little white patches composed of epithelial cells, pus-cells, mucus, and a large number of bacteria. These white deposits extend into the lacunar depressions. It is important to distinguish these white patches from a diphtheritic exudation with which they are sometimes confounded. In distinction from the latter the white masses in follicular tonsillitis form a pultaceous deposit, not a membraniform layer like the deposit of fibrin; they cannot be removed in strips like the latter; they are more easily removed, are more prominent, are more abruptly defined, and disappear spontaneously more quickly, than patches of fibrin. On close inspection the secretion will be seen to dip into the follicular depressions. The follicular deposit occurs without any abrasion of the mucous surface, but it is common for patients to consider the patches as ulcers.

Acute follicular tonsillitis is often attended by severe constitutional disturbance. The symptoms at the onset may be as severe as in diphtheria. Chilly sensations, or even a chill, may mark the beginning of the affection. The temperature rapidly rises, and within twenty-four or forty-eight hours may reach 104° or 105°. There are often headache, anorexia, insomnia, and other symptoms which usually attend marked pyrexia. The patient complains of dryness and soreness of the throat, and there may be considerable pain on swallowing. Usually within three to five days the fever and other symptoms subside and recovery is rapid. During, and sometimes for a considerable time after, the attack there may be marked prostration.

The prognosis is always favorable. The severity of the constitutional symptoms in many cases, together with the anatomical appearances, has often led to the diagnosis of diphtheria from which, as already stated, it is important that follicular tonsillitis should be carefully discriminated.

Parenchymatous Tonsillitis, or inflammation of the substance of the tonsil, may terminate in resolution, but usually it goes on to suppuration, constituting the disease called *suppurative tonsillitis* or *quinsy*. The tonsil of one side only or both tonsils may be affected. The tonsil becomes much enlarged and the seat of an abscess. The abscess may open at one or at several points on the surface of the tonsil. The inflammation usually extends to surrounding parts, the soft palate and uvula especially becoming hyperæmic and much swollen. The submaxillary gland is not infrequently enlarged.

Suppurative tonsillitis may attack a previously healthy tonsil, but it is particularly liable to occur in those whose tonsils are affected by chronic inflammation. The causation involves a predisposition to the affection, and persons predisposed are likely to experience repeated attacks. The immediate cause of the suppuration doubtless is to be found in micro-organisms which invade the substance of the tonsil.

Suppurative tonsillitis is a very distressing affection, in consequence of the pain and difficulty in deglutition. There is elevation of temperature, which may be considerable. Increased frequency of pulse, loss of appetite, headache, sleeplessness, pain in the limbs, are other symptoms which mark the febrile disturbance. Pain is a prominent symptom, this being most marked upon attempting to swallow. Sometimes pain is referred to the ear. The throat is at first dry; and subsequently there is a secretion of viscid mucus and of saliva, often in abundance. The voice is husky, and articulation may be difficult or almost impossible. Difficulty of breathing occurs when both tonsils are greatly swollen. A rare complication is œdema of the glottis, which may cause death. The symptoms increase in severity until the abscess bursts or is opened, and then there is a notable and usually rapid abatement of the local and constitutional disturbances. A very exceptional occurrence is suffocation produced by escape of the pus into the larynx when the abscess ruptures during sleep. Fatal hemorrhage has been known to take place from ulceration of the abscess into the internal carotid or internal maxillary artery. These unfortunate terminations, however, are very rare, and the prognosis is to be considered as favorable.

It is not rare for little yellowish-white concretions to form in the crypts of the tonsils. These concretions consist of desiccated epithelial and pus-cells and low vegetable organisms. When crushed, these whitish pellets emit a very offensive odor. These concretions, which may contain lime salts, are sometimes mistaken for tubercles.

In acute simple tonsillitis the measures of *treatment* are those which will be mentioned in connection with Simple Pharyngitis.

In the follicular variety a solution of alum, borax, or the chlorate of potassa may be used as a topical application by means of a camel's-hair pencil brush. Gargles do not bring the solution in contact with the affected parts. In cases with marked pyrexia the tincture of aconite in small, frequently repeated doses should be given, until an impression is made on the pulse.

The treatment of parenchymatous tonsillitis, or quinsy, consists of poultices or the water-dressing to the neck, the inhalation of warm vapor, and anodynes to lessen discomfort. Milk is the form of nourishment best taken. When fluctuation is perceived, the abscesses may be opened and the patient will be relieved sooner than if they had been left to break.

Pharyngitis.

A variety of forms of pharyngitis are distinguished, many of which can receive only passing mention within the limits of this work.

In *Acute Simple Pharyngitis* the mucous membrane is congested, swollen, and coated with more or less of a muco-purulent secretion. The inflammation usually extends to the tonsils and soft palate, and it may also involve the nasal passages. This variety of pharyngitis is one of the forms of "a cold" or ordinary sore throat. There is usually more or less pain, especially in the act of swallowing. There is at first a sensation of dryness. The secretion of mucus at a later stage provokes efforts at hawking and expuition. If the inflammation extend low in the pharynx, coughing is excited. The cough is of a peculiar character, easily recognized and distinguished as a throat-cough, the forcible current of expired air being brought to bear on the pharynx. The limitation of the inflammation to the pharynx or its non-extension into the larynx is shown by the absence of huskiness or hoarseness of the voice. Febrile disturbance, except sometimes in children, is slight. The extent and intensity of the inflammation are to be ascertained by inspection of the throat. The involvement of the vault of the pharynx, the posterior nares, and the posterior surface of the palate and the lower part of the pharynx can be ascertained by the use of the laryngoscope and of the rhinoscope.

Acute simple pharyngitis sometimes occurs as an *epidemic* affection, in which case the disease is to be regarded as a form of fever rather than simply as a local inflammation. I have analyzed a series of twenty-three cases which occurred during such an epidemic in the western part of New York State in 1857.[1] From this analysis the conclusion was reached that the disease was an epidemic fever characterized by mild erythematic inflammation of the pharynx and fauces as the only constant local complication. The febrile movement was in a marked degree out of proportion to the local affection. Its duration was from three to five days. The epidemic prevailed extensively for about two months, both sexes and different ages being affected without notable discrimination. No proof of contagion existed. The affection was not scarlatina, but it resembled influenza, save that the pharyngeal and not the bronchial mucous membrane was the seat of inflammation. It is perhaps proper to regard the epidemic disease as a form of influenza.

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[1] *Buffalo Medical Journal*, vol. xii. p. 718.

Acute pharyngitis is a common complication in many acute infectious diseases, especially in the exanthematous fevers. Reference will be made hereafter to *Exanthematous Pharyngitis* in connection with scarlatina, measles, and smallpox.

The pharyngitis is designated as *erysipelatous* when it complicates erysipelas, particularly facial erysipelas. Usually, the inflammation is of the acute simple variety, but it may be gangrenous or suppurative, and in the latter case the prognosis is grave. It is claimed that erysipelas may attack the pharynx primarily.

Gangrenous Pharyngitis may occur also in connection with diphtheria, and rarely as a complication of scarlet fever, smallpox, typhoid fever, or other acute infectious disease. The symptoms assume a typhoid character and and the termination is usually fatal.

In *Phlegmonous Pharyngitis* there is an accumulation of pus in the submucous and deeper tissues of the pharynx, constituting a *retro-pharyngeal abscess.* This affection may occur traumatically, as by the penetration of a sharp piece of bone. More frequently it is secondary to caries of the cervical vertebræ.

Pseudo-membranous or *Fibrinous Pharyngitis* constitutes a part of the lesions in croup and in diphtheria, with which diseases it is considered.

Subacute Simple Pharyngitis is a very common affection, and is generally treated without the advice of a physician. The lesions and symptoms are similar to those in acute simple pharyngitis, but they are less intense.

Chronic Pharyngitis is an extremely common affection in this country. It is commonly known as "the catarrh." It may follow repeated attacks of acute pharyngitis, but it is generally a subacute affection at the beginning, and is developed imperceptibly. It is customary to distinguish two forms of chronic pharyngitis—namely, simple chronic pharyngitis, and chronic follicular or granular pharyngitis. In *simple chronic pharyngitis* the mucous membrane is inflamed without especial involvement of the lymphatic follicles of the pharynx. The membrane is more or less reddened and thickened. Frequently small, dilated veins can be seen mapping out irregularly the surface of the pharynx. Viscid mucus adheres to the surface of the inflamed mucous membrane. In *follicular* or *granular pharyngitis* the follicles of the pharynx are enlarged in consequence of hyperplasia of their lymphoid cells. These enlarged follicles form little nodules studding the surface of the pharynx. They are surrounded by congested and inflamed mucous membrane. In both forms of pharyngitis—between which, in fact, no sharp line of distinction can be drawn—the inflammation may extend to adjacent mucous membranes. When the inflammation extends to the Eustachian tubes or their openings, deafness is produced in greater or less degree. When chronic pharyngitis has existed for a long time, portions of the mucous membrane are likely to become atrophied.

Chronic pharyngitis in some persons occasions but little inconvenience. It is often found when the patient makes no complaint of any trouble in the throat. In other cases it occasions more or less annoyance. An uncomfortable sensation is felt in the throat. The presence of adhesive mucus excites efforts of hawking and coughing. The proximity of the inflammation to the larynx may give rise to a dry, hacking cough. The voice becomes hoarse after considerable use in speaking or reading, and the use of the voice is followed by a sense of fatigue in the vocal organs.

The affection is much more frequent in men than in women. It occurs most frequently after puberty and in middle life. The form of the disease which is sometimes called *clergyman's sore throat* is usually of the follicular variety. This form of the disease occurs in those who are under the necessity of using the voice a great deal in public speaking, but it is probable that in these cases the excessive use of the voice is not the only cause, for chronic pharyngitis in general occurs most frequently in persons of sedentary habits who suffer from the wear and tear of continued application without physical and mental relaxation. The number of persons is very large in this country who overtask the powers of the system by steady labor in the office, the counting-house, or the workshop, continued without intermission for many years. It is especially among those who in this way violate the laws of health that chronic pharyngitis prevails. It seldom occurs among the so-called laboring-classes, and it is much more frequent in cities than in the country. Occurring in persons of sedentary habits, it is usually accompanied by symptoms denoting impairment of the general health. The patients complain of debility and a want of their accustomed energy; they are generally depressed in spirits; and they often fancy the existence of some serious disease, especially pulmonary consumption. Dyspeptic ailments frequently coexist.

The diagnosis of chronic pharyngitis is readily established by inspection of the throat. The use of the pharyngeal mirror is of service in determining the extent of the lesion.

The prognosis of chronic pharyngitis is favorable so far as life is concerned, but it is not favorable as regards the return of the mucous membrane to its healthy state. The tendency of the disease is to long continuance. There is no tendency of chronic pharyngitis to eventuate in laryngitis or bronchitis, nor is there any tendency to the development of tuberculosis.

The annoyance from chronic pharyngitis is often much enhanced by patients concentrating their attention upon it. They sometimes fall into the habit of constant hemming or "clearing the throat," which is not only fatiguing to themselves, but extremely disagreeable to others.

Tuberculous Pharyngitis is characterized by the growth of tubercles and the formation of tuberculous ulcers in the pharnygeal mucous membrane. Although simple pharnygitis is not uncommon in chronic pulmonary tuberculosis, a genuine tuberculous pharyngitis is infrequent. Sometimes tubercles can be seen during life in the floor or sides or neighborhood of the ulcers; but at other times there is nothing distinctive in the gross appearance of the ulcers, although microscopically their tuberculous nature is evident. Tuberculous ulcers form most frequently at the sides of the larynx and on the palatine folds; and from these situations they may extend to the soft palate and to the posterior wall of the pharynx. Portions of the mucous membrane, particularly that of the uvula, often become irregularly thickened and of a grayish color. In tuberculous pharyngitis there is usually marked pain upon deglutition. The lymphatic glands of the neck are usually swollen.

The diagnosis is to be based upon the recognition of tuberculous disease elsewhere, particularly in the lungs, and by the detection of tubercle bacilli in the secretion from the ulcers.

Tuberculous pharyngitis sometimes runs an acute course, and at other times it is chronic.

Syphilitic Pharyngitis may occur during any stage of syphilis, either congenital or acquired. The manifestations of syphilis begin more frequently upon the tonsils and palate than upon the pharynx. Initial syphilitic sores have been seen upon the tonsils, the palate, the pharynx, and the epiglottis. An erythematic inflammation of the tonsils and palate, extending to the pharynx, is among the earliest and most frequent manifestations of secondary syphilis. The inflamed regions are usually symmetrically situated. Mucous patches are common. These are due to thickening and opacity of patches of epithelium, together with infiltration of the subjacent tissue with lymphoid cells. A secretion, sometimes purulent, takes place from these patches. The mucous patches are usually of short duration, especially under appropriate treatment. Syphilitic ulcers may form in the secondary, but they are most common in the tertiary, stage of syphilis. Tertiary ulcers may be due to the softening and destruction of gummata, or they may result from suppuration. Syphilitic ulcers occur most frequently on the soft palate and palatine folds, but they may extend to the pharynx or may involve the latter primarily. These ulcers are irregular in shape, with undermined edges and surrounded by a zone of congestion. A sequel of syphilitic ulceration is the formation of cicatrices, the recognition of which may aid in the diagnosis of pre-existing syphilis. In rare instances these cicatrices by their extent and contraction occasion serious distortions and stenoses of the pharynx.

The symptoms of syphilitic pharyngitis do not differ materially from those of simple and ulcerative pharyngitis due to other causes, except that pain is rarely a marked symptom, and frequently there is little or no increased sensibility of the throat.

The diagnosis is made by means of a syphilitic history, by the appearances of the inflammation and ulceration already described, and especially by the readiness with which the lesions usually respond to antisyphilitic treatment.

The prognosis is good. Destructive ulcerations with extensive involvement of the bones are much less frequent now than in former times.

Thrush, a disease belonging more appropriately to the affections of the mouth, will receive only brief consideration here. Thrush is characterized anatomically by the appearance of white or brownish-white, curdy patches upon the affected mucous membrane. These patches are composed essentially of the mycelium and spores of a fungus which is usually called *Oidium albicans*, but the exact systematic classification of the fungus has not been determined. The mucous membrane beneath these parasitic patches is usually congested, but not ulcerated.

Thrush is observed most frequently in the mouth and on the palate, but it may involve the pharynx, and it is not infrequent in the œsophagus. It has even been seen in the stomach.

Thrush is a symptomatic affection. It occurs most commonly in nursing infants, especially in those fed by bottle in hospitals and asylums where sufficient attention is not given to cleanliness of the mouth and of the nursing-bottle. Thrush, may, however, occur in adults, and is then observed in conditions of great prostration of the vital forces, as in old people, and toward the termination of such exhausting diseases as carcinoma, tuberculosis, diabetes, dysentery, and typhoid fever.

As regards the TREATMENT of thrush, the borate of sodium is an efficient remedy. This remedy is best applied dissolved in glycerin. Applications by means of a camel's-hair pencil brush may be made three or four times daily. In addition to these applications the indications for treatment are to be derived from the pathological conditions with which this local affection is associated.

Acute simple pharyngitis, occurring as a sporadic disease or as the anatomical characteristic of an epidemic fever, is usually a mild affection, having no tendency to end fatally. Œdema of the glottis is an accident which may occur, but it is extremely infrequent. I have met with one example of its occurrence in a case of sporadic pharyngitis. The therapeutical measures indicated are mild purgatives, followed by anodyne remedies, quinine, and the chlorate of potassa. Gargles of the chlorate or the nitrate of potassa are usually prescribed, but the liquid can in this way be brought into contact with the inflamed membrane to only a limited extent. A mucilaginous liquid or glycerin taken in small quantity and swallowed will allay the dryness and irritation of the throat. Small pieces of gum allowed to dissolve in the mouth have the same effect. The chlorate of potassa, taken in the form of troches, is more efficient than in solution and used as a gargle. Caustic or stimulating applications to the inflamed membrane are not called for. A sinapism externally, liniments containing chloroform, aconite, and laudanum, and the water-dressing give relief.

The treatment of chronic pharyngitis, to be effective, must have reference to the system. Topical applications alone are rarely efficacious. A fair trial of them, however, should be made. The nitrate of silver in strong solution (20 or 30 grs. to the ounce) in some cases, especially those of the granular variety, is highly useful. It is to be applied by means of a probang, mop, or large camel's-hair pencil brush. A solution of tannin is often of service. The chloride, iodide, or sulphite of zinc and iodoform are recommended. Inhalations are not appropriate; they carry the medicated application too far. On the other hand, gargles are of little use; they do not carry the application far enough. Projecting a spray of medicated solutions into the throat is an efficient mode. Applications to the posterior nares may be made by means of the nasal douche; if made by injecting from the fauces, there is danger of the fluid passing into the Eustachian tubes and causing otitis media. The constitutional remedies which exert a curative influence in certain cases are the iodide of potassium, the bromides, the chlorate of potassa, and the hydrochlorate of ammonia. Alteration of the habits of life is first in importance. Relaxation, recreation, and out-of-door life are far more efficacious than medicines, and the latter are of little use without the former. When circumstances permit, relinquishment of business for a time for travel or rural occupations is of signal benefit. Tonic remedies may often be advantageously conjoined with proper hygienic management. Tonics, to be efficacious, should be continued for a long period, and the form of tonic changed from time to time. The diet should be nutritious. The object of treatment, in short, is to restore the general health.

For syphilitic pharyngitis local and general antisyphilitic measures of treatment are indicated. (For these the reader is referred to works which treat of syphilis.)

Diseases of the Œsophagus.

Inflammation of the œsophagus (œsophagitis), acute, subacute, or chronic, is extremely rare. As an idiopathic, non-traumatic affection it may be said almost never to occur. It may be produced by the ingestion of corrosive poisons, by the irritation of foreign bodies, and by an extension of inflammation from adjacent parts. In some parts of this country, especially in the Southern States, inflammation and stricture are frequently caused by drinking accidentally a caustic preparation used for washing and scouring purposes known as "concentrated lye."[1] In cases of diphtheria the diphtheritic affection sometimes extends into the œsophagus, and ribbon-like pseudo-membranes may be expelled. The eruption of smallpox occasionally occurs in this situation.

The diagnostic symptoms of inflammation are pain and soreness localized in this situation, and especially manifest in acts of deglutition. The passage of an œsophageal sound occasions pain, and on its withdrawal mucus or pus may be adherent to it. The latter are evidence of œsophagitis only when carcinoma of the cardiac orifice of the stomach is excluded by the absence of obstruction.

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[1] Vide article by Henry F. Campbell, M. D., in *Trans. American Surgical Society*, vol. i., 1883.

The rational indications for treatment are the nourishment of the patient by a bland liquid diet, of which milk should be the basis, and, if this occasion pain, rectal alimentation may be substituted therefor.

Stenosis, caused by morbid growths, cicatrized ulcers, etc., is ascertained by exploration with the sound, in conjunction with the evidence of obstruction to the ingestion of food. Primary carcinoma occurs in the œsophagus. The consideration of these affections belongs to surgery. In this category belong foreign bodies, the presence of vegetable and animal parasites, dilatations, and diverticula. Rupture of the œsophagus is an occasional accident.[1]

Obstruction may depend on compression by an aneurism or a tumor of some kind. The obstructing cause in these cases is generally apparent. The fact of obstruction in a person over forty years of age should always suggest exploration for the signs of an aneurismal tumor. Rupture of aneurisms sometimes takes place into the œsophagus. Ulcers having the characteristics of gastric ulcer may occur in the lower end of the œsophagus.[2]

Perforation from the presence of a foreign body may establish a communication with the pericardial or the pleural cavity, and it may cause subcutaneous emphysema of the neck and the escape of swallowed fluids into the areolar tissue. Cases have been reported of softening and perforation of the lower portion of the œsophagus by the gastric juice.

Paralysis of the muscular coat, in connection with that of the pharyngeal muscles, may occur after diphtheria, in connection with bulbar paralysis, and in some cases of general paralysis and of cerebro-spinal multiple sclerosis.

Spasm of the œsophagus (œsophagismus) of transient duration is one of the hysterical manifestations. Irrespective of that connection it may cause obstruction for hours and days. In a case reported by Dr. J. J. Henna, which I saw in consultation, complete obstruction from spasm had existed for eight days, and was instantly and permanently removed by a single introduction of a sound.[3]

(For a full account of the diseases of the œsophagus the reader is referred to Cohen's treatise on the *Diseases of the Throat*, and to his articles in Pepper's *System of Medicine, by American Authors*, vol. ii.)

Parotiditis—Mumps.

The affection commonly known as mumps may be appropriately noticed in this connection as seated in an organ accessory to the buccal cavity. The disease is infectious and contagious. It therefore involves a special cause which, reasoning from analogy, is a parasitic organism. The existence of this *contagium vivum* has not as yet been demonstrated. Mumps often prevail as an epidemic. The most marked local affection in this disease is inflammatory swelling of the parotid gland and surrounding connective tissue. As the disease rarely terminates fatally, little is positively known of the anatomical changes in the gland. The interstitial tissue of the gland is believed to be infiltrated with serum and to contain a few emigrant white blood-corpuscles. The epithelium of the acini and ducts of the gland may be supposed to swell up and undergo parenchymatous degeneration. An inflammatory œdema of the connective tissue about the gland exists in most cases. More serious structural changes than those described are not supposed to exist, as the restitution of the gland and of the surrounding tissues to their normal condition occurs with such ease and rapidity. Virchow teaches that the inflammation has its point of departure in the ducts, and is propagated from simple inflammation of the buccal mucous membrane (stomatitis). According to this view, the infectious agent gains access to the gland from the mouth by Steno's duct. A similar coincident inflammation of the submaxillary and sublingual glands is not infrequent. Cases have been described in which these glands were involved and the parotid escaped. Erythematous inflammation of the mouth and pharynx is frequently present in cases of epidemic parotiditis.

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[1] Vide case reported by Prof. Fitz, *Am. Journ. of Med. Sciences*, Jan., 1877.

[2] Vide Quincke, "Ulcus Œsophagi ex Digestione," *Deutsches Archiv für klinische Medicin*, 1879, Bd. 24, S. 72.

[3] Vide *New York Hospital Gazette*, Oct. 18, 1879.

The period of incubation varies from ten to eighteen days. The disease is frequently ushered in by a slight chill or by chilly sensations, and in its progress it may be accompanied with moderate febrile movement, diminished appetite, pain in the head, and general malaise. A certain degree of febrile disturbance usually precedes for a short time the enlargement of the parotid gland, and generally subsides before the swelling fully disappears. The swelling usually begins in one gland, but with few exceptions both glands become involved, although the one first affected suffers more. The amount of swelling in the site of the parotid gland varies in different cases. If considerable or great, it causes a notable change in the physiognomy. The skin generally is not discolored, but there may be an inflammatory blush. The mucous membranes of the mouth, pharynx, and conjunctiva are often moderately inflamed. Pain upon moving the jaws and difficulty in swallowing are experienced. The disease continues for from four or five days to two weeks. The swelling, pain, and soreness gradually subside and disappear. The inflammation has no tendency to suppuration or to continue in a chronic form. An abundant perspiration sometimes takes place at the time of convalescence.

In males, after the age of puberty, one, rarely both, of the testicles may become inflamed. This orchitis is usually called metastatic. When it appears the swelling in the parotid sometimes subsides. This so-called metastatic orchitis is not very frequent. It seems to be more frequent in some epidemics than in others. Most of the statistics bearing on its frequency have been derived from military practice. The largest compilation from this source has been made by Granier.[1] In 495 cases of parotiditis in soldiers, orchitis occurred in 115; that is, in 24 per cent. of the cases. Atrophy of the affected testicle often follows this inflammation. According to Granier, atrophy occurs in 44 per cent. of the cases of orchitis, and in 10½ per cent. of the cases of parotiditis. The shorter the duration of the orchitis, the less probability is there of atrophy of the testicle. In females a similar inflammatory swelling of the mammary gland and of the ovary has been observed as an exceptional occurrence. When parts other than the parotid are involved, this is not, strictly speaking, from a metastasis, but from the operation of the same internal morbid condition which occasions the inflammation of the parotid.

In very rare instances epidemic parotiditis is of an unusually severe character. The fever and constitutional disturbance may be exceptionally high. Maniacal delirium and other severe cerebral symptoms have been observed.

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[1] *Lyon méd.*, No. 26, 1879.
In Laveran's collection of 432 cases in soldiers, orchitis occurred 156 times. In 32 cases of orchitis observed by Laurens (out of 118 of parotiditis), one testicle alone was affected in 26 cases, and both testicles in 6 cases. In 16 of these cases Laurens observed distinct atrophy within two months.

The patient has been known to sink into a typhoid state. Convulsions sometimes occur in children; but facial spasm is less infrequent. The gland has been known to suppurate in this disease. These occurrences, however, are so rare as to be regarded more as curiosities than of practical importance.

The communicability of this disease is generally admitted, but it has been denied by some writers of high authority; for example, by Valleix. Persons between twenty and thirty years of age are most susceptible to the contagium, but it occurs not infrequently under puberty. Males are more susceptible than females. It is one of the diseases which affect the same person but once.

The prognosis is generally favorable, the complications and accidents which have been mentioned being exceptional. Except that it occasions considerable discomfort, the disease in most cases is trivial. In the way of treatment it claims only soothing embrocations and the application of flannel, cotton batting, or wool to the neck, with some anodyne remedy if the pain be considerable. Bloodletting, purgation, or other of the so-called antiphlogistic measures are not indicated. The popular apprehension of danger from "taking cold" in this disease is based on the idea that if the affection of the parotid be arrested a metastasis is likely to take place. There is little ground for this idea, but a patient probably passes through the disease more comfortably by avoiding exertion and exposure.

Parotiditis occurs as an occasional complication of typhus and typhoid fever, pyæmia, and pneumonic fever. Under these circumstances it is a widely different affection from mumps. Suppuration takes place in the great majority of cases, the abscesses evacuating either externally or into the meatus auditorius, and frequently there is considerable sloughing of the areolar tissue. As a complication of fever it is attended with much suffering and danger.

Acute suppurative inflammation of the connective tissue around the submaxillary gland is called by German writers *angina Ludovici*. There is present in the suprahyoid region diffuse cellulitis, combined often with gangrene and sloughing of the tissues. The inflammation may extend to surrounding parts. The affection is a grave one. The most favorable termination is in abscess. Death occurs from œdema glottidis, secondary pneumonia, or septicæmia. The treatment is mainly surgical.

CHAPTER II.

INFLAMMATORY DISEASES OF THE STOMACH AND OF THE DUODENUM.

Acute Gastritis.—Subacute Gastritis.—Chronic Gastritis.—Phlegmonous Gastritis.—Chronic Interstitial Gastritis.—Gastro-duodenitis.—Duodenitis.

Acute Gastritis.

THE term acute has been applied to inflammation of the stomach by different authors with a widely different scope of signification. In the cases of gastritis to which heretofore, by many authors, the term acute has been restricted, the disease is attended with extreme danger to life, ending fatally as a rule. In these cases almost invariably the disease is caused by toxical agents—namely, the corrosive or acrid poisons. Acute gastritis was considered as thus restricted in the previous editions of this work. On the other hand, some authors include under the name acute gastritis or acute gastric catarrh gastric inflammation of every grade of intensity, and also certain affections generally regarded as functional. It is in accordance with the application of the term acute to other diseases to extend its scope, as applied to gastritis, so as to embrace all cases in which the inflammation has considerable severity, obliging patients generally to keep the bed. Acute gastritis will be considered in this sense in the present edition of this work. Cases in which the intensity of the inflammation is not sufficient to entitle it to be called an acute disease will be included under the heading Subacute Gastritis. Cases of chronic gastritis are those in which subacute inflammation is more or less persistent.

Adopting the more comprehensive scope of the term acute gastritis, the disease, exclusive of toxical cases, is not of very frequent occurrence—a fact which would not *a priori* be supposed, considering the quantity of ingesta received into the stomach daily, the various stimulating and irritating substances which often enter into these, the great functional activity of the organ, the amount of blood which it claims during digestion, and the extensive glandular apparatus which it contains.

Anatomical Characters.—More knowledge and caution are requisite in determining the existence of inflammation of the stomach than of any other organ. The stomach often presents after death various appearances simulating disease, but which are independent of any morbid process. These deceptive appearances are dependent chiefly upon two factors—namely, the varying amount of blood in the organ within physiological limits, and the rapid post-mortem softening of the mucous membrane in consequence chiefly of self-digestion. These changes, which are most intense in the fundus of the stomach, will be mentioned subsequently under the head of Softening of the Stomach.

It is very difficult to discriminate the milder grades of gastric inflammation from post-mortem changes. Hence, the observations of Beaumont upon Alexis St. Martin, who had a fistulous opening into his stomach, are of special value, and are usually cited as descriptive of the mild forms of acute inflammation of the stomach. These morbid appearances were noticed after indiscretion in eating or following the abuse of ardent spirits, and were accompanied with corresponding local and constitutional symptoms. They rapidly disappeared under proper medication and attention to diet. Beaumont noticed during these attacks of mild gastric inflammation a livid, erythematous redness distributed in irregular, small patches. Sometimes the mucous membrane was pale. Ecchymoses and oozing of grumous blood from certain red spots of the gastric mucous membrane were also observed. The secretion of gastric juice was deficient, and was replaced by a considerable quantity of ropy, alkaline mucus which became yellowish or muco-purulent when the inflammation was unusually severe. Slightly elevated discolored spots, resembling deposits of false membrane and called apthous by Beaumont, were also described.

In cases of more severe gastritis the mucous membrane usually presents irregularly scattered spots of congestion. Occasionally the redness is more diffused, and it may happen that the inner surface of the stomach presents a pale rather than a congested appearance. Ecchymoses, especially upon the summits of the gastric rugæ, may be present in large number. The mucous membrane is thickened, softened, and more opaque than normal. Its surface is coated with a thick layer of tenacious mucus or muco-pus. The swelling may extend to the submucous tissue. The changes are most marked usually in the pyloric portion of the stomach, and are more distinctive of inflammation when in this situation than when in the fundus. Hemorrhagic erosions, which consist of round or oval superficial abrasions of the mucous membrane upon a hemorrhagic base, are not infrequent.[1] Upon microscopical examination hyperæmia of the capillaries of the mucous membrane, particularly of those about the summits of the gastric tubules, and extravasations of blood, are detected. The intertubular tissue is thickened by the presence of serum and of extravasated red and white corpuscles. The accumulation of white corpuscles or pus-cells is most evident about the muscularis mucosæ. They may also be present in the submucous tissue. The epithelial cells of the gastric tubules may either remain unchanged or they may undergo cloudy swelling; that is, become enlarged by the accumulation of albuminous granules (p. 53). In some cases this may be followed by fatty degeneration. Sometimes the tubules appear to be filled with a granular matter containing nuclei, but without distinct cell-boundaries. This parenchymatous degeneration in the glands of the stomach often occurs in fevers, particularly in infectious diseases, and sometimes in phosphorus-poisoning, without evidence of inflammation, but with symptoms of disturbed digestion during life.[2] The cylindrical epithelial cells lining the inner surface of the stomach and the mouths of the gastric tubules become swollen with a secretion of mucus which escapes from their free ends. These cells are found in the mucus coating the mucous membrane, but to what extent they desquamate during life is a matter of uncertainty. The solitary follicles of the stomach (or lenticular glands, as they are also called), when present, are often swollen. They sometimes seem to be increased in number in inflammatory conditions of the stomach. Wilson Fox describes the development of follicular ulcers from these structures.

It is not intended to describe here the manifold morbid appearances belonging to toxic gastritis; that is, gastritis produced by swallowing irritant poisons. These are in some cases characteristic of the action of special poisons, and are described in works on special pathological anatomy and on forensic medicine. The corrosive poisons, such as strong mineral acids and alkalies, cause necrosis and sloughing of the mucous membrane to a greater or less extent, and may produce such destruction of the coats of the stomach as to lead to perforation of its wall during life, with the escape of its contents into the peritoneal cavity. Some of these poisons cause the formation of a diphtheritic exudation in the gastric mucous membrane. They may be accompanied by characteristic changes in the color of the mucous membrane and of the contents of the stomach. When corrosive poisons are swallowed, more or less inflammation, and perhaps sphacelation, may be present in the mouth, pharynx, and œsophagus, but these signs are sometimes wanting. The inflammation in cases of acute gastritis may extend into the duodenum.

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[1] Brinton describes a special and severe form of gastritis characterized by the presence of a large number of hemorrhagic erosions. The contents of the stomach are stained with blood. He calls the affection hemorrhagic erosion of the stomach or ulcerative gastritis. Hemorrhagic erosions, however, are frequently found in the stomach under a great variety of conditions, and it is not probable that they possess much pathological significance.

[2] Virchow, in conformity with his views of inflammation, describes this parenchymatous change as inflammatory, and designates the process in this situation as glandular gastritis or gastradenitis; but Cornil and Ranvier justly remark that the inflammatory nature of this lesion is very doubtful. Ebstein finds, in cases of poisoning by phosphorus or by alcohol, the parenchymatous degeneration most marked in the chief, or adelomorphous cells of the gastric tubules (*Virchow's Archiv*, 1872, Bd. 55, S. 469).

CLINICAL HISTORY.—Acute gastritis gives rise to pain more or less intense, referred to the epigastrium, together with a painful sense of constriction. The pressure of the diaphragm upon the stomach in inspiration sometimes increases the pain, so that the breathing may be costal and the number of respirations per minute is increased. Nausea and vomiting are prominent symptoms. In some cases the stomach is intolerant of water or the blandest liquids, even in very small quantities. The vomited matter is muco-serous, of a greenish color, the presence of bile being evident from the bitter taste, and not infrequently it is sanguinolent. The acts of vomiting occasion suffering. The thirst is frequently, but not always, intense. The desire for cold water is sometimes so irresistible that the patient is not deterred from drinking it by the painful acts of vomiting which it provokes. Tenderness on pressure over the epigastrium is often marked. The pulse is more or less frequent and small. The temperature of the skin is moderately, and sometimes considerably, raised. The thermometer, however, as a rule, shows but a moderate fever-heat, rarely exceeding 101° or 102° F. The bowels are constipated except in cases of poisoning. Dysphagia and aphonia are occasional symptoms. The tongue presents no special morbid appearances. The buccal fluids are diminished and frequently have an acid reaction. The patient suffers from cephalalgia. The mind is depressed and the expression is anxious and haggard. If life be not speedily destroyed and the disease run on to a fatal termination, vomiting of black, grumous, coffee-ground matter may occur, especially in cases of toxical gastritis. Toward the close of life the contents of the stomach are ejected by acts of regurgitation rather than by vomiting. The prostration in fatal cases becomes extreme. Hiccough occurs. The pulse becomes very frequent and thready. Coldness of the surface is marked. The mode of dying is by asthenia. If the disease end in recovery, improvement in all the symptoms takes place slowly; convalescence is gradual, and the inflammation may persist in a chronic form. In cases of gastritis produced by the corrosive poisons, if perforation of the stomach occur, the symptoms of peritonitis are added. Jaundice occurs in some cases, showing that the inflammation extends into the duodenum. The foregoing sketch of the symptomatology applies to cases of marked severity. In less severe cases the symptoms are essentially the same, but with less intensity.

PATHOLOGICAL CHARACTER.—The stomach, aside from its office as a receptacle of the ingesta, is a glandular organ, furnishing daily a large quantity of an important secretion—the gastric juice. In its reluctance, so to speak, to take on acute inflammation from the internal and inappreciable causes which give rise to the so-called spontaneous inflammations in other situations, it resembles other glands, such as the liver, pancreas, and kidneys. When acute inflammation is developed in this situation it does not differ in pathological character from acute inflammation seated elsewhere in a mucous structure. The gravity of the disease depends on the physiological relations of the stomach. Acute gastritis produced traumatically by the action of caustic or irritant poisons is developed like inflammation from the action of the same local causes in other situations.

CAUSATION.—Exclusive of the acrid or corrosive poisons, the most frequent cause in this country is spirit-drinking. I have known the disease to directly follow a debauch, and to prove rapidly fatal. A person with chronic gastritis induced and kept up by habitual spirit-drinking is liable to the acute affection after an unusually excessive indulgence. Exposure to cold may act as an auxiliary cause. In a case under my observation, the patient being an habitual drinker, the disease followed sleeping in a cold cellar. Over-repletion of the stomach with stimulating and indigestible food may sometimes give rise to acute gastritis. This so-called crapulous gastritis is generally subacute. It is a traditional error to consider as exciting causes drinking cold water when the body is perspiring, intense mental emotions, and contusions of the abdomen.

DIAGNOSIS.—The symptoms of acute gastritis are strongly diagnostic. There is greater probability of the disease being incorrectly supposed to exist than of its being mistaken, when existing, for other diseases. Vomiting, however frequent and persistent, is never alone sufficient evidence of gastritis. I have known a case of chronic meningitis with persistent vomiting to be treated for acute gastritis, the error not being discovered prior to the autopsy. Peritonitis, if accompanied with vomiting, may assume an appearance of gastritis, but the diffusion of tenderness over the abdomen, the tympanitic distension, and the rigidity of the abdominal muscles generally suffice for this differential diagnosis. Gastritis has been mistaken for pneumonitis, but physical exploration of the chest should enable the physician to detect or exclude the latter affection.

Gastralgia was formerly confounded with gastritis. The paroxysmal recurrence of pain or its occurrence in marked exacerbations, the absence of febrile movement, of vomiting, as a rule, and of tenderness on pressure, and the ability to take food, sometimes with relief of the pain, sufficiently characterize the affection as neuralgic.

With a view to appropriate antidotal treatment, and also in a medico-legal point of view in certain cases, it is desirable to determine from the symptoms whether acute gastritis be attributable to poison. This is always to be suspected in severe cases if other causes be not evident. Burning in the throat is complained of if a caustic or corrosive poison have been taken, and the local action of the poison may be seen in the mouth and fauces. The matters vomited are likely to contain blood. Diarrhœa is likely to occur. If caused by a poison the disease is developed suddenly, and inquiry is to be made to ascertain if the sudden development were not immediately preceded by a meal or the ingestion of something in the way of food or drink.

PROGNOSIS.—Cases of acute gastritis, in the restricted sense in which the term acute was formerly used, were, in the great majority of cases, fatal. In the more comprehensive scope of the term the disease ends in recovery, as a rule, if toxical cases be excluded. Death sometimes takes place within a few hours, patients falling quickly into a condition of collapse. These cases, however, are exceptional. The mode of dying is by slow asthenia.

When the disease is due to the action of a corrosive poison the danger will depend, other things being equal, on the amount of local injury.

TREATMENT.—The indications for bloodletting are not present in this disease. Bearing in mind that the danger is from asthenia and that life is sometimes quickly destroyed, the abstraction of blood is not admissible. The first and most important object in the treatment is to secure for the inflamed organ absolute rest. With reference to this object the less of anything introduced into the organ the better. To allay intense thirst small pieces of ice may be swallowed, or iced water taken often in a very small quantity at a time. The physician should resist the temptation to try in succession the host of remedies to allay vomiting, all of which will be likely to aggravate this distressing symptom. It is desirable to administer opiates in order to quiet the stomach and render the system more tolerant of the disease. A salt of morphia placed dry upon the tongue may be tried, and if vomiting be provoked by this mode of administration it should be administered hypodermically or some form of opiate given per enema. Fomentations or a blister may be applied over the epigastrium. Cathartics are inadmissible. The bowels may be moved by stimulating enemas. Mercury, either in large or small doses, is not indicated. The importunities of the patient for an emetic are to be resisted. There may be a feeling as if the stomach were distended or as if there was an accumulation aggravating the distress. This feeling arises from the inflamed state of the membrane, as tenesmus is incident to inflammation of the rectum, and it may be called gastric tenesmus.

Alimentation is not less important than in other diseases, and in proportion as the symptoms denote danger in the direction of asthenia supporting measures of treatment are indicated. The stomach will not tolerate alcoholics and food; moreover, their introduction into the stomach conflicts with the most important object of treatment—namely, absolute rest of the inflamed organ. The rectum is to be substituted for the stomach as regards the reception of ingesta. This is a disease in which rectal alimentation is of invaluable service. Alimentary support may be adequately provided thereby during a period as long as may be required; that is, until the acute symptoms have subsided and the stomach will tolerate bland nourishment. From three to four ounces of defibrinated blood, Leube's pancreatic meat emulsion or his "meat solution," or milk with the addition of egg (the election of any of these articles being made after experimental trials, and perhaps the different articles employed in alternation), may be injected every four or six hours. If alcoholic support be indicated, spirit may be added to the rectal food according to the urgency of the indication, or the latter may be administered hypodermically. The addition of a little laudanum to each injection is generally advisable. Thirst may be relieved by the injection of water from time to time and frequent sponging of the body. The danger from the disease is much lessened and the duration shortened by pursuing this course.[1]

Milk with the addition of lime-water will be likely at first to be best borne by the stomach, given in small quantities at short intervals. Other forms of food are to be gradually added. After the inflammation has ceased the stomach is left in an atonic condition, and remedies of a tonic character are indicated during convalescence. If the acute end in the chronic form of inflammation, the treatment due to chronic gastritis is to be employed.

Acute gastritis caused by the acrid or corrosive poisons is to be treated after the plan just stated, with the addition of measures having reference to the evacuation and neutralization of the poisonous substance. Emetics are to be promptly given, and if they fail to act efficiently the stomach-pump is to be resorted to, provided the poison be one which can be ejected. Arsenic, corrosive sublimate, alcohol, antimony, cantharides, copper, phosphorus, together with a great number of vegetable irritants, such as capsicum, gamboge, croton oil, etc., may be expelled from the stomach; but if any of the corrosive acids have been taken, reliance must be placed on the prompt administration of antidotes. Antidotes are also to be given after the employment of emetics or the stomach-pump to effect the removal of acrid poisons.

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[1] The pancreatic meat emulsion is prepared as follows: To 5 ounces of finely scraped meat add 1½ ounces of finely chopped pancreas and 3 ounces of warm water. The mixture should have the consistence of thick soup. Leube's meat solution is prepared by adding hydrochloric acid with water, and employing heat by means of a Pepin's digester. (Vide "Ueber die Therapie der Magenkrankheiten," von W. O. Leube, *Sammlung klinischer Vorträge von Volkmann*, No 62, Leipzig, 1873.) The bowels should be thoroughly washed out by a large enema both before beginning rectal alimentation and afterward from time to time. Laudanum added to the injections promotes their retention, and probably their absorption.

(For details respecting the management of cases of poisoning the reader is referred to works which treat of toxicology.[1]) I shall simply subjoin, for the convenience of the practitioner, an enumeration of the antidotes for the more important of the poisons which act locally upon the stomach.

Arsenical Preparations: The hydrated sesquioxide of iron is the special antidote. It should be recently prepared and given in large quantity. If this preparation be not immediately available, the sesquioxide or common red oxide of iron (crocus martis) may be given in its stead. Bouchardat advises to combine magnesia with the preparation of iron, in order to promote the passage of the iron from the stomach into the intestines.

Corrosive Sublimate and other Salts of Mercury: The most reliable antidote is albumen, and the white of eggs is the best and most available article.

Salts of Copper: Albumen is an antidote. Iron reduced by hydrogen and the hydrated persulphate of iron are said by Bouchardat to be antidotal both to the salts of mercury and of copper.

Tartar Emetic: Substances containing tannin in abundance and magnesia.

Nitrate of Silver: Chloride of sodium.

Mineral Acids: Magnesia or chalk mixed with water or milk; the alkaline carbonates or soap.

Oxalic Acid: Magnesia, chalk. Whiting or plaster scraped from the wall of an apartment may be given, mixed with water. Alkalies are not to be given.

Ammonia: Vinegar or any of the vegetable acids.

Potassa and Soda: The same.

Subacute Gastritis.

Subacute gastritis, not becoming chronic, but of transient duration, occurs not infrequently. It is not easy to say with positiveness how frequently it occurs, because practically it is difficult to draw the line of demarcation between slight inflammation and merely functional disturbance in this situation. Subacute gastritis doubtless enters into certain cases of the affection which will be noticed in connection with functional disorder of the stomach under the name of Acute Dyspepsia. Subacute inflammation of the stomach is much more frequent in infants and young children than in the after periods of life. As here considered, reference will be had chiefly to the affection occurring at a time subsequent to the period of infancy.

The degree of inflammation in different cases of subacute gastritis varies, being either slight or moderate as evidenced by the local and general symptoms. The local and general symptoms are marked according to the degree of the inflammation. The ingestion of food is followed by uneasiness or distress referred to the epigastrium, and by symptoms denoting indigestion—namely, flatulence, eructations, etc. Thirst is a symptom usually more or less prominent. Tenderness over the epigastrium and a furred or coated tongue are other symptoms. The urine is high-colored and loaded with the urates. In some cases there is constipation, and in other cases diarrhœa, the latter being due to intestinal indigestion. Pain in the head is a frequent and sometimes a prominent symptom. There may be slight pyrexia, but it is frequently or generally wanting, except after the ingestion of food or stimulants. When transient febrile movement is not thus produced, the pulse is usually feeble and the extremities are cold. There is more or less general debility, but the sense of weakness exceeds the actual reduction of strength.

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[1] *Taylor on Poisons* may be consulted for this purpose; also, article "Poisons" in *Dunglison's Medical Dictionary.*

The spirits are depressed, and the suffering may be considerable from malaise without any definite location of pain or distress.

This is a sketch of a class of cases which the practitioner meets with frequently, and which, together with cases of the affection to be considered in another chapter under the name of Acute Dyspepsia, is properly known as a "bilious attack."

If the gastric inflammation be greater, approximating in intensity to acute gastritis, the local and general symptoms are more marked. There is suffering from gastric distress, and sometimes from a dull, obtuse pain in the region of the stomach. The epigastric tenderness is often considerable. Anorexia is complete, with loathing of food and nausea. Vomiting is often a prominent symptom. Mucus is vomited in abundance, and if the efforts of vomiting be prolonged bile is rejected. The patient experiences a temporary sense of relief after vomiting. The feeling is as if the stomach were distended, and acts of vomiting are provoked and aided by voluntary efforts with the idea that if the contents of the stomach were effectually discharged the relief would be permanent. This feeling arises from the inflamed condition of the membrane, and is analogous to the sensation connected with tenesmus in dysentery. It may be called gastric tenesmus. Thirst is generally urgent and cold drinks are craved. The tongue is coated at first, but in the course of the affection it may become clean and reddened. Either constipation or diarrhœa may exist. There is more or less febrile movement, with evening exacerbations, as shown by the pulse and axillary temperature; the latter, however, rarely exceeds 100° F. Chilly sensations and rigors are common, continuing for a few seconds and recurring irregularly. Cephalalgia is generally marked, and in some cases is accompanied by a sense of tension or a feeling as if the head would break. There is often a sense of prostration, with depression of spirits and sometimes a notable degree of mental apathy. In cases of maximum severity subacute gastritis so closely approximates to acute gastritis that there is scarcely a well-marked line of division between the two forms of the disease.

In cases of gastritis with considerable pyrexia the disease is sometimes called "gastric fever." This name implies that the disease is primarily or essentially a fever, whereas the fever is purely symptomatic and secondary to the gastric inflammation.

Subacute gastritis, if the inflammation be slight or moderate, is usually of short duration, convalescence taking place in a few days. If the inflammation have considerable intensity the duration of the disease is from a week to three weeks. If the inflammation persist much beyond the latter date it is to be considered as chronic.

Subacute gastritis in some cases is evidently of dietetic origin. It is sometimes attributable to the decomposition of ingested aliment, owing to a deficiency of gastric juice or to the quantity of food ingested being out of proportion to the digestive capability of the stomach, and to the ingestion of food either difficult of digestion or in which putrefaction had begun prior to its ingestion. Persons who are habitually addicted to over-indulgence in eating, or to highly seasoned dishes and the free use of condiments, are doubtless more liable to this affection than others as a consequence of a gluttonous meal or when from any cause gastric digestion is arrested. The affection sometimes follows a prolonged debauch. The habitual free use of spirits begets a liability to it. It occurs especially among drunkards. It may be caused by any of the acrid or irritant poisons, not taken in sufficient quantity to produce acute gastritis. The so-called uræmic gastritis is caused by the irritant effects of the carbonate of ammonia, formed by the decomposition of urea, upon the gastric mucous membrane; and it is not improbable, as some authors have maintained (Garrod, Charcot), that in some cases of gout, uric acid is eliminated by the stomach, and by a similar irritant effect causes gastritis. In a certain proportion of cases the causes, both remote and proximate, are not apparent.

An affection to which subacute gastritis is closely affiliated is that to be considered under the name of Acute Dyspepsia. Gastric inflammation doubtless enters into certain cases of the latter affection, but the functional disturbance is disproportionately great as compared with the amount of inflammation, and is not to be accounted for by the latter; hence the propriety of considering it as a functional affection of the stomach. The differential diagnosis of these affections is not, practically, of great importance. The symptoms of gastritis are present in certain cases of remittent fever, and perhaps they proceed actually from subacute inflammation of the stomach in these cases. The symptomatic phenomena of remittent fever, however, are sufficiently distinctive to render its discrimination from simple gastritis easy. Cases of ephemeral fever, or febricula, are to be discriminated by the want of proportion between the febrile phenomena and the gastric symptoms. Cases of continued fever, typhus, and typhoid very rarely present symptoms of gastritis with sufficient prominence for the differential diagnosis to be a matter of question. In general, the diagnosis of subacute gastritis is not difficult.

In the TREATMENT of subacute gastritis rest of the inflamed organ is of primary importance. If the inflammation be but slight or moderate, it will suffice to place the patient on a diet reduced in quantity, consisting of milk with lime-water and farinaceous articles. In cases of greater severity complete abstinence for two, three, or four days may be advisable. The vomiting and loathing for food will be likely to secure this end without any injunctions on the part of the physician. In the mean time, if the symptoms denote exhaustion the patient may be supported by rectal alimentation. The abstraction of blood either locally or by venesection is never indicated. Benefit may be derived from sinapisms or a blister over the epigastrium. Emetics and purgatives are contraindicated. The sensations of the patient may lead to a desire for both, especially the former, but they do not effect any desirable object, and their local action upon the inflamed organ will do harm. If the bowels be constipated simple enemas may be employed. Mercury is not indicated, any more than in cases of inflammation affecting the mucous membrane or other portions of the digestive system. On the other hand, remedies to soothe the inflamed membrane and to allay vomiting are indicated—namely, bismuth, hydrocyanic acid, hyoscyamus, and especially the salts of morphia. Small pieces of ice may be swallowed, or iced water given in a small quantity at a time and often repeated, if found to be grateful to the patient. Carbonated water is not only taken with comfort, but appears to be useful.

It is important not to continue the abstinence from food or a great reduction of diet too long. As soon as nutritious articles of food are well tolerated by the stomach they are admissible. The improvement of diet should be made gradually and tentatively. During convalescence the mineral acids are useful as tonics, and after the inflammation has ceased other tonics may be appropriate. Patients who are spirit-drinkers should be told of the connection of the disease with their habits.

Chronic Gastritis.

Chronic gastritis is not infrequent, although it is not as common an affection as was supposed some years ago. Chronic functional disorders were formerly considered as inflammatory. It is not always easy to discriminate clinically between chronic inflammation and the functional affections embraced under the name of dyspepsia. The differential points involved in this discrimination will suffice for the clinical history and diagnosis of the affection. The affection occurs oftener in middle and advanced life than in children and young persons. It occurs in men oftener than in women.

The ANATOMICAL CHANGES in chronic gastritis are most frequently present and most marked in the pyloric region of the stomach. This localization applies also to the majority of morbid processes in the stomach. The color of the mucous membrane varies in different cases. It may be red, brown, ash-gray, slate-colored, or whitish. A bluish-red color, either diffuse or in spots, is usually present in the chronic inflammation attendant upon prolonged passive congestion of the stomach, such as accompanies cirrhosis of the liver, emphysema of the lungs, and heart disease. Ecchymoses and hemorrhagic erosions are frequently present. The mucous membrane, as a rule, is more or less pigmented, and this gives rise to brown, ashy, and slate colors. The pigment generally appears as little dots scattered over the mucous membrane. The same pigmentation often coexists in the intestinal tract. It is most frequent where there is mechanical congestion of the stomach in consequence of portal obstruction. It is referred to alterations in the hæmoglobin of extravasated red blood-corpuscles (Part I. p. 57), and is therefore regarded as a sign of preceding congestion and hemorrhage. The pigment exists in the form of dark granules, either free or in cells, in the intertubular tissue, and sometimes in cells within the tubules. The inner surface of the stomach is coated with a thick layer of viscid mucus or muco-pus. The consistence of the mucous membrane is sometimes peculiarly firm and tough; at other times the membrane is more or less softened. The thickness of the mucous membrane varies in different cases and in different parts of the stomach in the same case. Chronic inflammation leads both to thickening and to thinning of the mucous membrane. The latter result is often, although not invariably, consecutive to the former. The mucous membrane is usually thickened, but not infrequently it is atrophied in certain places, and exceptionally the whole membrane may be abnormally thin. Both in healthy and diseased stomachs the mucous membrane of the pyloric region not infrequently presents little rounded elevations separated by slight furrows. This condition, compared sometimes to granulations upon wounds, is called mammillation or *l'état mamelonné* of Louis. When present in healthy (and often in diseased) stomachs it is due to contraction of the muscularis mucosæ (Brücke's muscle). In chronic gastritis, however, it may also be referable to irregular thickening of the mucous membrane. The furrows may correspond to atrophic spots. Exceptionally, polypoid growths, due to localized increase of connective tissue and enlargement and proliferation of the gastric tubules, are present, even in considerable number (gastritis polyposa). Little cysts about a millimetre in diameter are frequently observed in the mucous membrane. They are the result of distension of parts of the gastric tubules with a clear mucoid secretion. Sometimes the cysts attain a much larger size. The submucous tissue is frequently thicker and denser than normal. There may also be some hypertrophy of the muscular coat. Upon microscopical examination the interstitial tissue of the mucous membrane is usually found to be increased in amount. The gastric tubules may be enlarged in consequence of swelling and parenchymatous or fatty degeneration of their epithelial cells. Usually the tubules are affected in groups, some remaining unchanged. The degenerative change may lead to complete destruction of the epithelial cells. The tubes are then partly or completely filled with albuminous and fatty detritus. Finally, the tubules may become atrophied, and in some places even obliterated. According to W. Fox, fatty degeneration may occur in the connective tissue as well as in the glands. The cystic transformation to which reference has been made is attributable to partial occlusion of the tubules in consequence of the new growth of interstitial tissue. A change which occasionally occurs is proliferation or adenomatous growth of the tubules. In the neighborhood of the pylorus little villous projections of vascularized connective tissue are often met with in chronic gastritis. In the atrophied portions of mucous membrane the tubules are shorter and narrower than normal. Such places have usually a smooth surface and are pigmented.

SYMPTOMS denoting indigestion or difficult digestion are present in cases of chronic gastritis, but these alone are not diagnostic, since they alike exist in cases of dyspepsia. Pain may not be present, and if present it is not distinctive. A burning sensation or a feeling of heat in the gastric region is somewhat diagnostic. Tenderness over the epigastrium is somewhat diagnostic, provided it be limited to that region and constant; that is, not present merely during the process of digestion or when the stomach is distended. Thirst is diagnostic if habitual; that is, present not only during the process of digestion, but at other times. The appetite is much oftener impaired in connection with chronic gastritis than in cases of dyspepsia, the appetite being generally preserved, and often craving, in the latter. The nutrition is oftener defective in cases of chronic gastritis. Dyspeptics often preserve their weight undiminished. In chronic gastritis stimulating articles of food, such as meat and condiments, are not as well borne as bland aliments, the reverse being oftener the case in dyspepsia. Alcoholic and other stimulants produce gastric distress in cases of chronic gastritis, whereas they often relieve distress incident to merely functional disorder. Nausea and vomiting are more likely to occur in connection with chronic gastritis, excluding cases of so-called acute dyspepsia and certain cases of functional disorder characterized by vomiting. In ordinary cases of dyspepsia nausea and vomiting are not common. A slight grade of febrile movement is sometimes observed in chronic gastritis, and very rarely in a purely functional disorder.

The DIAGNOSIS is to be based on the combination of more or less of the foregoing differential points. In making the diagnosis certain structural affections of the stomach are to be excluded—namely, carcinoma and ulcer. The diagnostic symptoms of these affections will be considered in Chapter III.

Chronic gastritis may follow an acute or subacute inflammation of the stomach developed spontaneously or produced by the acrid or corrosive poisons. It may proceed from excesses in eating or spirit-drinking, and on the other hand it is stated that long fasting or too rigid dieting may give rise to it. In dogs destroyed by starvation Andral found the stomach inflamed and ulcerated; but similar experiments made by Collard de Martigny were negative as regards appearances of inflammation in this organ.[1] Stark, who fell a victim to experiments in dietetics in 1869, had symptoms of gastritis developed during prolonged abstinence. Arsenic introduced into the system by absorption from an external wound gives rise to gastritis, as was ascertained by Brodie and verified by the observations of others. Chronic gastritis also frequently results from long-continued venous congestion of the stomach in consequence of obstacles to the circulation in the liver, lungs, and heart. This chronic congestion is most intense, *cæteris paribus*, when the obstacle is in the liver. Emphysema of the lungs, chronic pleurisy, valvular lesions of the heart, and especially cirrhosis of the liver, are often accompanied by chronic gastritis.

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[1] Magendie, *Journal de Physiologie*, tome viii.

It occurs also in connection with gout and with pulmonary phthisis. Chronic gastritis occurs in certain cases of renal disease involving uræmia; the explanation being that already given—namely, that the carbonate of ammonia which is formed from the decomposition of the urea eliminated by the gastric mucous membrane acts as a local irritant.

The first point in the TREATMENT is the removal of dietetic causes which may have originated, or which may tend to perpetuate, the affection. The patient is to be placed on a bland and easily-digested diet, consisting of milk, eggs, stale bread, soft-boiled rice, and other farinaceous articles—articles digested in the small intestine. Meat and stimulants are to be interdicted. For a short time it may be advisable to reduce the quantity of food below the amount which the wants of the system require. Food should be taken in small quantities and at shorter intervals than in health. Moderate counter-irritation over the epigastrium is useful. The remedies to be administered are few. Solicitations for emetics and cathartics are to be resisted. Patients often declare that they are "bilious" or that there exists "foulness of the stomach," and hence that they need evacuant medicines; but in place of medicines which from their irritant action will tend to increase or prolong the inflammation, remedies to soothe the inflamed membrane are indicated—namely, small doses of morphia or codeia, hyoscyamus, hydrocyanic acid, bismuth, etc. After the inflammation has ceased functional debility or atony remains. Tonics and improved diet are then required. It is a point of delicacy to determine when to make this change in the treatment. It is like resorting to passive motion in cases of fracture or dislocation. The change is to be begun tentatively, and to be continued or not according to the effect. The symptoms of functional disturbance and the indications for treatment which may be present after the inflammation ceases will be considered in connection with Dyspepsia. (Vide Chapter IV.)

Phlegmonous Gastritis.

By phlegmonous gastritis is understood suppurative inflammation of the walls of the stomach. This disease is very rare. The submucous tissue is primarily involved, but the inflammation may extend to the other coats of the stomach. Synonyms for this disease are gastritis submucosa (Dittrich), suppurative linitis (Brinton), and interstitial suppurative gastritis (Auvray). The affection appears either as a circumscribed collection of pus in the walls of the stomach or as a diffuse purulent infiltration of the ventricular coats. The former condition, which is the more frequent, is also called abscess of the stomach, which may be single or multiple. It may attain the size of the fist. The purulent collection is in the submucous coat, and it frequently invades the muscular layer. The mucous and serous layers may be intact. There is usually, however, a localized peritonitis over the seat of the abscess. The abscess may rupture through the mucous membrane into the cavity of the stomach, or less frequently through the serous coat into the peritoneal cavity. In rare instances recovery may take place after the escape of the pus into the cavity of the stomach. A cicatrix then results, and this may lead to stenosis of the stomach.

Purulent infiltration of the submucous coat usually involves only a part of the stomach; it may, however, affect its whole extent. The suppurative process may also extend into the lower part of the œsophagus and into the duodenum. The wall of the affected portion of the stomach is thickened; and it may be six or eight times thicker than normal. The mucous membrane is usually swollen and spongy in texture. It generally presents a number of little round openings, through which pus can be pressed out. Sometimes larger irregular ulcers are present. Upon cutting through the wall of the stomach the accumulation of pus is found to be greatest in the submucous coat, whence purulent septa often extend between the bundles of fibres in the muscular coat. The subserous tissue also frequently contains pus. In fact, while the lax submucous and subserous tissues suffer the most, microscopical examination usually shows the presence of large numbers of pus-cells in the firmer mucous and muscular coats of the stomach. Purulent infiltration may be accompanied by the presence of circumscribed abscesses. The suppurative inflammation exceptionally may not have advanced to the breaking down of tissue, so that the infiltration with pus-corpuscles is evident only upon microscopical examination. Upon microscopical examination, in addition to large quantities of pus-cells, are found many micrococci, which, at least in some cases, are streptococci. Circumscribed inflammation of the serous covering of the stomach (perigastritis) is often present, and not infrequently there is general peritonitis. Infectious thrombi may form in the veins of the stomach, and by the separation of emboli may lead to secondary abscesses in the liver and lungs. Phlegmonous gastritis is one of the causes of suppurative pylephlebitis. In a number of cases there have been evidences of a previous chronic gastritis, with fibrous thickening of the submucosa.

Etiologically, phlegmonous gastritis may be divided into two varieties—a primary, idiopathic; and a secondary, metastatic. Metastatic phlegmonous gastritis, which is usually in the form of circumscribed abscess, is secondary to severe infectious diseases, such as pyæmia and puerperal fever. The primary variety has been observed most frequently in drunkards. Other causes which have been assigned, the efficiency of which is doubtful, are injuries in the region of the stomach, errors in diet, and ulcer of the stomach. In most cases of the primary variety it is impossible to assign an efficient cause. Males are more frequently attacked than females. It has usually occurred in the middle period of life.

The SYMPTOMS of the metastatic variety are usually obscured by those of the primary disease. The idiopathic form generally runs an acute course, terminating in from one to three weeks. Its progress, however, may be chronic and insidious. As a rule, the disease begins with a chill and may be attended by repeated chills. The temperature is elevated, often considerably. The pulse is rapid and feeble. The patient suffers from thirst and anorexia. There are usually severe pain and tenderness in the epigastric region, but these may be absent. Vomiting and retching are prominent symptoms. The stomach is generally more or less distended with gas. In some cases there has been constipation, and in others diarrhœa. Prostration sets in early. Brinton observed icterus in some cases. Toward the end the patient becomes delirious. Death is usually preceded by a state of collapse. The fatal termination is sometimes sudden. These symptoms may be complicated by those of acute peritonitis. As a rule, no pus has been detected with the naked eye in the vomited material. Sometimes, however, when an abscess has burst into the stomach a large quantity of pus has been vomited. Occasionally an abscess in the wall of the stomach is of sufficient size to be felt during life as a tumor. The duration of the disease is usually from two to ten days, but it may be longer.

There are no diagnostic symptoms of phlegmonous gastritis. Its recognition during life is usually impossible. It may, however, sometimes be suspected by excluding other conditions, especially general peritonitis. In some cases its presence has been rendered probable by the detection of a swelling connected with the stomach, the disappearance of which was followed by the vomiting of a quantity of pus. But even in this case it would be difficult to exclude the rupture of an abscess which had taken its origin outside of the stomach; for instance, a circumscribed collection of pus in the peritoneal cavity.

The termination of phlegmonous gastritis is usually fatal. There is reason, however, to believe that abscess of the stomach may end in recovery.

The TREATMENT indicated in phlegmonous gastritis has for its objects complete rest of the affected organ and the relief of pain. The first of these objects is secured by rectal alimentation and opiates. The relief of pain is effected by the latter in conjunction with emollient and soothing applications over the epigastrium.

Croupous and diphtheritic inflammations of the stomach have been described, but they are little understood and are without clinical interest, being unattended by distinctive symptoms. Pseudo-membranous gastritis is usually secondary to some other disease, such as croup and diphtheria, typhoid fever, pyæmia, or puerperal fever. It may be produced by swallowing acrid poisons.

Chronic Interstitial Gastritis—Cirrhosis of the Stomach—Hypertrophic Stenosis of the Pylorus.

The disease known under these names is characterized by thickening of the walls of the stomach in consequence of new growth of connective tissue. The affection was clearly described by Brinton, who proposed for it the names plastic linitis and cirrhosis of the stomach. It has also been called fibroid degeneration or infiltration, induration, sclerosis, and hypertrophy of the stomach. The name chronic interstitial gastritis implies that the morbid process is a chronic inflammation of the interstitial tissue of the stomach, analogous to that of the liver in cirrhosis. The affection is referred to by most German writers under the name of chronic catarrhal gastritis, with which, although often associated, it should not be confounded. The disease, although rare, has been observed in several typical cases. In such cases the stomach is small and increased in weight and density. The diminution in its capacity may be extreme. Its cavity may not be larger than a medium-sized pear. In certain instances the stomach has been described as normal in size or even dilated. The thickness and increased density of the ventricular walls may be such that upon incision the organ does not collapse. The stomach-wall may measure an inch or more in thickness. The organ may be uniformly affected or the change may be greatest in the pyloric region. When the pyloric region is alone affected the remainder of the stomach is dilated instead of contracted. Upon examining a section through the wall of the stomach the coats are found to present an unnatural uniformity. Their consistence is firm, almost cartilaginous. The new growth of connective tissue, to which the change is mainly due, involves usually all of the coats of the stomach, but especially the submucous, which appears as a dense, white layer. The muscularis, especially the transverse layer, is frequently much hypertrophied, and presents a grayish, translucent appearance. It is usually invaded by the new growth of fibrous tissue. The subserous tissue is also thickened and fibrous. The serous coat is of a peculiar opaque, pearly-white color. The mucous membrane may be intact, but it is generally involved in the morbid change. Its interstitial tissue is thickened, the tubules are atrophied, and they may even disappear over a considerable extent of the stomach.[1] In some cases no changes in the gastric glands have been noticed.

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[1] In a case reported by Nothnagel, and examined microscopically by W. Müller, glands were found only in the pyloric region (*Deutsches Archiv f. klin. Med.*, 1879, Bd. 24, S. 353).

The thickening is sometimes confined to the neighborhood of the pylorus. This condition has been called fibroid degeneration of the pylorus (Habershon) and hypertrophic stenosis of the pylorus (Lebert). In works devoted specially to diseases of the stomach it appropriately receives separate consideration. The thickening here also is due to formation of new fibrous tissue and to hypertrophy of the muscular coat. This lesion induces stricture of the pylorus, which may amount almost to complete closure of the orifice. In consequence of pyloric obstruction, the stomach becomes dilated and its muscular coat is hypertrophied. In a case of hypertrophic stenosis of the pylorus, related by Nauwerk,[1] the stomach occupied two-thirds of the abdominal cavity. This lesion has been confounded with scirrhous cancer of the pylorus, from which it is to be distinguished only by microscopical examination. In some cases of hard cancer of the stomach the cancerous alveoli are few in number and separated by a large quantity of fibrous tissue.

The SYMPTOMS of cirrhotic contraction of the stomach are not sufficiently distinctive to admit of a positive diagnosis. The affection is a grave one, and usually terminates fatally. Sometimes, like other severe affections of the stomach, it is unattended by symptoms. Usually the symptoms at first are those of simple chronic gastritis, such as indigestion, loss of appetite, vomiting, and feeling of oppression in the epigastrium; but these symptoms are inconstant and possess nothing characteristic. As the disease progresses a more severe affection is suspected, usually cancer of the stomach. The patient loses flesh and strength. In an interesting case reported by Nothnagel the patient presented all of the essential symptoms of pernicious anæmia, including hemorrhages in the retina and elsewhere. To a certain degree characteristic of cirrhotic contraction are the inability to take more than a small quantity of food or drink at a time and the sense of fulness which follows the ingestion of this small quantity. The patient thus suffers from inanition, both in consequence of the small capacity of the stomach and from the disturbance of its digestive functions. Sometimes, but not always, a hard mass presenting the contracted contours of the stomach can be felt through the abdominal wall. In a few cases the vomiting of coffee-ground material has been noted, but this occurrence is exceptional. Pain is not usually a prominent symptom. Constipation has been the rule.

In a case under my observation, in which the walls of the stomach were found to be from half an inch to an inch in thickness everywhere except at the pyloric third, the patient up to a few weeks before death supposed that he was simply dyspeptic, his ailments not leading him to seek medical advice. In this case the organ formed a resisting, movable tumor felt through the abdominal walls, and the disease was supposed to be carcinoma.

When the fibroid metamorphosis is confined to the pylorus the symptoms are those of stenosis of the pyloric orifice, combined with dilatation of the stomach. Prominent among these symptoms are vomiting of accumulated ingesta and obstinate constipation. The symptoms are explicable by the obstacle to the entrance of substances from the stomach into the intestine. The symptoms and signs of dilatation of the stomach will be considered hereafter. Nauwerk, in the report of the case of hypertrophic stenosis to which reference has been made, gives an interesting example of the length of time during which ingesta may remain in the stomach. He found in the stomach, at the autopsy, cherry-stones which had been swallowed three months previously, and which had remained, although the stomach had been washed out and the patient had vomited repeatedly. Sometimes the thickened pyloric part of the stomach can be felt during life as a small, hard, movable tumor.

The DIAGNOSIS of cirrhotic contraction of the stomach and of hypertrophic stenosis of the pyloric valve cannot usually be made with any degree of certainty. The disease is most likely to be confounded with cancer of the stomach. The following points are to be considered in attempting to reach a probable diagnosis: Cirrhosis of the stomach is often of much longer duration than cancer; it may attack persons much younger than those usually affected with cancer; pain in the region of the stomach and vomiting of blood are less frequent and prominent symptoms in cirrhosis than in cancer; the inability to take more than a small quantity into the stomach is a more marked symptom in cirrhosis than in cancer. Upon no single one of these points, however, can much positive value be placed in a differential diagnosis; nor is too much weight to be placed upon the detection of a hard tumor simulating the contours of the stomach. Such an occurrence is rare and would not suffice to exclude cancer. It has been proposed to determine the size of the stomach by causing the evolution of carbonic acid gas in its cavity, according to the method first proposed by Frerichs (vide Dilatation of the Stomach, Chapter III.), but this procedure has not yet been employed in contraction of the stomach, and it could not well serve as a basis of a positive diagnosis. The diagnosis of cirrhosis of the stomach, then, may in certain cases be rendered probable by a combination of favorable circumstances, but it can hardly become a certainty.

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[1] *Deutsches Archiv f. klin. Med.*, 1878, Bd. 21, S. 578.

The ETIOLOGY of chronic interstitial gastritis is obscure. Most writers concur in regarding spirit-drinking as one of the causes. It is most frequent in men and after forty years of age. It has, however, been met with in temperate persons of either sex and in early life. It is often preceded and accompanied by the symptoms of chronic simple gastritis, but that there is no essential connection between these processes is proven by the infrequency of cirrhosis of the stomach compared with the frequent occurrence of simple chronic gastritis. The prognosis is grave. The disease usually runs a chronic course and terminates by asthenia.

The TREATMENT embraces remedies to palliate irritation of the stomach, abstinence from spirits and other stimulants, careful regulation of diet, and, if there be pyloric obstruction, the means indicated in cases of dilatation of the stomach.

Gastro-Duodenitis—Duodenitis.

In acute gastritis the inflammation, as already stated, may extend into the duodenum. This is also true of subacute and chronic gastritis; but it would appear from the symptoms that subacute duodenitis may occur as a separate affection, not connected on the one hand with gastritis, or on the other hand with inflammation of the lower portion of the small intestine. The symptoms are—obscure pain, uneasiness or distress occurring some hours after the ingestion of food—that is, after it passes from the stomach into the duodenum—and tenderness below the epigastrium and over the site of the affected organ. Duodenitis, occurring either separately or in connection with gastritis and enteritis, is interesting and important as standing in a causative relation to jaundice. The jaundice is to be explained by obstruction either from swelling of the mucous membrane of the duodenum at the opening of the ductus choledochus or from an extension of the inflammation into the duct. A plug of mucus is frequently found in the ductus communis near its opening into the duodenum. The treatment is the same as that of subacute gastritis. The affection will again be referred to in the consideration of Jaundice.

It is a curious fact, first ascertained by Curling and confirmed by other observers, that a sloughing ulcer sometimes forms at the upper part of the duodenum within a few days after the occurrence of a severe burn of the skin. Perforation may occur under these circumstances. Ulceration of the duodenum, however, is by no means a constant effect of an extensive burn.

CHAPTER III.

STRUCTURAL DISEASES OF THE STOMACH.

Softening.—Gastric Ulcer.—Carcinoma of the Stomach.—Dilatation of the Stomach.—Degeneration of the Gastric Tubes.—Waxy Degeneration of the Stomach.

Softening of the Stomach—Gastromalacia.

SOFTENING of the mucous tunic of the stomach has been already mentioned as one of the anatomical characters of gastritis. The importance of not considering mere softening as an evidence of inflammation was then dwelt upon. At autopsies softening of the mucous membrane of the fundus is very often found. Softening may, however, be much more extensive. Not only may it involve the entire inner surface of the stomach, but it may extend in depth so as to affect the remaining coats of the stomach, and it may even lead to perforation of the stomach and the escape of its contents. Nor does the process always stop there. The adjoining organs may be attacked and undergo a similar softening. Thus the liver may be partially destroyed, or the diaphragm may be perforated and the contents of the stomach be found in one of the pleural cavities. There has been much controversy as to the nature of this softening, but it is now admitted that it is a post-mortem occurrence due to self-digestion. It has been rendered probable by the experiments of Pavy that the stomach is protected during life from self-digestion by the alkalinity of the blood circulating in its walls. The alkaline blood, which flows in an especially rich network of capillaries in the superficial layers of the mucous membrane, neutralizes the acid of any of the gastric juice which may penetrate the mucous membrane. With death the neutralizing influence of the circulating blood ceases. If the stomach now contain a sufficient quantity of gastric juice, and if the temperature be suitable, there is nothing to prevent the digestion of this organ and the extension of the digestive process to surrounding parts. Inasmuch as softening of the stomach, in the traditional acceptation of this term, is purely a post-mortem process, it cannot claim consideration in works the purpose of which is chiefly clinical. Fuller descriptions of this process are to be sought in books on pathological anatomy and on physiology.

Gastric Ulcer.

The form of ulcer to be here considered constitutes one of the most frequent and dangerous of the diseases of the stomach. It is to be distinguished from certain ulcerations which sometimes occur in connection with inflammation of the stomach—namely, the so-called catarrhal ulcers, follicular ulcers, and superficial hemorrhagic erosions. To these conditions, as well as to ulcerations produced by corrosive poisons, reference has already been made. Genuine tuberculous ulcers occur in the stomach, but they are very rare and require no further mention.

To the form of ulcer now under consideration various designations have been applied, such as simple (in distinction from cancerous), chronic (a term not always applicable), round, perforating, eroding, and digestive (ulcus ventriculi simplex, chronicum, rotundum, perforans, corrosivum, ex digestione). Simple ulcer of the stomach presents certain ANATOMICAL CHARACTERS which are met with elsewhere only in a similar form of ulcer in the lower part of the œsophagus and in the upper part of the small intestine.[1] The most marked of these peculiarities is that in its early stages the ulcer presents no evidences of inflammation in its base or its edges. The shape of the ulcer is round or oval, and sometimes, but rarely, irregular from the coalescence of two or more ulcers. The floor of the ulcer usually lies deeper than the mucous coat. From its tendency to extend in depth the ulcer is called perforating. The floor is not infrequently the muscular tunic; but it may be the serous coat, or, if the ulcer have perforated the walls of the stomach and adhesions have formed, the base of the ulcer may be occupied by some of the adjacent viscera, such as the pancreas or liver. As the ulcer extends in depth each successive tunic of the stomach is invaded in less extent than the preceding. The ulcer thus acquires a characteristic funnel-shape, the large opening being in the mucous membrane and the apex directed toward the peritoneum. The successive layers can sometimes be recognized as strata or steps in the wall of the ulcer. This funnel-shaped appearance is not always present. The edges are sometimes abrupt instead of sloping. If the ulcer be recent, its edges are smooth and clean-cut like those of a hole punched out of the mucous membrane. They may be infiltrated with blood, but they are not swollen by inflammatory exudation. The base may be smooth or soft and pulpy, and it may also have a hemorrhagic aspect. More frequently, old ulcers are found in which the edges are somewhat thickened and indurated by the growth of fibrous tissue. The floor in old ulcers is usually smooth, pale, and hard. Chronic gastritis is not infrequently present. The size of the ulcer varies much in different cases. The average size is perhaps that of a silver dime or quarter of a dollar. The ulcer may be very minute. In two cases reported by Murchison pore-like holes from one to two lines in diameter were found, which had penetrated one of the gastric arteries and produced fatal hemorrhage. A similar case has been reported by Reid.[2] On the other hand, the ulcer may be as large as the palm of the hand, and may extend along the lesser curvature from the pyloric to the cardiac orifice.[3] As a rule, only a single ulcer is found, but it is not a great rarity to find two or more ulcers (according to Brinton, in 21 per cent. of the cases). Still less infrequent is it to meet with the cicatrices of old ulcers. Sometimes ulcers are found opposite each other on corresponding parts of the stomach. The most frequent seat of gastric ulcer is in the pyloric region on the posterior surface and in the immediate neighborhood of the lesser curvature.[4] Ulcers of the anterior surface are much rarer, but are more dangerous, as will be explained presently. The microscopic examination of ulcers still progressing shows in their base and edges a granular substance which is the result of molecular disintegration of the surrounding tissue. It is by a process of molecular necrosis that the ulcer extends. In rare instances granulation-tissue may be present in the wall of the ulcer. In the ulcers with hard, thickened edges, these as well as the base are occupied by fibrous tissue containing fusiform and round cells; the blood-vessels in this tissue present thickened walls and diminished calibre; and they may contain thrombi. Newly-formed connective tissue may extend for some distance into the surrounding mucous membrane and compress the tubules, which may be thereby distorted and even obliterated. Simple ulcer may be complicated by cancer of the stomach. In fact, some writers believe that ulcer may even predispose to the development of cancer.

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[1] This is true, unless the rare variety of ulcer described by Clarke and by Klebs as ulcus corrodens uteri—corroding ulcer of the cervix uteri—be included in this category. (Consult Klebs, *Handbuch der Pathologischen Anatomie*, Bd. 1, S. 873.)

[2] *Buffalo Medical Journal*, 1846, vol. ii. p. 641.

[3] Cruveilhier has described an ulcer 6½ inches long and 3⅓ wide; Law, one measuring 6 by 3 inches.

[4] Of 793 ulcers, in 288 the ulcer occupied the lesser curvature; in 235, the posterior wall; in 95, the pylorus; in 69, the anterior wall; in 50, the cardia; in 29, the fundus; in 27, the greater curvature (article by Welch on "Gastric Ulcer," in *Pepper's System of Practical Medicine, by American Authors*, vol. ii.).

Ulcer of the stomach not infrequently heals by cicatrization; in fact, cicatrices are found oftener than open ulcers. The resulting cicatrix is usually a whitish stellate scar, attended with puckering of the adjacent mucous membrane. If the ulcer be large and deep, cicatrization may cause considerable, and even extreme, deformity of the stomach. Thus the stomach may be constricted so as to present an hour-glass shape. Stricture of the pylorus, with consequent dilatation of the stomach, may be caused by cicatrization of an ulcer; but instead of cicatrizing the ulcer may penetrate all of the coats of the stomach. When the ulcer is situated on the posterior surface of the stomach, as is usually the case, a localized peritonitis over the seat of the ulcer generally leads to the formation of adhesions with surrounding organs, so that when perforation is effected the floor of the ulcer is formed by some part adjacent to the stomach. Sometimes, however, the development and the progress of the ulcer are so rapid that time is not allowed for the formation of adhesions, and the contents of the stomach escape into the peritoneal cavity. It is a noteworthy fact that ulcers on the anterior surface, although the least frequent, are the most liable to perforate without protective adhesions. The explanation of this peculiar danger lies in the active movements of this part of the stomach and its unfavorable relations to surrounding parts. The pancreas most frequently forms the floor of ulcers situated on the posterior surface, but the stomach may also become agglutinated to the liver, the spleen, the diaphragm, lymph-glands, etc. The ulcer may continue to extend into the surrounding organs which form its base. The pancreas usually offers considerable resistance to the progress of the ulcer, but the liver is not infrequently invaded. The funnel-shaped appearance is not preserved in the organs secondarily involved. On the other hand, cavities of considerable size may be formed which communicate by a small opening with the stomach. The reactive inflammation in organs invaded by gastric ulcer leads to the formation of fibrous tissue, and sometimes, particularly in the liver, to abscesses. Fistulous communications of the stomach with adjacent parts may be formed. Thus the ulcer may open into the colon, the duodenum or some other part of the intestine, the pleural cavity, the lungs, or the gall-bladder. Gastro-cutaneous fistulæ may be produced, but they are very rare. The most frequent are gastro-colic fistulæ. If the ulcer open into the peritoneal cavity—and this accident may take place even after adhesions are formed—peritonitis ensues if immediate death do not follow. Death sometimes occurs suddenly from perforation of the stomach, and is attributed to paralysis of the heart from shock. The peritonitis following perforation is usually general. It is sometimes, however, localized, in consequence either of the slow escape of the gaseous and fluid contents of the stomach through a small opening or of the formation of previous adhesions. On p. 145 reference has been made to subphrenic abscesses containing air and simulating pneumo-pyothorax. The external opening in the stomach is usually round, and its edges are sharp and well defined in cases of perforation.

The hemorrhages which constitute one of the chief dangers to life in gastric ulcer may proceed from capillaries and small vessels in the walls or base of the ulcer, or they may come from erosion of a large artery or vein. In cases of profuse fatal hæmatemesis an eroded artery will generally be found in the floor of the ulcer. The ulcerated artery may be either one of the arteries of the stomach or an adjacent vessel, as the splenic. Gastric ulcer has been known to perforate the portal vein, and in more than one instance a simple or a suppurative pylephlebitis has been produced by this disease. In several cases small aneurisms have been found in the floor of the ulcer.

The *pathogenesis* of simple ulcer of the stomach has been a subject of much experimentation and discussion. The absence of inflammation, and the fact that this variety of ulcer is met with only in the lower part of the œsophagus, the stomach, and the upper part of the small intestine, early suggested the view that it is produced by the solvent action of the gastric juice; and this is the opinion now generally entertained. Hence the names peptic ulcer, digestive ulcer, and ulcus ex digestione are sometimes employed. In the preceding article was stated Pavy's view, which is widely accepted, that self-digestion of the stomach is prevented during life by the constant flow of alkaline blood in the rich capillary network of the gastric mucous membrane. If this view be adopted, it must be assumed that the primary cause of simple gastric ulcer is to be found in some local disturbance of the circulation in the gastric mucous membrane. As to this also most writers are agreed. As to the nature, however, of the change thus interfering with the circulation there is the widest diversity of opinion. An absolute basis of fact is lacking for nearly all the theories propounded to explain the diminished resistance of the mucous membrane to the action of the gastric juice. The anatomical investigation of the ulcer as a rule furnishes little or no clue to its primary cause. The following conditions have been assigned as causes of gastric ulcer by interfering with the circulation and consequently diminishing the alkalinity of circumscribed portions of the wall of the stomach: Embolism and thrombosis of branches of the gastric arteries; thrombosis of the corresponding veins; diseases of the coats of the same vessels, especially atheroma, fatty degeneration, and amyloid degeneration; spasm of the arteries, causing anæmia of the districts they supply; spasm of the muscular coat of the stomach, compressing the veins and leading to hemorrhages; obstructions in the portal circulation, inducing chronic congestion and hemorrhages from the gastric veins; hemorrhages into the wall of the stomach from rupture of the vessels incident to injuries, vomiting, ingestion of corrosive substances, increased blood-pressure, and aneurismal and varicose dilatations; inflammatory infiltration compressing the blood-vessels. Disturbed nervous influence has also been adduced as a cause.[1] Böttcher's view, that gastric ulcer is of mycotic origin and produced by the action of micrococci, has met with no confirmation. Finally, increased acidity of the gastric juice has been thought to be an element in the causation of digestive ulcer. It would lead too far to attempt a criticism of these different theories. The embolic origin is naturally suggested by the funnel shape of the ulcer corresponding to the wedge-shaped area of distribution of branches of the gastric arteries, but this theory is almost wholly devoid of anatomical support. No embolus in a gastric artery is found in ulcer of the stomach, and in the majority of cases there is no source for an embolus.[2] Gastric ulcer is by no means a frequent occurrence in disease attended by embolism in various parts of the body. If a thrombus be found in a vessel near a gastric ulcer, it is as proper to consider it the effect as the cause of the ulcer. The most plausible view, and the one which has the greatest number of supporters, is that a considerable number of ulcers of the stomach are produced by hemorrhagic infiltration of a circumscribed portion of the wall of the stomach. It is to be noted, as pointed out especially by Axel Key, that the hemorrhage must involve more than the superficial layer of the mucous membrane—must extend probably to the deeper and less vascular tissues. Superficial hemorrhages are followed by superficial erosions, the extension of which in depth is arrested by the great vascularity of the mucous membrane. The mode of production of hemorrhagic erosion is thus similar to that of simple ulcer. The causes of these deeper hemorrhages are doubtless various, and they include many of the causes of ulcer which have been enumerated. Perhaps spasm of the muscular fibres of the stomach attendant upon cardialgia and vomiting should be specially mentioned; but, while considering hemorrhage in the coats of the stomach as a probable, and perhaps frequent, cause of ulcer, it is not necessary to consider it the only cause, or to deny the efficacy of other causes which have been cited, the importance of which, with our present knowledge, is less evident. It is interesting that ulcers which may be produced in various ways artificially in the stomachs of animals readily heal. Anæmia, however, has been proven experimentally to be an obstacle to the process of repair. From this point of view the problem is quite as much to explain why ulcer of the stomach does not heal as it is to explain how it is produced. It is probable that various constitutional conditions, and also diseases of the blood-vessels in the walls of the ulcer, impede the process of repair.

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[1] In support of this view have been cited the experiment of Frerichs, who found an ulcer of the stomach in a cat after division of the splanchnic nerves and of the cœliac axis, and the experiments of Ebstein, who by injuries to the brain and cord in animals produced ulcers upon a hemorrhagic base. A not very appropriate comparison has also been made between gastric ulcer and ulcer of the cornea after section of the trigeminus.

[2] In a case presented by Janeway to the New York Pathological Society in 1871 a fibrinous plug was found in a branch of the gastro-epiploic artery leading to an ulcer. No source for an embolus could be found.

Gastric ulcer is not an infrequent affection. Open gastric ulcer is found in from 1 to 2 per cent. of persons dying from all causes. Cicatrices are found about three times as frequently as open ulcers. Statistics appear to show a considerable variation in its frequency in different geographical situations. In this country gastric ulcer, although not rare, is less frequent than in many parts of Europe.

The CLINICAL HISTORY embraces as the most prominent symptoms pain, tenderness on pressure, vomiting, and hemorrhage from the stomach. These are the important symptoms in a diagnostic point of view. Pain is rarely wanting. It is burning or gnawing in character, coming on directly or soon after the ingestion of food, and continuing until the contents of the stomach have either passed into the duodenum or been ejected by vomiting. The quickness with which the pain follows the ingestion of food is a diagnostic point. If pain be due to simple indigestion or neuralgia, it generally occurs after a greater or less interval from the time of taking food. It is situated at or near the epigastrium, and is generally confined to a small space. A similar gnawing pain in the back, between the shoulders, is not uncommon. The probable cause of the pain is irritation of the ulcerated space by the contact of the ingesta and the gastric juice. The pain from this cause is especially marked after the ingestion of stimulating articles of food or drink, and when, from indigestion, the contents of the stomach undergo chemical changes. Pain may be caused also by circumscribed peritonitis and by the traction of adhesions which have resulted from peritoneal inflammation. The latter causes may account for the exceptional occurrence of pain irrespective of the ingestion of food. The pain caused by the action of the gastric juice is felt directly after eating, because the secretion of the juice is excited by the presence of food in the stomach. The pain is sometimes absent for several consecutive days. It is affected by the position of the body, being in some cases increased by lying on the back and relieved by bending forward; in other cases the reverse is observed. The explanation is afforded by the situation of the ulcer on the posterior or the anterior wall of the stomach. As in the majority of cases the ulcer is on the posterior wall, relief is oftener obtained by bending forward than by recumbency on the back. Tenderness on pressure over the epigastrium is almost invariably present, confined, like the pain, within a small area, and varying considerably in degree in different cases. Vomiting is a very frequent but not a constant symptom, occurring after a period from the time of taking food varying in different cases, the act of vomiting usually not violent but complete, and the ejection of the contents of the stomach followed by notable relief of pain and distress. Mucus may be expelled in more or less abundance with the ingesta. As respects vomiting, much will depend on the quantity and kind of food ingested. Indigestible and stimulating articles are more likely to provoke it, as well as to increase pain, than those which are bland and easy of digestion. Hot substances are not as well borne as cold. Hemorrhage, as shown by the vomiting of blood in greater or less quantity, is a symptom of frequent occurrence. The vomiting of blood generally occurs after a meal. The hemorrhage is sometimes so abundant that life is lost from this cause. Cases have been reported in which life was lost from hemorrhage, although no blood was vomited, the stomach being found, after death, filled with clots. The blood vomited is dark or grumous from the change of hæmoglobin to hæmatin by the action of the gastric juice, provided the vomiting do not immediately follow the hemorrhage. If vomiting quickly follow a profuse hemorrhage, the blood may retain its alkaline reaction and its arterial color. As a rule, simple inspection of the vomited matter suffices to determine the presence or the absence of blood. In cases of doubt resort may be had to microscopic or spectroscopic examination. Blood-corpuscles, usually much shrivelled, can generally be recognized by the microscope. Sometimes, however, they are so disorganized by the action of the gastric juice that their detection by this instrument is difficult or impossible. In this case the spectroscope will usually afford the desired information. The stools also should be examined with reference to the gross and microscopical appearances of blood, since hemorrhage may take place without vomiting, the blood passing into the intestinal canal.

The bowels in cases of gastric ulcer are generally constipated, a symptom easily understood in the cases in which vomiting is a prominent symptom. Amenorrhœa is common. There is, however, little foundation for the idea that hemorrhage from the ulcer occurs vicariously in the place of menstruation, but the latter ceases or is deficient, as in cases of anæmia produced by hemorrhages elsewhere or from other causes.

The perforating ulcer occurs oftenest in young girls, and particularly in young maid-servants. Statistics show that females are more liable to ulcer, the proportion being 2 males to 3 females. In females the ulcer most frequently develops between twenty and thirty, and in males between thirty and forty, years of age. Clinical observation fails to show any dependence on particular habits of diet or other causes, nor has it any known relationship with other affections. It may be stated, however, that persons of feeble or impaired constitution are more liable to it than the robust.

The DIAGNOSIS of gastric ulcer may be made with much certainty, provided the diagnostic symptoms be present—namely, pain of a burning or gnawing character limited to a circumscribed space in the epigastrium, with tenderness, also circumscribed, the pain felt directly after the ingestion of food, and especially after certain kinds of food, vomiting occurring during the process of stomach digestion, and followed by relief, and, finally, gastrorrhagia or hæmatemesis. Analogous symptoms, it is true, may be present in cases of cancer of the stomach, but there are points of contrast involved in this differential diagnosis which will be noticed presently in considering cancer. Moreover, the absence of a tumor appreciable through the abdominal walls goes far toward the exclusion of cancer. Chronic gastritis and ulcer have symptoms to some extent in common; but in chronic gastritis pain is less prominent, vomiting does not occur so frequently, and hemorrhage is wanting. In cases of gastralgia the pain might suggest the idea of ulcer; but the pain in gastralgia is not aggravated, and, on the contrary, is often relieved, by the ingestion of food; tenderness over the epigastrium is not present, but the pain is often relieved by pressure; vomiting is an occasional symptom only; hæmatemesis does not occur. Attacks of gastralgia and gastric ulcer are, however, sometimes associated. In cases of merely functional disorder pain, vomiting, and hæmatemesis are all wanting. Hæmatemesis, it is to be borne in mind, is not alone evidence of ulcer. It occurs, irrespective of disease of the stomach, in certain cases of cirrhosis of the liver. It is the union of the several symptoms just named that renders the diagnosis complete. These diagnostic symptoms, however, are not united in all cases of gastric ulcer. Hemorrhage is not invariably present; vomiting is not always a prominent symptom, and may be wanting; pain and tenderness are not in all cases marked: the affection, in short, is sometimes latent, more or less difficulty or disturbance of digestion only being manifested. Under these circumstances a positive diagnosis cannot be made. A strong suspicion of the existence of the affection may in some cases be reasonably entertained when the symptoms do not warrant a positive diagnosis.

Gastric ulcer proves fatal in different modes. One of these is by perforation, diffuse peritonitis being induced by the escape of the contents of the stomach. Perforation into the general peritoneal cavity occurs in about 6½ per cent. of all cases of gastric ulcer. In the great majority of cases diffuse peritonitis caused by perforation of the stomach proves fatal. Recovery, however, is within the range of possibility. This accident would take place oftener than it does were it not for the adhesion of the stomach to the adjacent organs as a result of circumscribed peritonitis. Perforation is most likely to occur when the ulcer is situated on the anterior surface of the stomach. The perforation generally takes place after the ingestion of a meal, or in the act of straining or sneezing, or in some sudden and forcible movement of the body. In some cases peritonitis, following perforation, occurs without having been preceded by any symptoms pointing to the existence of ulceration of the stomach. The following case is illustrative of the statement just made: A well-nourished woman of about thirty-five years of age had complained only of indigestion, when suddenly there occurred severe abdominal pain, followed by speedy collapse and death. Poisoning was suspected, and the autopsy was made by direction of the coroner. A round, funnel-shaped ulcer of the size of a quarter of a dollar was found on the anterior wall of the stomach. Perforation had taken place and given rise to acute peritonitis. As has been stated, peritoneal adhesions having previously taken place, perforation may open a communication with the transverse colon. The evidence of this is stercoraceous vomiting.

Another mode in which it proves fatal is by hemorrhage. Life may be slowly destroyed by the continued escape of blood or by repeated attacks of hemorrhage, or the loss of blood may be so rapid as to destroy life quickly. Death from hemorrhage occurs in from 3 to 5 per cent. of all cases of gastric ulcer.

Another mode in which it proves fatal is by inanition. Death takes place by slow asthenia when the stomach is so intolerant of ingesta that aliment sufficient for the support of life is not retained. In other words, patients are gradually starved to death, provided life be not cut off by some other disease which the system may be rendered unable to resist in consequence of the debility incident to the stomach affection.

The duration is very variable. Death sometimes takes place from perforation or hemorrhage within a few weeks, or even a few days, after the time when the symptoms first denoted gastric trouble; or, as already stated, perforation and hemorrhage may occur when previous symptoms denoting gastric ulcer were wanting. But if these accidents do not prove the immediate cause of death, life is usually prolonged for many months, and it may be for many years. Brinton states that he has notes of one case in which probably a continuous open ulcer existed for thirty-five years, and of two cases in which the duration was thirty years, together with three or four of twenty, four or five of fifteen, and several of ten, seven, five, and four years' duration. It is not uncommon for the symptoms from time to time to subside or to disappear, and subsequently to return. These remissions are probably due to healing of the ulcer, which afterward again opens; in other words, relapses are liable to occur.

Gastric ulcer, although a serious affection, is by no means incurable. The mortality is estimated by Welch as about 15 per cent. In consequence of the various disturbances resulting from cicatricial contraction of an ulcer, gastric symptoms may remain after the ulcer has healed in the anatomical sense.

The TREATMENT of gastric ulcer embraces the following objects: *First*, and most important, securing for the stomach absolute rest; and *second*, the arrest of hemorrhage and the palliation of pain. Rest of the stomach is secured most effectually by resorting to rectal alimentation. This is advisable whenever symptoms render the diagnosis of gastric ulcer probable. It is now well established that not only life, but complete satisfaction of the sense of hunger and fair nutrition, may be maintained indefinitely by the introduction of food into the rectum. Indeed, an increase of weight under an exclusively rectal diet has been observed. I can bear testimony to the prompt success of this method of treatment. The kinds of food to be employed and the practical rules to be observed are the same as in the treatment of acute gastritis. (Vide p. 410.) When the stomach tolerates food—that is, when pain and vomiting are no longer caused by ingesta *per os*—the diet should consist for a time of milk, other articles being cautiously added or substituted. Considering the frequency of relapses, care to adjust the diet to the digestive power is advisable for a considerable period.

Hemorrhage is to be arrested by small pieces of ice swallowed, and by the application of cold to the epigastrium if the loss of blood be considerable. The fluid extract of ergot may be given *per enema*. Pain may require the use of opiates, the form being selected which is found on trial to be the least objectionable. They may be administered either by the rectum or hypodermically. Pain and vomiting cease generally to be prominent symptoms if complete rest of the stomach be secured by rectal alimentation, so that the laudanum added to the nutritive injections suffices.

It is doubtful whether any remedies exert a direct effect to promote cicatrization. The nitrate of silver and other remedies have been given with the expectation that they will exert a topical effect upon the ulcer. Clinical observation appears not to furnish evidence of their utility in this way. Cicatrization is promoted indirectly by remedies which arrest the peristaltic movements of the stomach and quiet the irritability of the organ; in other words, remedies which have reference to the first object of treatment—namely, securing rest.

Carcinoma of the Stomach.

Most of the tumors met with in other parts of the body may also occur in the wall of the stomach—fibroma, lipoma, sarcoma, myoma, gumma, tubercle, papilloma, adenoma, and carcinoma. Of these, only carcinoma possesses clinical importance. (For general considerations concerning the origin and structure of cancer, the reader is referred to Part I. p. 45.)

Next to the uterus, the stomach is the most frequent seat of primary carcinoma.[1] Secondary cancer has also been met with in this organ, but it is rare. The forms of carcinoma of the stomach are scirrhous (fibrous), medullary (encephaloid), colloid (alveolar), and cylindrical-celled epithelioma. Of these, colloid is by far the least frequent. As the distinction between scirrhous and medullary cancer is based upon the difference in consistence, the former being hard and the latter soft, and as all degrees of combination and of transition exist between the two, statistics as to the frequency of the one or of the other are only of limited value. Combinations of hard and of soft cancer and tumors of medium consistence are more frequent than either the very hard or the very soft varieties. Cylindrical-celled epithelioma is a common form of gastric carcinoma—according to some authors, the most common. The most frequent seat of cancer is the pyloric region. The posterior surface and the lesser curvature are more frequently affected than the opposite regions. The cardia is affected in about 8 per cent. of all cases.[2] Scirrhous cancer is characterized by the large amount of fibrous stroma and comparatively small development of cancerous alveoli. In the great majority of cases it is situated near the pylorus, where it sometimes appears as a partial or complete ring constricting the pyloric orifice. Scirrhus may appear either infiltrated or in the form of distinct nodules. In medullary cancer the spaces filled with cancer-cells predominate over the stroma, which is usually richly infiltrated with round cells. This variety grows more rapidly and is more likely to be attended by metastases than the preceding. It forms a soft tumor, readily ulcerating, and sometimes presenting fungous or villous excrescences upon its surface (so-called villous cancer). Colloid cancer is rare. It may be combined in a nodular form with one of the other varieties, or it may appear as a diffuse thickening of the walls of the stomach, involving apparently its deeper coats. In the latter case the peritoneum, as a rule, is extensively affected by the same process. This species of cancer is readily recognized by the clear, gelatinous substance found in its large alveoli. In cylindrical-celled epithelioma the alveolar structure conforms more closely than in the other varieties of cancer to the type of the normal gastric tubules. The cells contained in the alveoli, more particularly those which line the walls of the alveoli, are cylindrical in shape. In consistence this variety resembles medullary cancer.

The opinion was formerly held that cancer of the stomach develops primarily in the submucous tissue and invades secondarily the other coats of the stomach. Since Waldeyer's view as to the epithelial origin of cancer-cells has been so widely accepted, it has been the teaching of many writers that carcinoma develops primarily in the mucous membrane. The process, as described by Waldeyer, is briefly as follows: A group of tubules (from ten to twenty) enlarge, lengthen, and give off offshoots. They are filled with cells resembling usually the adelomorphous variety, but more granular and more deeply staining. The growth of the tubules with their branching offshoots is downward through the muscularis mucosæ. When the lax submucosa is reached the progress is more rapid. Groups of cancer-cells now push their way in all directions, chiefly in the lymphatic vessels and spaces. Cancerous masses penetrate between the bundles of smooth muscular tissue, and often extend to the subserous and serous coats. The growth may be so much more extensive in the submucous coat that the tumor appears to originate there, but according to Waldeyer careful microscopical examination will always disclose some band of connection with the tubules of the mucous membrane. The growth, however, may from the first involve chiefly the mucous coat, and in any case this is, as a rule, sooner or later extensively invaded.

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[1] Of 31,482 primary cases of cancers collected by Welch, the stomach was the seat of the cancer in 21.4 per cent., and the uterus, in 29.5 per cent. (*Pepper's System of Practical Medicine, by American Authors*, vol. ii. p. 533).

[2] Welch's analysis of 1300 cases of cancer of the stomach gives—pyloric region, 60.8 per cent.; lesser curvature, 11.4; cardia, 8; posterior wall, 5.2; the whole or greater part of stomach, 4.7; multiple tumors, 3.5; greater curvature, 2.6; anterior wall, 2.3; and fundus, 1.5 per cent.

It has already been mentioned that cancer of the stomach may appear in a diffuse or in a nodular form, also as a ring, especially about the pylorus. Ulceration is a frequent occurrence. This is the rule with medullary and cylindrical epithelial, and is not infrequent with scirrhous cancer. There is less tendency to ulceration in colloid cancer than in the other varieties. Ulceration is not always to be regarded as an unfavorable occurrence. The soft cancers especially may be much reduced in size by this destructive process. This reduction may relieve an existing stenosis of the pyloric or cardiac orifice. The shape of the ulcer, particularly when the cancer is medullary, has been often compared to that of a crater—a peripheral wall of cancerous tissue surrounding a central loss of substance. Exceptionally, in cases of scirrhus the ulcer may bear much resemblance to simple or round ulcer with indurated walls; and this fact is to be considered in weighing the statements of those who report many cases of cancer developing from round ulcer.[1] Scirrhous cancer often contains considerable cicatricial tissue, but there is no positive reason to believe that it ever heals. The muscular coat in the neighborhood of cancerous growths is almost always hypertrophied. If the tumor produce stenosis of the pylorus, the rest of the organ is dilated; if the cardiac orifice be constricted, the stomach is reduced in size. Chronic gastritis is a frequent attendant upon cancer. Chronic circumscribed peritonitis, with the formation of adhesions, is common in the neighborhood of the cancer. The wall of the stomach may be perforated by cancerous ulceration leading to invasion of the surrounding organs, to perforative peritonitis, or to fistulous communications. Peritonitis from perforation is less frequent with cancerous than with simple ulcer.[2] The erosion of large vessels is also less frequent in cancer than in ulcer. General peritonitis may occur without perforation.

Carcinoma may extend by continuous growths into surrounding parts. It may also lead to metastases or secondary deposits. The lymphatic glands near the stomach, also the liver, the peritoneum, the pancreas, and the lungs, are affected in frequency in the order named. Secondary deposits in the liver are those most frequently detected during life.[3] Metastases occur most frequently through the lymph-vessels, but they may also occur through the blood-current. An actual growth of carcinoma into the rootlets and main trunk of the portal vein has been observed. The secondary deposits generally conform in structure with the primary growth. It is worthy of note that large and abundant secondary deposits may attend small and insignificant tumors of the stomach which during life may have produced no symptoms, and after death may be overlooked on superficial examination. The blood in cases of cancer of the stomach may present the same alterations as in pernicious anæmia.

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[1] Thus Lebert states that in 9 per cent. of the cases cancer develops from simple ulcer (*Deutsches Archiv für klinische Medicin*, 1877, Bd. 19, S. 545).

[2] Perforation into the peritoneal cavity occurs, according to Brinton, in a little more than 4 per cent. of the cases; according to Lange, in a little more than 5 per cent.

[3] In Welch's statistics (1574 cases) the lymphatic glands were the seat of metastases in 35 per cent., the liver in 30, the peritoneum or intestine in 23, the pancreas in 8, the pleura and lung in 6, and the spleen in 2 per cent. of the cases.

The SYMPTOMS which make up the clinical history of this affection are as follows: Impaired appetite, or anorexia, is generally more or less marked, but in some cases the desire for food is preserved. Pain may be more or less prominent, and the character of the pain may be lancinating. In many cases, however, the lancinating pain which attends cancer in other situations is either slight or wanting. A circumscribed gnawing pain may be incident to the ulcer occurring as secondary to the cancer. Tenderness over the epigastrium may or may not exist. Vomiting is a frequent symptom, but it occurs less frequently than in cases of simple ulcer, and at a later period after taking food. The vomiting, if the cancer be at the pylorus, may be due chiefly to obstruction at this orifice and consequent accumulation of gastric contents. Yeast-fungi, sarcinæ, and torulæ may be found in the vomited matters when these have undergone fermentation within the stomach. Blood is not infrequently found in the vomited matters, generally in small quantity and having the coffee-grounds appearance. With these symptoms referable to the stomach are associated progressive loss of weight and strength, anæmia, usually constipation, but occasionally diarrhœa, and in some cases jaundice. The last-named symptom points to the probable occurrence of secondary cancer affecting the liver.

Certain general considerations pertaining to the causation of tumors have been alluded to in Part I. p. 41. As to the primary cause of cancer, we are without any positive information. It is probable that the cachexia is secondary to the development of the cancer. The cachexia may be explained partly by certain facts pertaining to cancer in general, and in part by the local injury which the growth inflicts upon an organ of such vital importance as the stomach. The general considerations are—the rapid growth of the tumor; its withdrawal in a short time from the circulation of a large quantity of nutriment employed only for its own noxious growth and for no useful purpose; its destruction of healthy and useful tissues; the production of metastases, which for their nutrition and growth, from their number and size, make even greater claims upon the organism than the primary tumor; and often the occurrence of exhausting hemorrhages. The local effects of cancer of the stomach—loss of appetite, pain, vomiting, mechanical disturbance of the digestive functions, and chemical alterations in the gastric fluids—are often in themselves sufficient to explain an extreme degree of cachexia. While these general and local effects suffice to explain the cachexia in most cases of carcinoma of the stomach, there are a small number of cases in which they do not seem to be operative, and in which it seems necessary to assume the entrance into the blood of some virus produced in the tumor.

The liability to cancer of the stomach is slight before the age of forty. Three-fourths of all cases occur between forty and seventy years of age; the largest number being between fifty and sixty. Men are somewhat more frequently affected than women, but the disproportion is not great.[1] Heredity is a well-established element in the causation of cancer of the stomach as of cancer elsewhere.

Cancer of the stomach is to be discriminated from merely functional disorder or dyspepsia, chronic gastritis, and gastric ulcer. In merely functional disorder, pain and vomiting are symptoms rarely as prominent as they are generally in cancer. The ailments in dyspepsia are seldom so protracted; they do not lead to the same amount of emaciation, feebleness, and pallor; hæmatemesis is wanting. The same is true of chronic gastritis. Persistent disturbance

[1] In 2214 cases of gastric cancer there was a ratio of 5 males to 4 females (Welch).

of digestion, especially if accompanied by pain and vomiting, leading to considerable loss in weight, diminution of strength, and anæmia in a person between forty and seventy, should always excite suspicion of cancer. Cases of gastralgia lack all the diagnostic features of cancer except pain. Gastric ulcer is liable to be confounded with cancer, and in view of the great difference as regards prognosis it is desirable to discriminate between these affections. They present certain points in contrast as regards the symptoms. In cancer the appetite is more impaired than in ulcer. Vomiting is more uniformly present in ulcer; it occurs earlier after the ingestion of food, and in general is evidently due to the irritation caused by the presence of food. In cancer it is present in only a certain proportion of cases; it does not follow so soon after taking food, and is frequently due to the mechanical distension of the stomach owing to the pyloric obstruction. Hemorrhage is more constant and more profuse in ulcer. If the patient be a young girl, the chances are much in favor of ulcer. The duration of the local symptoms is of importance in this differential diagnosis. If the symptoms have existed for a long period, the chances are in favor of ulcer rather than of cancer. Age is to be considered in the diagnosis, cancer occuring rarely before forty. Enlargement of lymphatic glands above the clavicles accompanies cancer in some cases and has some weight in the diagnosis.

Considerable importance has been attached to the complexion which has been supposed to denote the cancerous cachexia—namely, pallor with a faint tint of yellow or green, the conjunctiva remaining unaffected. It has been called a waxy complexion. This is sometimes well marked in cases of cancer, but it is by no means always present, nor is it pathognomonic when marked. If members of the family have had cancer, this fact is entitled to some weight in the diagnosis. So also is the coexistence of cancer in some other situation.

Considerable interest attaches to the discovery by Von den Velden that hydrochloric acid is habitually absent from the stomach in cases of gastric dilatation due to cancer. This diagnostic point will be considered in the following article.

In some cases of cancer there are few or no symptoms denoting any serious affection up to a short time before death. Like gastric ulcer, it is sometimes remarkably latent, the patient seeming to suffer only from moderate or slight dyspeptic ailments, the appetite remaining unimpaired, the strength not failing, and the aspect indicating health. Several cases illustrative of this fact have fallen under my observation. In these latent cases, and also when the differential diagnosis relates to ulcer and chronic gastritis, great importance belongs to the discovery or otherwise of an abdominal tumor in the site of the stomach. Exclusive of the cases in which the cardiac orifice is the seat of the cancer, a tumor is discoverable by physical examination in the majority of cases. According to Brinton, it exists in 80 per cent. of all cases of cancer. The tumor is situated generally between the false ribs and the umbilicus, a little to the right of the median line. It may be movable or fixed; much more frequently the latter. If movable, it will be likely to change its situation accordingly as the stomach is distended or empty. The tumor varies in size from a pigeon's egg to the size of the closed hand. The weight of the tumor may cause it to fall below the epigastrium, and it may descend nearly to the symphysis pubis. It is usually resisting and the surface is irregular or nodulated.

The presence of a tumor, if ascertained to be seated in the stomach, renders the diagnosis almost positive; but it is not always easy to decide positively that the tumor is seated in the stomach. A cancerous tumor may simulate aneurism of the abdominal aorta. The characters of an aneurismal tumor are expansile pulsation, thrill, and murmur. A cancerous tumor may present, in a more or less marked degree, the same characters. It may present not only a single but a double murmur. How are the two to be distinguished from each other? An aneurismal tumor has usually a smooth outline and may be diminished by pressure; the pulsation is felt laterally as well as on its anterior surface; by a change of position of the patient, as placing him on his hands and knees, the pulsation is not materially affected, whereas if the tumor be not aneurismal its pressure on the artery is taken off and the pulsation may be found to cease. But aneurismal tumors do not always pulsate, and both thrill and murmur may be wanting. Under these circumstances in this differential diagnosis reliance must be had upon the diagnostic points relating to the history and symptoms. These are, of course, always to be carefully considered. Other tumors, from their situation, are liable to be confounded with cancer of the stomach—namely, tumors connected with the left lobe of the liver and with the pancreas, fecal tumors, etc. The distinctive characters of these will be considered in other connections. A point of distinction, as contrasted with tumors seated in the liver, is that the latter are depressed in an act of deep inspiration, whereas the descent of the diaphragm does not affect the situation if the tumor be seated in the stomach. Moreover, percussion over an enlargement of the left lobe of the liver gives flatness, whereas a tympanitic resonance is elicited usually over a gastric tumor. There is a liability to mistake the normal pancreas for a tumor; and owing to different degrees of distension of the stomach the tumor may be felt at some times and not at other times.

Cancerous affection of the cardiac orifice, leading to stenosis, gives rise to symptoms quite different from those which accompany cancer of the stomach elsewhere situated. Food accumulates in the lower part of the œsophagus and is regurgitated directly after deglutition. The obstruction, if considerable, interferes with vomiting as well as with the ingestion of food. The food is regurgitated without change, and is devoid of the taste and odor of the gastric fluids. The patient is unable to take solid food, and is compelled to take liquids slowly and in small mouthfuls. In these cases a tumor is not discoverable. The symptoms are the same as in cases of stricture at the lower portion of the œsophagus from other than cancerous disease, and the differential diagnosis cannot always be made with positiveness. Spasmodic stricture of the œsophagus may simulate contraction from either cancer or some other organic affection. The passage of a probang into the stomach will enable the practitioner to exclude the latter. Adhering to the probang or sound passed into the cardiac orifice, may be cancerous particles, which establish the diagnosis.

A microscopical examination of the matters vomited in cases of suspected cancer may lead to the discovery of cancer elements. This test is available only at an advanced period of the disease, and then only in a very small proportion of cases.

The PROGNOSIS in cases of cancer of the stomach is as unfavorable as possible. If a cure be within the range of possibility, the probabilities are so vastly against it that we are not warranted in entertaining the least expectation of such a result in any individual case. A fatal termination is merely a question of time. Statistics show the average duration to be about a year from the date of the first gastric symptoms. The maximum duration is about three years, and the shortest period is a month. Death occurs by slow asthenia, except in rare instances when it follows profuse hemorrhage or perforation of the stomach. Œdema of the lower limbs and ascites occur in some cases. The former may be caused by thrombosis of the femoral veins; the latter may be produced by pressure upon the portal vein of a cancerous nodule connected with the liver or the formation of a thrombus within the vein.

The TREATMENT has reference only to the prolongation of life and the palliation of symptoms. A diet bland and nutritious, consisting of the articles of food which experimental trials in each case show to be best borne, taken in quantities and at intervals to be determined also by trial; remedies to relieve pain and quiet the irritability of the stomach; avoidance of cathartics and any measures which impair the vital powers,—these are the points to be kept in view in the management of this distressing and hopeless affection. Nourishment by means of rectal alimentation in cancer of the stomach involving pyloric obstruction prevents pain and vomiting resulting from the accumulation of ingesta and the chemical changes incidental thereto. It may be resorted to as supplementary to a limited quantity of food taken into the stomach. Withdrawal from time to time of the accumulated contents of the stomach by means of the pump or siphon, in the manner to be described in connection with Dilatation of the Stomach, has been found to afford marked relief. In cases of cancer causing obstruction of the cardiac orifice the sensation of hunger is removed and life prolonged by resorting to rectal alimentation. These desirable results were manifested in a case under my observation. Buttermilk is sometimes better tolerated in cases of cancer than any other article of diet. Cases have been reported in which the condurango appeared to have a beneficial effect.

Since the preceding edition of this work there have been more than 40 published resections of cancer of the pylorus. Of these, about 30 have died from the immediate effects of the operation. In no case does life seem to have been prolonged more than two years after the operation. Only a very small proportion of the cases of gastric cancer can be considered as suitable for operation. The possibility of successful surgical interference renders it very important to make an early diagnosis. In the hands of a skilful surgeon an exploratory laparotomy is attended with little or no danger.

Dilatation of the Stomach—Gastrectasia.

Since Kussmaul's publication,[1] in 1869, concerning the treatment of dilatation of the stomach by means of the stomach-pump increased attention has been devoted to the etiology, symptomatology, and treatment of this affection. As the normal dimensions of the stomach vary in different individuals within wide limits, it is manifestly impossible to give an exact anatomical definition of this disease. While well-marked examples cannot be mistaken in post-mortem examination, it is often impossible to recognize the lesser grades of this disease. During life certain characteristic disturbances of the physiological functions of the stomach are of great importance in reaching a diagnosis—sometimes of greater importance than the anatomical investigation.

When dilatation of the stomach is excessive the organ may occupy the greater part of the abdominal cavity, and may even descend into the pelvis. In less extreme cases the lowest part of the stomach lies somewhere between the umbilicus and the symphysis pubis. By increased weight the whole organ is generally dragged downward, especially the pyloric portion when it is movable, so that the position of the stomach is more nearly vertical than normal. When dilatation is due to stenosis of the pylorus the muscular coat is almost invariably hypertrophied. In the so-called atonic dilatation to be described presently the muscularis may be of normal or of reduced thickness. In one case fatty and colloid degeneration of the muscular fibres was found (Kussmaul and Maier). The mucous membrane is sometimes thickened and sometimes thinned. It is readily understood that neighboring organs may be more or less displaced by a widely-distended stomach.

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[1] *Deutsches Archiv f. klin. Med.*, Bd. 6, p. 455.

It is important to know that the stomach may be situated abnormally low in the abdomen without being dilated. Thus if, during life, the lower part of the stomach be found a hand's breadth below the umbilicus, it does not necessarily follow that it is dilated. The so-called vertical position (situs verticalis) of the stomach is a not infrequent malposition which simulates dilatation. In this condition the pyloric orifice is lower than normal, and it may be in nearly a vertical line with the cardia. The position may be a preservation of the fœtal state, or it may be acquired as a result of pressure (in women especially by tight lacing). As pointed out by Kussmaul, the greater curvature may occupy an abnormally low position in consequence of the so-called loop shape (Schlingen-form) of the stomach. In this state the pylorus is nearer the cardia than normal, and the lesser curvature is abnormally sharp.

Kussmaul distinguishes two forms of dilatation of the stomach, the hypertrophic and the atonic.[1] In the former the muscular coat is hypertrophied, and the cause in the great majority of cases is obstruction at or near the pyloric orifice. In the atonic form the muscular coat is relaxed, and after death it is found but little if at all hypertrophied, and it may be atrophied. Hypertrophic dilatation he compares, not inappropriately, to eccentric hypertrophy of the heart.

The causes of dilatation relate, in the first place, to mechanical obstacles to the passage of the contents of the stomach into the intestine. The obstacles may be various. They may be seated in the upper part of the duodenum as well as in the stomach. The most frequent and important are cancer and the cicatrices resulting from old ulcers. Other causes of organic stenosis of the pylorus are—tumors of neighboring organs, causing external pressure, particularly cancer of the liver and of the pancreas; the contraction of cicatricial tissue resulting from perigastritis and the inflammation of connective tissue near the stomach; hypertrophic stenosis (see p. 418); the pressure upon the duodenum of a wandering right kidney; and, as rarities, fibroma, lipoma, glandular and cystic polypi of the pylorus. A very rare condition is congenital stenosis of the pylorus.[2] Dilatation has been referred to traction upon the stomach produced by large scrotal hernias. According to Kussmaul, it may be referable to spastic stenosis of the pylorus. The sphincter pylori may be thrown into spasmodic contraction by painful erosions, ulcers, and inflammations in its neighborhood. Destruction of a part of the muscular coat near the pylorus is evidently an obstacle to the propulsion of food through this orifice, and so may lead to dilatation. In this way ulcers and cancers which destroy the muscularis may produce dilatation even when they do not obstruct the lumen of the stomach. It has already been mentioned that cirrhosis of the stomach, which is usually attended by contraction, may exceptionally be accompanied by dilatation.

Atonic dilatation may be due to weakness of the muscular tone of the stomach from imperfect nutrition in cases of anæmia, chlorosis, exhausting acute and chronic diseases, or amyloid degeneration of the gastric arteries. Important causes of dilatation of the stomach are chronic gastritis and dyspepsia, especially when attended by stagnation and fermentation of the gastric contents. Individuals of sedentary habits who eat and drink a great deal are liable to dilatation. The polyphagia which attends diabetes thus becomes a cause of dilatation. The dilatation which results from organic stenosis of the pylorus reaches a more extreme degree than that from other causes, and is accompanied by a greater hypertrophy of the muscular tunic.

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[1] Kussmaul, "Die peristaltische Unruhe des Magens," *Volkmann's Sammlung klinischer Vorträge*, Leipzig, 1880.

[2] Landerer, *Ueber angeborene Stenose des Pylorus*, Tübingen, 1879.

Vomiting occurs almost without exception when dilatation is due to pyloric obstruction; and in other cases it is a frequent but not a constant symptom. The characteristic features as regards this symptom are the occurrence of the vomiting after periods varying from a day to several days, the large quantity of the matters vomited, amounting sometimes to several quarts, and the presence of undigested aliment which had been taken the previous day or several days previously. The matters vomited are almost invariably acid, and frequently they are in a condition of fermentation, as indicated by a froth containing gas, sometimes inflammable, in the upper layers. Sarcinæ, the yeast-fungus, bacteria, and crystals of fatty acids are often found. Acetic, lactic, butyric, and other organic acids are usually present. Von den Velden has discovered an interesting fact in regard to the presence of hydrochloric acid, the normal acid of the gastric juice. He failed to find this acid in any case of dilatation due to cancer of the stomach. In dilatation from other causes it was often, but not invariably, present. According to this author, if hydrochloric acid be repeatedly detected in the contents of the stomach obtained by means of the stomach-pump, obstruction from cancer may be excluded. If this acid be persistently absent, no positive conclusions can be drawn.[1] Bile is not often found in the matters vomited or withdrawn by the stomach-pump if the dilatation be due to stenosis of the pyloric orifice.

The feature pertaining to the contents of the stomach which appears to be most diagnostic of dilatation is their retention for a day or for several days within this organ. This may be ascertained by washing out the stomach in the morning before food has been taken. In healthy persons the stomach does not contain in the morning any of the ingesta of the preceding day. The ingesta may remain overnight in cases of temporary indigestion, but if they are habitually present in the morning, it is certain that the power of the stomach to propel its contents into the duodenum is wanting. This condition leads to dilatation. It is called by Rosenbach insufficiency of the stomach, and of course it may exist for a time before dilatation ensues.

Dilatation of the stomach may occasion an abnormal prominence of the abdomen, especially marked at the epigastrium and extending more or less below the umbilicus. The projecting tumor yields at the upper part a tympanitic resonance on percussion having the characteristic gastric quality of sound. At the lower portion, if the stomach contain ingesta, there is flatness on percussion, and the relative situation of the flatness and tympanitic resonance changes when the position of the patient is changed from the vertical to the recumbent. On succussion a splashing sound is produced analogous to that in pneumo-hydrothorax. With the ear or stethoscope placed over the stomach, metallic tinkling may be heard when the patient swallows liquid. Sometimes a sizzling sound is produced by bubbles of gas generated by fermentation. These auscultatory signs, however, are not distinctive of dilatation. They may be caused by the presence of air and liquid in a stomach more or less distended, but not dilated. If the abdominal walls be thin, the form of the stomach may be apparent and its undulatory movements visible. According to Kussmaul, these peristaltic undulations are characterized by their intensity, their long duration, and by coming on speedily after meals. They may give rise to disagreeable sensations, whereas in health persons are unconscious of the peristole of the stomach. The enlargement of the stomach varies at different times according to the variations in the quantity of ingesta and gas. A sense of fluctuation may be perceived by the touch when the stomach contains considerable liquid.

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[1] The methods of testing for free hydrochloric acid in the gastric contents are described in *Pepper's System of Practical Medicine*, vol. ii. p. 544.

With the object of ascertaining the amount of dilatation by the eye and by percussion the method of generating gas within the stomach, as first proposed by Frerichs, may be resorted to. The quantity of effervescing powder required to bring out distinctly the contour of the organ varies in different persons according to its capacity, the tension of the walls of both the stomach and the abdomen, the reflex susceptibility to vomiting, etc. From 20 to 30 grains of bicarbonate of soda and from 15 to 20 grains of tartaric acid may be at first introduced. The soda is to be given in a little lukewarm water and followed by the acid. Some persons with relaxed, dilated stomachs require 2 or 2½ drachms of soda and a corresponding quantity of acid.[1] The amount of dilatation may also be demonstrated by means of a stomach-sound, the length which is introduced being ascertained, and the extremity can sometimes be felt by the hand applied over the abdomen.

It is important to know that a person may be found to have an unusually capacious stomach, but if it do its work well—that is, if it be sufficient in the sense in which this term is used by Rosenbach—and is empty in the morning, there is no propriety in considering the large size of the organ as a morbid condition, any more than a similar condition of the bladder. On the other hand, the signs may not denote much enlargement; but if the organ be insufficient, its muscular power being inadequate to propel the contents during the night into the intestine, either dilatation exists or a condition leading thereto. Since attention has been directed to dilatation of the stomach more than heretofore, it has probably not infrequently been diagnosticated on insufficient grounds. No organ in the body varies in volume more widely within the range of health than the stomach, and an unusually large volume is not to be considered as dilatation unless the symptoms denote habitual undue detention of the ingesta.

In cases of dilatation the quantity of urine is often diminished and its reaction is alkaline. The bowels are constipated. The body wastes from innutrition. The symptoms of cancer of the stomach when the pylorus is obstructed are referable in a great part to the dilatation which ensues.

Paracentesis has been practised in a case of enormously dilated stomach under the belief that the affection was hydro-peritoneum. Physical examination of the abdomen, in addition to the symptoms characteristic of dilatation, should enable the practitioner to avoid this error. In hydro-peritoneum the enlargement is not uniform, but is greatest at the lower part of the abdomen, the reverse being true of dilatation of the stomach. In hydro-peritoneum the enlargement is proportionate anteriorly and laterally; in dilatation of the stomach the anterior projection is greater than the lateral. In hydro-peritoneum flatness on percussion exists at the lower part of the abdomen; in dilatation of the stomach intestinal tympanitic resonance will be likely to be found below the space which yields flatness—namely, in the hypogastric and iliac regions. The variation in the enlargement and its disappearance after complete vomiting or the use of the stomach-pump suffice for the diagnosis.

If not dependent on pyloric stenosis a considerable degree of dilatation may not give rise to notable inconvenience; but if the dilatation be great, nutrition suffers in consequence of the non-absorption of the accumulated ingesta. Rupture may occur if the walls be attenuated. A fatal termination, however, irrespective of carcinoma or other diseases which occasion obstruction, is rare.

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[1] Ebstein has described under the name insufficiency of the pyloric valve (incontinentia pylori) a condition in which the gas thus generated is not retained in the stomach, but rapidly escapes into the intestines through the pyloric orifice. (Vide "Ueber die Nichtschlussfähigkeit des Pylorus," *Volkmann's Sammlung klinischer Vorträge*, No. 155, 1878.)

Regulation of the ingesta as regards quantity and quality, together with tonic remedies, constitutes appropriate treatment. Indigestible food and articles which from decomposition develop gas in abundance should be interdicted. The diet should consist of meat, milk, soups, and eggs. Saccharine and amylaceous articles are to be avoided, in consequence of their tendency to fermentation. Strychnia or nux vomica may be given with a view to increase the muscular tone of the stomach. Artificial digestion may in a measure be effected by pepsin and hydrochloric acid. Food artificially digested before it is ingested (peptones) is appropriate. Kussmaul recommends the withdrawal of the contents of the stomach by means of the stomach-pump whenever there is evidence of undue detention, and claims that a cure may be sometimes effected by this method of treatment. The advantages of this method as a means of obtaining relief are obvious; but it is preferable, if practicable, to adjust the diet so that undue detention will not take place. Inasmuch, however, as this is not always practicable, if there be pyloric stenosis the use of the stomach-pump is a valuable resource. When the obstruction is so great that the system fails to receive sufficient alimentary supplies by the stomach, supplementary rectal alimentation becomes an important measure of treatment. In some cases it may be advisable to nourish for a time wholly by the rectum. Giving the stomach nothing to do in the way of digestion is preferable to the introduction into it of ingesta to be withdrawn. There is, moreover, risk of injury from the suction force of the stomach-pump. Portions of the mucous membrane of considerable size have been repeatedly torn away and withdrawn by the pump. As yet, however, no instance of serious injury from this cause has been reported. The siphon may be used instead of the pump, and is unattended by risk of injury, but it is ineffectual for the removal of solid contents. Kussmaul recommends washing out the stomach every alternate morning before taking food. Patients may learn to wash out their own stomachs.[1]

Degeneration of the Gastric Tubules—Atrophy of the Stomach.

Together with the liver, kidneys, and heart, the stomach is a frequent seat of *parenchymatous degeneration* or *cloudy swelling*. (See Part I. p. 53.) In this condition the mucous membrane is somewhat swollen, and may be either congested or pale. The microscope shows the glandular cells to be swollen and filled with albuminous granules, which, at least in great part, dissolve in acetic acid. The contours of the cells may be so obscured that the tubules seem to be filled with a granular mass. This change, when of much intensity, frequently leads to *fatty degeneration*. The cells now contain fatty molecules which refract the light more than the albuminous granules, and are not soluble in acetic acid. In extreme degrees of this degeneration, such as may occur in phosphorus-poisoning, the cells are more or less disintegrated and their places occupied by a fatty and albuminous detritus. The most frequent and important causes of parenchymatous degeneration are the febrile and infectious diseases, of which may be specially mentioned typhus and typhoid fevers, smallpox, septicæmia, puerperal fever, and yellow fever. Phosphorus-poisoning has been mentioned as a cause of the more extreme grades leading to fatty degeneration. Poisoning by arsenic, mercury, or the mineral acids is said to produce similar changes. It cannot be doubted that these alterations in the peptic cells impair the secretion of the gastric juice. It is reasonable to refer to them many of the disturbances in digestion which characterize the conditions named. Parenchymatous degeneration in the stomach, as elsewhere, is a condition from which recovery readily takes place when its causes are removed.

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[1] For further details, vide article by Leube in *Ziemssen's Cyclopædia*, vol. vii.; also, article by Prof. Welch in the *System of Practical Medicine, by American Authors*, vol. ii. p. 503.

While treating of the different forms of gastritis, reference was made to changes of the tubules, mostly of a degenerative character, which occur in inflammatory states. Parenchymatous and fatty degenerations and atrophy of the tubules were the most important of these alterations. An interesting comparison is often drawn between the changes in the gastric mucous membrane in chronic inflammation and those in the kidney in chronic diffuse nephritis. In both there are increase of the interstitial tissue, fatty degeneration of the glandular epithelium in certain places, atrophy of the tubules, with constriction of the tubules at certain points and cystic dilatation at others.

There is reason to believe that degenerative changes in the gastric mucous membrane occur independently of chronic inflammation and of the various causes of parenchymatous degeneration. *Atrophy of the stomach*, *atrophy or degeneration of the gastric mucous membrane or of the gastric tubules*, are names which have been employed to designate these changes. Alterations in the gastric glands have been studied especially by Handfield Jones, Wilson Fox, Habershon, Fenwick, and Henry and Osler. It is generally recognized that atrophic changes occur in the mucous membrane of the stomach, as in other organs, in chronic wasting diseases and in old age; but it is maintained by Fenwick and others that atrophy of the stomach occurs also as a primary morbid condition attended by grave symptoms; in other words, that it constitutes a disease by itself. So long ago as in 1860, in a published lecture on anæmia, I ventured the opinion that degeneration of the gastric tubules is the anatomical characteristic in certain cases now included under the name of pernicious anæmia. (See p. 384 *et seq.*).[1] In the third edition of this work (1868) I predicted that future investigations would disclose important facts pertaining to morbid conditions seated in the gastric tubules. In an essay published in 1871,[2] I endeavored to still further substantiate these views. My conclusions were based partly on deduction and partly on the anatomical studies of Jones, Fox, and Fenwick. My object was to show, from a clinical point of view, *first*, the existence of a well-defined class of cases characterized by anorexia, impaired digestion, progressive debility, anæmia, and death from inanition; and *second*, in view of these clinical characteristics, together with the absence of adequate lesions elsewhere, the probability that the essential disease is seated in the secretory glands of the alimentary canal. It is gratifying to be able to add that recent years have furnished several contributions to our knowledge of this still obscure subject. Fenwick, in a lecture on atrophy of the stomach in 1877,[3] published four cases with autopsies showing atrophy of the gastric tubules without thickening of the walls and without diminution of the lumen of the stomach. The symptoms resembled closely those which have been described in the article on Pernicious Anæmia. Quincke, in an essay on pernicious anæmia,[4] mentions a case in which the gastric symptoms were even more marked than is usual in pernicious anæmia, and in which after death the gastric mucous membrane was found to be thin and pale, the glands being few and far apart. He suggests that the atrophy of the gastric mucous membrane may have been the starting-point of the disease. Brabazon in 1878[1] reported a case of general atrophy of the stomach with absence of organic disease. The symptoms during life were anorexia and extreme anæmia. The walls of the stomach were found to be atrophied, but, unfortunately, no microscopical examination was made. In 1879, Nothnagel[2] published a case of cirrhotic contraction of the stomach attended by disappearance of the gastric glands in all parts except the pyloric region. The microscopical examination was made by W. Müller. The clinical history of this case was in all respects that of pernicious anæmia, and this was the diagnosis during life. Nothnagel does not hesitate to refer these symptoms to the atrophy of the gastric tubules. As he remarks, atrophy of the gastric glands appears under two conditions, the one with and the other without thickening of the walls of the stomach. The former condition corresponds to cirrhosis of the stomach, the latter to simple atrophy. It is the latter form to which Fenwick has called especial attention. In some of the reported cases an increase of the interstitial tissue of the mucous membrane has been observed, but whether it be primary or secondary is uncertain. Even if the interstitial change prove to be primary, there is no propriety, as Nothnagel justly says, in considering the process as an ordinary chronic catarrhal gastritis. A case of fatal atrophy, the history being that of pernicious anæmia, has been recently reported by Thorowgood, and a similar case has been carefully studied and reported by Henry and Osler.[3]

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[1] *American Med. Times*, 1860.

[2] "The Pathological Relations of the Gastric and Intestinal Tubules," by Austin Flint, M. D., *New York Med. Journal*, March, 1871.

[3] *The Lancet*, 1877, July 7 *et seq.*

[4] *Volkmann's Sammlung klinischer Vorträge*, No. 100 (Case 6).

The SYMPTOMS which have been observed in cases of atrophy of the gastric tubules are those of severe anæmia. The digestive symptoms are—loss of appetite, vomiting (usually but not always), distress after eating, and eructations. These are symptoms in cases of pernicious anæmia. It is desirable, as Nothnagel suggests, to pay special attention to the condition of the stomach in death from pernicious anæmia. The pathological conditions are probably not the same in all cases of this grave form of anæmia. That a certain proportion of the cases are referable to atrophy of the gastric tubules may reasonably be inferred from cases already published. From a clinical standpoint it is important to take cognizance also of atrophy of the glands of the stomach as a secondary change in various affections of this organ, especially in chronic inflammation and in cancer. It is to be expected that in the future greater attention will be directed to the condition of these important glands in disease, and that valuable contributions will thereby be made to our knowledge of this recently-opened field of research.

Waxy degeneration of the walls of the stomach is not a very infrequent condition in cases in which this degeneration is widely extended. As a rule, the metamorphosis is more marked in the small and large intestine than in the stomach. All the coats of the stomach may be involved, but generally the change is greatest in the muscularis mucosæ and the mucous membrane. Here, as elsewhere, the waxy material appears first in the walls of the small arteries and about the capillaries, and it may be confined to these structures. The change may, however, extend to the surrounding tissues. In the majority of cases the epithelial cells of the tubules do not undergo the waxy metamorphosis. According to Kyber, amyloid degeneration of the stomach is generally accompanied by chronic inflammatory changes and pigmentation. Extensive amyloid degeneration may so impair the nutrition of the gastric mucous membrane as to favor the development of round ulcer. Although, according to Kühne, the amyloid material outside of the body is extremely resistant to the action of the gastric juice (see p. 55), it would appear from the studies of Kyber that this substance in the living body may be digested by the gastric juice. Amyloid degeneration is generally an accompaniment of severe wasting disease (p. 56). That the functions of the stomach are impaired by this metamorphosis is in the highest degree probable, but we are without positive information on this point.

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[1] *British Med. Journal*, July 27, 1878.

[2] *Deutsches Archiv für klin. Med.*, Bd. 24, p. 353.

[3] Thorowgood, *Med. Times*, Feb. 5, 1881; Henry and Osler, *Amer. Journ. Med. Sciences*, April, 1886.

CHAPTER IV.

FUNCTIONAL DISEASES OF THE STOMACH.

Dyspepsia and Indigestion.—Polyphagia, etc.—Gastrorrhagia.—Inanition.—Gastralgia.

Dyspepsia.

OF the functional disorders of the stomach and intestine, a large proportion relate to digestion; that is, to the processes involved in the changes which the ingesta undergo in the alimentary canal. The processes of digestion take place within the stomach and small intestine. Certain of the functional disorders of digestion are commonly embraced under the name *dyspepsia.* This name signifies simply difficulty of digestion, but conventionally it is applied to cases in which the digestive processes are disturbed in various ways, as well as to the cases in which their performance appears only to be difficult. It is, however, convenient to make a distinction between simply difficult digestion—that is, dyspepsia in the literal sense of the term—and disordered digestion. The latter may be distinguished as indigestion. Generally, dyspepsia denotes ailments which are sufficiently persisting to be considered as chronic. The term *acute dyspepsia,* however, is applied to a disorder of the digestive organs attended with comparatively violent symptoms and having a short duration. The disorder so called claims a brief notice.

The term *acute dyspepsia* denotes an affection called by French writers *embarras gastrique*, and popularly known with us as a *bilious attack.* When accompanied with more or less febrile movement it is sometimes called *gastric fever.* The latter term is objectionable, because it implies that the affection is essentially a fever, whereas the febrile movement is symptomatic. The term bilious attack is also objectionable, because it is by no means clear that the pathology of the affection involves special disturbance of the liver. In the absence of precise knowledge of the pathological character of the affection the term acute dyspepsia is to be preferred, inasmuch as, pathologically, it expresses no more than a functional disorder pertaining to digestion. The term, in fact, is applied to cases embracing probably different morbid conditions.

In certain cases of acute dyspepsia the disorder is caused either by over-repletion of the stomach, the ingestion of indigestible articles of food, or an arrest of digestion by strong emotions, fatigue, or other causes. It is manifested by a sense of weight, fulness, or pain in the epigastrium; nausea and perhaps vomiting may occur, or after a time looseness of the bowels ensues; some febrile movement may be induced, with pain in the head and general malaise. Succeeding these symptoms are loss of appetite, coating of the tongue, an unpleasant taste, with more or less uneasiness referable to the digestive organs for several days. Such cases are common. In the way of treatment they claim only a mild evacuant remedy and regulated diet for a few days, with perhaps some tonic remedy.

The practitioner meets often with cases which differ from the preceding in the disorder not being distinctly referable to indigestion. There is impairment or total loss of appetite; nausea and slight vomiting may occur, but oftener a liquid containing bile is regurgitated; uneasiness or undefined distress is felt in the neighborhood of the stomach; the bowels are sometimes constipated and sometimes loose; the patient complains of a bitter taste, and the breath is frequently offensive; the tongue becomes furred or coated; pain in the frontal region of the head is a frequent symptom, which if accompanied by nausea goes by the name *sick headache;* the urine deposits an abundance of urates; in some cases there is moderate febrile movement; and the countenance frequently presents a sallow appearance. Patients imagine that their malady is very clearly defined when they declare that they are bilious. It is frequently, if not generally, difficult to trace the affection to any cause. It occurs undoubtedly oftener in the spring than in other seasons of the year. It continues a few days, and gradually the normal condition of the stomach and system is regained. It is probable that in many, if not most, cases of acute dyspepsia there is subacute gastritis. In certain cases the affection is that called by some authors catarrh of the stomach, to which reference has been already made.

Patients with acute dyspepsia are likely to desire active treatment. They fancy they need cleansing remedies, and in this light they regard emetics and cathartics, or they solicit medicines to act upon the liver. Readjustment of digestion, etc. takes place without medication, but a mercurial cathartic often appears to afford prompt relief. A few grains of calomel or blue mass, followed by a saline purgative, and afterward a mild tonic, such as a grain or two of quinia two or three times daily, with regulated diet, are appropriate measures of treatment.

The term *dyspepsia*, without the prefix *acute*, is applied to a more persistent affection than that just considered. As the name of a functional affection it is not correctly used when applied to disorders of digestion connected with inflammation or structural lesions of the digestive organs, or to disorders incident to fever and other general diseases. The term is much less frequently used now than formerly. One reason for this is, that formerly the term had a wider latitude than now, embracing affections which progress in pathological knowledge has placed nosologically elsewhere; but the affection is undoubtedly less prevalent in this country than it was half a century ago.

The source of chronic dyspeptic ailments and indigestion may be either the stomach or the small intestine; hence the names gastric and intestinal dyspepsia and indigestion are appropriate. These two forms of disorder can generally be differentiated. Without treating of these forms separately, I shall indicate the symptoms on which the differentiation is to be based.

The symptoms of dyspepsia are, in the first place, local—that is, referable directly either to the stomach or to the small intestine, and in the second place, manifested elsewhere—in other words, sympathetic or consecutive. The local symptoms, which will be first noticed, are varied in character, and might serve as a basis for the subdivision of dyspepsia into several varieties.

These symptoms are conveniently classed as follows: *First*, symptoms denoting labored or difficult digestion—that is, dyspepsia in the literal sense of the term; and *second*, symptoms proceeding from imperfect or disturbed digestion—that is, indigestion.

In certain cases the processes of digestion appear to be fully accomplished, but they are accompanied with uncomfortable sensations. In healthy persons, as is well known, a good digestion is conducive to a sense of comfort. It is quite the reverse in some dyspeptics. They are often miserable while the digestive processes are going on; they complain of uneasiness in the stomach or intestine, of a sense of distension, and of a general sense of discomfort; and these symptoms may afford the only evidence of disorder. The food appears to be digested and the body may be well nourished, whereas in other cases these symptoms are associated with those denoting imperfection or disturbance of the digestive processes. On the other hand, when digestion is incomplete or disturbed various symptoms arise in different cases, and with these there is sometimes comparatively little suffering. It is a curious apparent inconsistency that some persons manifest habitually more or less derangement of digestion without much annoyance, while other persons appear to digest perfectly, but pass a wretched existence. In cases of dyspepsia without evidence of indigestion the disorder has been distinguished as *nervous dyspepsia.* Leube has demonstrated, by withdrawing the contents of the stomach at different periods after the ingestion of food, that labored digestion, or the so-called nervous dyspepsia, is not incompatible with the complete performance of the digestive process within the stomach.

The more prominent of the local symptoms in cases characterized by gastric indigestion may be arranged under the following heads: Regurgitations (including pyrosis), cardialgia, tympanites, and vomiting.

Certain cases are characterized by regurgitations from the stomach. The liquid regurgitated is sometimes intensely sour from the presence of lactic, butyric, acetic, or other organic acid. If the regurgitations occur, as is usual, during the progress of stomach digestion, the acidity is due to chemical changes in the ingesta; but occurring, as is sometimes the case, after long fasting, when the stomach contains no food, it may come from the gastric glands. The presence of hydrochloric acid in the stomach may prevent the chemical changes in the ingesta, and hence this acid is sometimes a remedy in cases characterized by acidity of eructations. The regurgitated liquid is sometimes acrid, seeming to scald the throat; or it may be greasy or nauseous, having the odor and taste of rotten eggs, the latter denoting putrefactive changes. Some persons possess or acquire the faculty of voluntary regurgitation, and are able to expel at will the contents of the stomach—a faculty analogous to that of rumination in herbivorous animals.

The regurgitation of a considerable quantity of a liquid which is either insipid or saltish or brackish, and is sometimes acid, when the stomach is empty of food, and usually in the morning, is called pyrosis or water-brash.

Cardialgia signifies pain at the cardiac orifice of the stomach of a burning character, and shooting into the chest and up the œsophagus into the throat. It is vulgarly called heartburn. Every one has occasionally experienced this symptom. It characterizes certain cases of indigestion, and it is evidently dependent on the presence of an acid in the stomach, as it is quickly and completely relieved for a time by an alkaline remedy.

A painful sense of fulness after eating is common without any abdominal distension, but more or less distension from air or gas is a prominent symptom in certain cases, constituting tympanites. In most cases the gas is derived from putrefactive or fermentative changes in the ingesta. The nature of the gas will depend on the kind of food ingested. In the decomposition of albuminoid substances sulphuretted hydrogen is produced, and the gas expelled from the stomach has a characteristic fetor; the butyric fermentation furnishes hydrogen and carbonic acid gas, and the latter is also furnished by the acetic fermentation. The formation of lactic acid is unattended by the production of gas. The accumulation of gas within the stomach, if considerable, occasions painful distension, embarasses respiration by interference with the descent of the diaphragm, disturbs the action of the heart, and interferes with the gastric peristaltic movements. The formation of gas resulting from over-repletion of the stomach may distend the stomach to such an extent as to cause sudden death. Two instances of this kind have fallen under my observation.[1]

A source of flatulent distension of the small intestine is from gas secreted or evolved in some way independently of chemical changes in the ingesta. Some persons suffer from an habitual tympanites. Females are more liable to it than males.' Cases are occasionally met with in which the rapid development of tympanites occasions great suffering, the abdomen becoming enormously distended, the respiration embarrassed by interference with the descent of the diaphragm, and in females the suffering being increased by pressure upon the uterus and bladder. In a case under my observation paroxysms of intestinal tympanites, occasioning intense distress, had occurred daily for several successive months, an inodorous gas sometimes passing off by the rectum, and sometimes the tympanites disappearing without any escape of gas. A host of remedies had been employed in this case unsuccessfully, and at length improvement took place under simple palliative treatment. In a case of this description the appearance of the abdomen during an attack of tympanites was extraordinary. The convolutions of the small intestine and their peristaltic movements were distinctly visible through the distended abdominal walls. So far as my observation goes, such cases occur only among females, and the tympanites is associated with more or less neuropathic disorder. Hysterical phenomena are sometimes associated with it.

There is another source of flatulent distension, the seat of the tympanites being the stomach—namely, the ingestion of air. More or less air is habitually commingled with food and drink, but the quantity received in this way is never sufficient to distend the stomach. A habit of swallowing air is sometimes acquired, resembling crib-biting or wind-sucking in horses. Some writers refer to this habit as not uncommon, but the effort required in the deglutition of air is such that the act could not escape detection; and, judging from my own experience, cases are by no means frequent in this country. In a case which came under my observation the patient was about fifty years of age and had suffered from a disorder of the digestive organs for twenty-five years. In order to show how he performed the act of swallowing air, he drank a quantity of water precipitately, and appeared to make violent efforts to force it downward. During these efforts the face became flushed and the eyes watery. Shortly the efforts were discontinued, and at the same moment a loud gurgling sound was heard in the region of the stomach. It appeared as if there were some obstruction in the œsophagus, but a probang was readily passed into the stomach. The patient stated that the habit had existed for twenty-five years. He was led to it gradually in order to prevent regurgitation, which he stated always occurred if he failed to ingest air in conjunction with his food and drink. He was accustomed to belch air frequently. Percussion over the stomach showed considerable gastric tympanites.

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[1] Reported in *Boston Med. and Surg. Journal*, March 10, 1841.

An habitual tympanites of the stomach occasions in some persons, after the ingestion of liquid, a succussion sound in walking resembling the sound frequently heard in horses. This is a source of much annoyance and mortification, especially to women. It may be avoided by taking into the stomach only very small quantities of liquid at a time. This symptom alone is not proof of dilatation of the stomach

Vomiting is not common in cases of chronic dyspepsia. It is more frequent in occasional fits of indigestion. It is, however, characteristic of a form of chronic dyspepsia occasionally met with, occurring generally in young girls. In this variety of disorder vomiting almost constantly follows the ingestion of food or drink; and the stomach seems to be intolerant of all forms of nourishment, although taken in small quantities. The vomiting occurs directly or soon after food is taken, and in some cases it is surprising that there is not more evidence of inanition, since everything taken into the stomach appears to be ejected; yet, although the disorder may continue for a long time, the patient does not become greatly emaciated or notably enfeebled. The disorder often resists all the usual remedies to relieve irritability of the stomach, such as bismuth, creasote, opiates, hydrocyanic acid, oxalate of cerium, etc. It is usually accompanied with mental depression, hyperæsthesia of the surface, and marked tenderness over the spinal column. In determining that the affection is purely functional, gastritis, gastric ulcer, subacute meningitis (especially in children), pregnancy, and disease of the kidneys are to be excluded. Restricting the patient to milk or some other article of diet, in very small quantities and repeated at very short intervals, will sometimes succeed; but the plan of treatment which in my experience has proved most successful consists in change of scene and living as much as possible out of doors. I have known a sea-voyage to prove efficacious.

The local symptoms just noticed are not peculiar to dyspepsia or indigestion as a functional affection. They may occur in connection with subacute gastritis and structural lesions of the stomach. In arriving at the diagnosis—that is, determining that only a functional affection exists—inflammation and the lesions which have been considered are to be excluded. They are to be excluded by the absence of their diagnostic symptoms, and for these the reader is referred to the preceding chapters.

Of symptoms other than those referable directly to the digestive organs mental disorder is one of the most noticeable. During the processes of digestion dyspeptics often experience an uncomfortable heaviness or dulness, rendering it difficult to concentrate the attention upon any subject. The following extract from Chambers's work gives a graphic representation of the condition in many cases: "There is great languor and incapacity for exertion, coming on generally about an hour after food, and accompanied in some cases by an almost irresistible drowsiness which lasts for several hours. This symptom is particularly marked in the afternoon if the patients dine in the middle of the day and endeavor to continue their employment afterward. Yielding to the drowsiness and taking a siesta seem to make matters worse, for there is usually felt after such an indulgence an intense headache or giddiness, which continues longer than the symptoms would have done had the inclination to sleep been resisted. The depression of spirits is not so remarkable as the feeling of utter helplessness, both of mind and body, accompanied in persons naturally active with a sense of shame at their unwonted apathy."[1] This condition may be connected with the process of digestion in the small intestine, and may be unaccompanied by symptoms denoting disorder of stomach digestion.

In most cases of dyspepsia patients suffer more or less from mental depression. They are devoid of buoyancy, gloomy, and disposed to look always on the dark side. Frequently, this depression is increased by anxiety respecting health. They are apprehensive of the existence of some grave disease, such as consumption or an affection of the heart. Their attention becomes concentrated upon themselves. They are constantly watching the sensations connected with digestion, and fall into the habit of counting the pulse, feeling the heart's beat, examining the abdomen, urine, etc. This mental disorder, carried beyond a certain point, eventuates in melancholia and hypochondriasis.

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[1] *Digestion and its Derangements*, Am. ed., p. 367.

In addition to these symptoms of mental disorder, various symptomatic phenomena belong to the clinical history of dyspepsia. Irregular action of the heart or palpitation is not uncommon. Constipation generally exists, sometimes alternating with diarrhœa. The skin is frequently dry and rough, and the extremities are often cold, showing deficient activity of the circulation. The tongue is usually furred or more or less coated. The urine deposits the lateritious sediment, and on microscopic examination crystals of oxalate of lime may be found in abundance: the latter, being incident to various morbid conditions and not uncommon in connection with trivial disorders, are incorrectly considered as denoting a special affection called *oxaluria.* Vigilance is a frequent source of complaint, and the sleep obtained is often incomplete and disturbed by dreams, the patient arising in the morning unrefreshed and miserable.[1] The appetite in the morning is often poor, becoming perhaps during the day morbidly craving. A dry hacking cough is reckoned among the sympathetic phenomena, but this is rare. The existence of cough should always excite suspicion of pulmonary disease or of chronic pharyngitis.

With reference to the pathology of dyspepsia and indigestion, it is to be considered that digestion is not a simple process. Including under this term all the changes which take place in the ingesta within the alimentary canal, there are certain changes in the stomach from admixture with the salivary fluids and the gastric juice, others in the duodenum from the addition of the bile and pancreatic secretion, and others in the small intestine below the duodenum from the action of the intestinal juice. For the proper activity of the organs and for the secreted liquids, the latter being vastly more abundant than was formerly supposed, a large supply of blood is required. The movements due to the muscular coat of the stomach and intestine are important. A certain influence derived from the nervous system is requisite for the secretion of the liquids and for the peristaltic movements. An analysis of the symptomatic phenomena in different cases of dyspepsia shows that the different elements which enter into the function of digestion may be affected either separately or with different degrees of predominance. Thus, vomiting and regurgitation show irritation and spasmodic action; pain or distress, morbid sensibility; prolonged digestion, with chemical changes, deficiency in the digestive liquids, etc. It is of importance to take cognizance of these several elements in the management.

As regards the differentiation of gastric and intestinal dyspepsia and indigestion, the period which elapses after eating before the inconvenience is felt, and the absence of symptoms of disorder referable to the stomach, show the affection to be intestinal rather than gastric. In cases of intestinal disorder from two to four hours elapse after the ingestion of food before the manifestation of the local and general symptoms. Diarrhœa is a frequent symptom of intestinal indigestion. This is the source, in most cases, of functional diarrhœa.

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[1] The poet Cowper, who appears to have suffered from intense mental depression in connection with dyspepsia, describes his feeling in the morning in the following forcible language: "I awake like a toad out of Acheron, covered with the ooze and slime of melancholy."

The *causation* involves agencies affecting the digestive processes either directly or indirectly. Excesses in eating and drinking act as direct causes. The digestive powers may be overtasked by the quantity or over-excited by the quality of food. Under these circumstances dyspepsia has been called "the remorse of a guilty stomach." The habit of spirit-drinking engenders dyspepsia. Drunkards after a time becomes dyspeptics. Dyspepsia may follow subacute gastritis or enteritis produced by dietetic error or other causes; inflammation leaving the organs in a weakened state. These direct causes, however, are not so largely operative as is generally supposed.

The indirect more than the direct agencies are involved in the causation. These are various. Anæmia is one of the indirect causes. It gives rise to dyspepsia by diminishing the functional activity of the gastric and intestinal glands, the digestive liquids being lessened, or affected as regards their quality. Clinical observation shows that in cases of anæmia the digestive power is generally more or less weakened. On the other hand, impaired digestion may stand in a causative relation to anæmia. A notable change in habits as regards physical exercise is not infrequently causative. The vigor of the digestive organs is in a measure commensurate with the demand for nutritive material, and the latter is dependent on the waste of the tissues. Now, certain dietetic habits are formed in accordance with occupations involving physical activity and a proportionate amount of disassimilation. Persistence in these dietetic habits after a change from an active to a sedentary life is likely to induce disorder. This is, in part at least, an explanation of its occurrence among those who exchange the farm or workshop for the desk or study, and among those who retire from business to enjoy leisure.

In a large proportion of cases dyspepsia originates and is perpetuated by mental causes. It is induced and kept up by anxiety and depression. Disorder is often, in the first place, produced by mental causes, and then it reacts upon the mind, increasing its morbid condition. An examination into the previous history of cases will frequently show the affection to have been preceded by mental disquietude of some kind. Persons of a certain mental constitution are especially prone to dyspepsia—namely, those so constituted as to be constantly anxious about something, such as acquiring success in life, getting out of debt, securing an independent position, or about imaginary troubles. The period of life when persons are most liable to dyspepsia is that in which anxieties are likely to be greatest—namely, between adult age and middle life. After the latter period the events of life are taken more calmly and there are fewer sources of intense anxiety. Persons who have been dyspeptics for many years are often free from this affection after having entered upon middle life. It may be doubted if the affection be often produced by overtasking the intellect. When it occurs in those who are devoted to intellectual pursuits, it arises generally from the sedentary habits connected with these pursuits or from mental anxiety; and, on the other hand, deficient exercise of the intellectual and moral faculties frequently leads to it. Conditions in life involving ennui and dissatisfaction predispose to it. Under these circumstances the attention tends to become concentrated on the functions of the body, which go on best without close surveillance. These views are sustained by observing the classes of persons who suffer from dyspepsia. Epicures and gormandizers by no means of necessity become dyspeptics; and they often escape this affection, when their indulgences lead to other consequences, such as gout, etc. The laboring-classes are not prone to dyspepsia, notwithstanding the use frequently of the coarsest and most indigestible kinds of food, prepared without regard to the art of cookery; but cases are met with especially among retired rich persons without occupation or mental resources, among those who have no useful or proper aims in life, and among those who are debarred from the interests of domestic life. In these several classes it may be developed irrespective of any dietetic errors.[1]

TREATMENT.—In the management of dyspepsia and indigestion, as of other affections, the first point is to remove or obviate causes, as far as this is practicable. The practitioner may be able to control causes relating to dietetic errors, but he may not be able to reach those connected with the mind, and hence there is difficulty in effecting a cure. Measures of treatment may be arranged in three divisions—namely, 1st, those relating to diet and regimen; 2d, measures addressed to the mind; and 3d, medicinal remedies.

1. If the affection have been induced by dietetic excesses it is sometimes useful to give the digestive organs a short period of rest, and with this view to reduce the diet for a time below the wants of the system. This, however, should be but temporary. A cure is not to be effected by persisting in this course. Not only the system is reduced, but the digestive functions are impaired by too great or too protracted abstemiousness. Dyspepsia is, in fact, perpetuated, and may be produced, by attempts to live on as little and as coarse food as possible. Persons who think it vulgar, unrefined, or worldly-minded to eat well are likely to be subjects of dyspepsia as well as of other affections. This is true also of those who fancy that most maladies arise from over-eating, and that the great problem of health-preservation is to learn to eat sparingly. The object of treatment is to bring the digestive powers up to such a point of improvement that particular care in diet is not requisite.

As regards the diet for dyspeptics, there are no rules suited to all cases. Individual experience in each case is not to be altogether ignored, but there is a liability to error in regard to this experience. Unusual difficulty of digestion, or indigestion, after a meal is often imputed to certain articles of food when it is due to some other incidental circumstance. Idiosyncrasies in relation to particular articles of food are far less common than the statements of patients would lead one to suppose. In general, articles which are wholesome to most persons are not unwholesome to any. It is rarely true that "what is one man's meat is another's poison." As a rule, when a patient says that he cannot take such and such articles which general experience shows to be digestible and nutritious, it is fair to presume self-deception; and of this the patient may generally be convinced if persuaded to persist in their use. At first, the expectation that an article will prove hurtful contributes to render it so; but after a time the idea is overcome. It is often an object in the treatment of dyspepsia to do away with these fallacious ideas respecting various kinds of food. Some persons are fond of encouraging the notion that their digestive organs are endowed with peculiar susceptibilities which prevent them from conforming to general rules of diet. Their egotism is gratified by the thought that they differ in this regard from the common herd. In most cases animal food is best digested, especially old and tender meats plainly but well cooked; but in some cases a milk and farinaceous diet is found to agree best. An obvious reason why so many persons imagine they do not digest milk well is that it is often taken as a beverage after or with solids, the fact that it is, in effect, a solid article of food not being appreciated. Bread, to be readily digestible, should not be new, nor is it desirable that it should be stale. Well-boiled rice, corn-meal mush, and other analogous farinaceous preparations are easy of digestion. Crude vegetables are digested with more difficulty, but they are not to be interdicted as a matter of course. Some dyspeptics find even the much-abused cucumber grateful to the stomach as well as to the palate. Pastries, rich puddings, and sweetmeats are generally to be eaten sparingly or to be discarded. Ripe fruits in proper moderation are useful. It is never advisable for the patient to adopt a restricted range or any particular system of diet. On the contrary, it is important to persevere in attempting to digest all the varied forms of wholesome food, not being restricted to meat or a vegetable diet, but aiming to eat like persons in health, without the need of particular care in the selection.

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[1] For a fuller exposition of these views of the causation of dyspepsia, vide article by the author in the *Am. Journ. of Med. Sciences*, Jan., 1841.

Different kinds of food are suited to different cases according as the functional disorder is chiefly gastric or intestinal. If gastric, farinaceous articles will be likely to be best suited, as these are digested mainly in the small intestine. Fatty articles may be well digested in these cases. On the other hand, if the disorder be intestinal, farinaceous and fatty articles of food will be likely to be difficult of digestion, and albuminoid substances or an animal diet will be found to occasion less inconvenience.

Restriction to a few meals a day is not an uncommon error. If a patient have no appetite for breakfast, an early lunch should be taken. If the dinner be near the middle of the day and an early tea be taken, some light food in the evening is advisable. Long intervals between the periods of taking food tend to weaken the powers of digestion. Food should be eaten slowly. Deliberate mastication prepares the food for the action of the gastric juice. Moreover, slowness in eating gives the stomach a chance to express satiety, and is therefore a protection against over-eating. Errors, however, in this respect are likely to be over-estimated.

With regard to the use of wine or spirits, the danger of intemperance is always to be considered, especially as dyspepsia not infrequently leads to a morbid desire for stimulants, and in view of the fact that the mental state is favorable for the formation of intemperate habits. Yet an alcoholic stimulant taken with meals is often useful until the normal powers of the digestive organs are restored. Stimulants should never be taken when the stomach is empty.

It may be well to remind the practitioner that it is unphilosophical to judge concerning his patients from the conclusions derived from his own personal experience. The physician who has his notions of what kinds of food agree with himself best is often inclined to apply these notions indiscriminately to his patients.

To consider fully the subject of dietetics would be incompatible with the scope of this work; but, practically, this is less desirable than many might suppose. My experience has led me to adopt a plan of dietetic treatment which has the merit of great simplicity, and which I have found to be very successful. I am accustomed to say to patients that if they expect to acquire a good digestion they must not pay particular attention to diet—that they must follow the dictates of instinct rather than any precise rules as respects the articles of food, the quantity to be taken, and the time of taking it. I have never known a dyspeptic to recover vigorous health who undertook to live after a strictly regulated diet, and I have never known of an instance of a healthy person living according to a strict dietetic system who did not become a dyspeptic. On the other hand, in a great number of cases in which persons had been sufferers for years on a regulated diet, health has been speedily regained by eating simply in accordance with appetite.

The clothing of dyspeptics should be such as will secure uniformity of temperature and maintain the functions of the skin, without over-accumulation of heat or exciting perspiration. A sense of comfort is the criterion in this regard. Exercise is of importance, but to be useful it must be taken with a motive and end aside from the sanitary object. Exercise taken simply as exercise tends to keep the mind of the patient on his disorder, and it will not be likely to be persisted in. There is a risk of resorting at once to over-exercise, a too violent change of habits in this regard proving hurtful and discouraging the patient. The exercise should be in the open air. A change of business from one requiring sedentary habits to one involving out-of-door occupation is often advisable. Travelling, especially in foreign countries, where the attention is diverted with a succession of novel scenes, is highly useful. Sir James Johnston said that no case of purely functional dyspepsia could resist a pedestrian tour over the Alps.

2. The moral treatment consists first in establishing confidence by attention and sympathy. Inattention to the details into which dyspeptic patients are fond of entering, and ridicule of hypochondriacal fears, prevent the physician from being of service to this unhappy class of patients, and lead them to seek for aid from quacks and secret remedies. If the confidence of the patient be secured, the assurances of the physician will often remove needless apprehensions, and afford an encouragement which in itself will contribute not a little toward improvement. If the patient be convinced that his mental state is in a measure due to a disordered condition of the body, he derives consolation and encouragement from this belief. The treatment relating to the mind will embrace, as far as possible, measures having reference to the removal of mental causes which may have produced or which perpetuate the affection. It is important to incite to occupations which divert the attention from the bodily functions, especially during the process of digestion. It is desirable that the mind should be pleasantly occupied. It is often observed by dyspeptics that if they dine alone, and are left by themselves to watch the sensations connected with digestion, they are sure to suffer, whereas they experience no inconvenience from a greater amount of indulgence in eating if they be in agreeable company during and after dinner. Popular works on indigestion do harm by directing to the subject the attention of those prone to dyspepsia. The benefit of travel is, in a great measure, moral, involving recreation as well as exercise, and keeping the mind occupied with extrinsic subjects.

3. The division of the treatment which relates to medicinal remedies is relatively least important, but it is by no means unimportant. If the symptoms denote morbid sensibility of the stomach, counter-irritation on the epigastrium is often useful. Small blisters, the strong aqua ammoniæ, or the croton oil may be used for this purpose. The benefit is probably in part due to a moral effect. The nitrate of silver may be given in these cases, care being taken not to continue it too long. If anæmia be associated, the chalybeate tonics are indicated, of which in cases of dyspepsia the tincture of the chloride of iron is one of the best. The addition of an equal part of dilute phosphoric acid and of the syrup of ginger or orange makes this an agreeable remedy. The citrate of iron and quinia is an excellent form of tonic in dyspepsia associated with anæmia. The powder of iron reduced by hydrogen is another eligible form. If there be constipation, the bowels are to be regulated by appropriate measures, but purgation is to be avoided. Constipation will be considered as a distinct functional affection. There are no special indications for mercurial remedies except as an occasional cathartic or laxative. In general, the various vegetable tonics are useful, the tonic remedy being varied from time to time. A grain or two of quinia twice or thrice daily, continued steadily for several weeks, appears to be often useful. A few drops of hydrochloric acid after eating may frequently be prescribed with advantage. The benefit of the tincture of the chloride of iron is probably in part due to the hydrochloric acid. Pepsin, in doses of five or ten grains after each meal, appears often to be of service. The principle on which it is employed—

namely, to supply a deficient element in the gastric juice—entitles it to be considered only a palliative, not a curative remedy. A convenient and eligible form in which pepsin may be given is the preparation commonly known as rennet wine. This is prepared by putting a fresh rennet, cut up into small pieces, into a pint of sherry wine, which after maceration for two weeks is to be strained, and it is then ready for use. Of this preparation from half a teaspoonful to a teaspoonful is to be given shortly after taking food. I have used for several years past, with great satisfaction, as a stomach tonic, salicin in ten-grain doses, dissolved in a wine-glass of water and taken just before each meal. The benefit derived from this remedy is often remarkable. It is useful especially when fermentation and flatulence result from indigestion. The salicin in these cases acts as an antiseptic as well as a tonic remedy.

Symptoms characterizing certain cases of dyspepsia claim particular remedies. Pyrosis is generally relieved by bismuth in doses of a scruple or half a drachm combined with a carminative, such as powdered cinnamon, in conjunction with measures relating to diet, hygiene, etc., and followed by tonic remedies. Cardialgia calls for alkaline remedies—namely, lime-water, liquor potassæ, bicarbonate of soda, and magnesia, the two latter especially if there be constipation. Flatulence and tympanites are relieved by carminative stimulants, the tincture of the essential oils, the aromatic powder, and charcoal powder, the latter being useful on account of its power of absorbing gases. All these remedies, however, are merely palliatives. The conditions on which depend the several symptoms just named are, if possible, to be removed by tonic remedies, such as salicin, quinia, and the mineral acids. Intestinal tympanites occurring in paroxysms from the secretion of gas is an extremely obstinate form of disorder. Purgatives are not useful. Opiates to relieve present suffering are required. Carminatives and alcoholic stimulants will sometimes afford relief. Asafœtida given by the mouth or rectum is highly useful, especially when hysterical symptoms are associated. In the intervals between the paroxysms measures, medicinal and hygienic, to invigorate the system are indicated, together with the use of antispasmodic remedies, such as belladonna, valerianate of zinc, etc. Small doses of strychnia, or of nux vomica in the form of tincture or extract, are useful in these cases.

The management in certain cases of dyspepsia characterized by persistent vomiting after taking food has been already alluded to. Change of scene and out-of-door life are the most efficient measures. A sea-voyage will be likely to prove effectual; but various remedies may be employed in succession, each of which will sometimes be found useful if not successful. The salts of morphia or codeia may succeed if the patient be not unpleasantly affected by opiates of any kind. The hydrocyanic acid in some cases is an efficient remedy. Creasote sometimes acts efficiently given in doses of one or two minims repeated after each act of vomiting. In young children this remedy, given in doses of one-eighth or one-sixth of a drop, frequently acts like a charm in arresting vomiting. Bismuth in large doses is a valuable remedy in some cases. Strychnia or the nux vomica has been found useful. The oxalate of cerium, from two to five grains at a dose, is another useful remedy. Counter-irritation on the epigastrium by means of blisters or dry cupping is frequently followed by relief. Milk, with lime-water added, is sometimes retained when other articles of food are rejected. Vomiting is symptomatic of a great variety of affections, exclusive of inflammation or structural lesions of the stomach. Occurring as a form of functional disorder of the stomach, it is sometimes considered as an individual affection, but it may with propriety be included among the symptoms of dyspepsia.

There is a variety of vomiting which claims a brief separate notice. Reference is had to cases in which a liquid is thrown from the stomach in greater or less abundance, emitting an odor of fermenting wort, and, after standing a few hours, becoming covered with a mass of brownish froth resembling yeast. Goodsir in 1842 discovered in this liquid in great abundance a vegetable organism which is now commonly known as the *Sarcina ventriculi*, called by Robin *Merismopœdia ventriculi.* The sarcinæ are distinguished by their oblong or square form and by their being divided by lines into four equal squares, so that they resemble a package tied with a cord, as the name sarcina denotes. The sarcinæ are now classed with the bacteria or schizomycetes. The vomited liquid containing sarcinæ may also contain the yeast-fungus in abundance. The liquid is sour, according to Budd, from the presence of acetic acid, and the vegetable productions are incidental to a process of fermentation attended with the formation of this acid, together with the evolution of carbonic acid. There does not appear to be ground for the belief that the presence of the vegetable productions is the source of disorder. Their production within the stomach appears to be owing to an undue retention of its contents from pyloric obstruction or other causes. The treatment, therefore, relates not alone to the circumstances immediately involved in their production, but to the morbid conditions giving rise to these circumstances. Remedies, however, designed to arrest the fermentative process are indicated; and of these one of the most efficient is the bisulphite of soda. These remedies are valuable as palliatives whenever, from defective power of digestion, fermentative processes in the ingesta take place. Small doses of carbolic acid, salicin, salicylic acid, and gaultheria are other efficient remedies. The value of these remedies in cases of dyspepsia depends on their power in arresting fermentation. Washing out the stomach by the siphon process is especially useful in cases characterized by fermentation of the gastric ingesta.

Dyspeptics frequently derive advantage from the use of mineral-spring waters, especially if connected with a sojourn under agreeable circumstances at a pleasant watering-place. Much of the advantage, doubtless, is due to change of habits and relief from care, together with the moral influence of the expectation of improvement. Of the various mineral waters, those from the gaseous and chalybeate springs are best suited to cases of dyspepsia. Cold sea-bathing and the invigorating influence of a bracing sea-air are frequently useful. The hygienic discipline of the hydropathic system is undoubtedly useful in some cases, the benefit being due probably in part to a physical, but more to a moral, effect. There is perhaps no affection in which the anticipation of improvement from the employment of certain measures contributes to their efficacy more than in dyspepsia.

Polyphagia, Malacia and Pica, Polydipsia, and Dipsomania.

These names express morbid conditions which relate to the ingestion of solids or liquids—conditions which severally do not constitute individual affections, but which may be the most prominent, or perhaps the chief, ailments.

Polyphagia—or, as it is more commonly called, *bulimia*—denotes a craving for food beyond the wants of the system. As thus defined, these terms are not correctly applied to the greatly augmented appetite felt frequently during convalescence from fevers and other acute diseases, or when from any cause the system for a time has been deprived of alimentary supplies adequate to its needs. The inordinate desire for food under these circumstances is an expression of the requirements of the system, and the ability to digest food may be proportionately augmented—a fact which illustrates in a striking manner the connection existing between nutrition and the function of digestion. True bulimia does not exemplify this physiological connection, the amount of food craved far exceeding the requirements for nutrition. Bulimia may be another name for gluttony. The love of eating may be cultivated to such an extent that little else is thought of, and persons who fall into this habit may be said to live to eat, rather than to eat to live. The observation of every one will furnish examples of this kind. Habits of gluttony may lead to dyspepsia, which, by interrupting further indulgence, proves conservative, or, if the digestive function continue active, they may lead to obesity, fatty degeneration of the heart or other organs, and to various affections. Voracious eating characterizes certain cases of mental derangement. It may be a symptom of disease attended with progressive waste; as, for example, diabetes. In a more marked and rare form the craving for food greatly exceeds that under the circumstances just named. Curious instances have been reported in which the morbid appetite appeared to be insatiable, all kinds of food—raw meat, candles, etc.—being eaten in some cases with avidity and in enormous quantity. It is difficult to explain the morbid appetite in these instances. Recovery from this condition is to be expected. The indications are to regulate the diet as far as possible, to establish the general health by hygienic measures, to correct any disorder of digestion which may exist, and to palliate the excessive craving for food by opium, or sometimes by nauseant remedies. Swallowing pieces of ice has been found effective as a palliative measure.

Malacia and *pica* are terms applied to perversions of appetite; that is, to a morbid craving for particular substances. The terms are commonly used as synonyms, but strictly they denote different kinds of perversion. Malacia denotes a morbid craving for certain articles of food or articles which are not devoid of nutrition, whereas pica denotes a desire for innutritious substances. The craving for strange kinds of food sometimes attending pregnancy, and commonly known as "longings," is sufficiently familiar. Similar eccentricities of the appetite are sometimes observed in connection with hysteria, and they may be due in some cases to that desire to become objects of wonder or interest which weak-minded hysterical women sometimes present. Unnatural and disgusting perversions of the appetite are among the symptoms occasionally met with in cases of insanity. Perversions falling under the head of pica are sufficiently common. Innutritious substances frequently craved are charcoal, chalk, slate, and certain kinds of earth. The habit of eating these substances is sometimes carried to a great extent. In the case of a young woman seized shortly after marriage with epidemic dysentery, which proved fatal, the dejections contained an enormous amount of a black substance which on investigation was found to be from a species of slate-stone which she was accustomed to eat daily in excessive quantity. In some cases of pica the articles are at first taken with the idea of improving the complexion, and in this way the habit is formed; but in other cases a morbid uneasiness in the stomach leads to their use. The appetite is chiefly confined to females, especially young girls, and is generally associated with anæmia or chlorosis. The measures of treatment are to be addressed to the associated disorders, the continued indulgence of the perverted appetite being interdicted.

Dirt-eating, as it is vulgarly called (*chthonophagia*), is a morbid habit which heretofore prevailed to a considerable extent among the plantation negroes of the Southern States. It was described by John Hunter as prevailing among the negroes of the West Indies. The kind of earth selected is a loam or clay. Duncan gives the following account of this form of pica: "A very common disease among negroes on plantations in this part of the country (Louisiana) is a *state of anæmia* very often attributed, and perhaps justly, to the pernicious habit of *dirt-eating*. On examining negroes on a plantation a medical man is surprised to meet with so many of these cases. Almost every large plantation has three or four, and sometimes more of them. . . .

With many, no doubt, dirt-eating is a symptom only of a diseased condition of the digestive organs and of the system generally. With them dirt-eating proceeds from the same propensity which leads white females to resort to chalk, magnesia, etc. in order to relieve a disordered acrid condition of the stomach. This condition of system is often, in my opinion, produced by a deficiency of suitable nutriment."[1]

Polydipsia is a term used to denote a condition characterized by an excessive craving for liquids. Water is drunk in a very large or enormous quantity, amounting sometimes to several gallons in the twenty-four hours. In some cases the patient experiences constant or frequently recurring thirst, associated with dryness of the tongue and fauces. There is inability to sleep, except for short periods, in consequence of the urgency of the desire for drink. The quantity of urine is proportionately great, so that this condition is also denoted by a term expressing the latter fact—namely, *diabetes insipidus*. The urine voided is of low specific gravity, presenting a clear, limpid appearance like pure water, and containing neither sugar nor any abnormal constituent. In other respects the health may not be notably disordered. The appetite and digestion may be unimpaired, the functions generally are well performed, and the body does not waste. This condition at first suggests to the physician the probable existence of diabetes mellitus, but an examination of the urine leads to the exclusion of the latter disease. The condition may occur in early life or become developed at any age. It has been observed to occur as an intermittent malady, but in general it continues for an indefinite period or during life. It does not appear to lead to any disease, and is serious in itself only on account of the annoyance and debility which it occasions. Its pathological character, with our present knowledge, cannot be satisfactorily explained. Existing to the extent just described, it is so rare as to belong among the curiosities of clinical experience. In our ignorance of the pathology there are no rational indications for treatment, and experience has not furnished any reliable means of cure. Opium, valerian, camphor, and other antispasmodics have been found to diminish the thirst temporarily, and some benefit has been derived from the use of various tonic remedies.

Dipsomania is a term sometimes used to denote the peculiar delirium arising from the abuse of alcohol, but it is commonly applied to an uncontrollable desire for alcoholic drinks. A craving for this class of stimulants is sometimes developed in cases of dyspepsia, and may prove a source of intemperate habits. This fact has been already referred to. It is not to be lost sight of in judging of the propriety of prescribing or sanctioning the use of alcoholic stimulants in dyspeptic cases. A morbid craving for alcohol in some form is an element of intemperate habits whatever may have been their source. The habitual drunkard feels a desire for continued indulgence, which, with an impaired power of self-control and lessened self-respect resulting from intemperance, he is after a time wholly unable to resist. It is useless under these circumstances to attempt to reason him into temperance; he may appreciate the force of all the arguments which are presented, but he lacks moral strength enough to govern an acquired appetite which represents a morbid condition as much as the delusions of insanity. Reason and persuasion, however, may be effective before this condition of dipsomania is reached; that is, they may prevent this condition by leading to abstinence from alcoholic stimulants. Dipsomania is to be treated as a disease of body and mind. The cure is to be effected by withdrawing the patient from the use of alcohol in any form, and by the employment of medicinal and hygienic measures to invigorate the physical and mental faculties. The treatment must be continued long enough for the cessation of the morbid craving for stimulants and for the development of moral strength sufficient to enable the patient to adhere to a course of total abstinence, in which alone consists safety. It is evident that successful management is rarely practicable, except by means of institutions in which the patient is under the same restrictions as in insane asylums. In fact, dipsomania is to be treated as a form of mental derangement.

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[1] Fenner's *Southern Medical Reports*, vol. i.

There is a species of dipsomania which is constitutional and congenital. The desire for stimulants is paroxysmal, and an irresistible craving is developed by any indulgence, however small. Persons with this idiosyncrasy lose their power of self-control as soon as they feel the influence of alcohol. The only protection in such cases is in rigid abstinence.

Connected with the subject of dipsomania are questions pertaining to the causes of intemperance and the means of its prevention which are of the utmost importance to the welfare of mankind, but which do not properly fall within the scope of this work. The morbid effects of alcohol upon the system, aside from the development of dipsomania, will be referred to in connection with different individual diseases, especially those affecting the nervous system.

Gastrorrhagia.

Hemorrhage into the stomach and vomiting of blood are denoted by the term *hæmatemesis*. The term *gastrorrhagia* is in uniformity with the rule of nomenclature by which a hemorrhage and its situation are expressed, and is therefore to be preferred. Moreover, the term gastrorrhagia applies to all cases of gastric hemorrhage, whether vomiting take place or not, the blood sometimes not being expelled by the mouth, but passing into the intestinal canal and being evacuated from the bowels. In the great majority of the cases in which hemorrhage takes place into the stomach, blood is vomited, and, more or less blood also passing into the intestinal canal, the stools present appearances to be described under the head of Enterorrhagia.

When blood is expelled from the mouth the first point is to ascertain the source of the hemorrhage. It may be from the mouth or posterior nares. Coming directly from either of these sources, it is traced without difficulty; but if blood be swallowed and vomiting occur, it may sometimes be a question whether gastric hemorrhage exist or not. In cases of bronchorrhagia blood may be swallowed and vomited. In a child too young to expectorate bronchial hemorrhage might thus be supposed to be gastric. Although instances both of bronchorrhagia and of gastrorrhagia in young children have been recorded, such an occurrence is very rare. The rupture of an aneurism into the pharynx or œsophagus is another source of hemorrhage which may appear to be gastric; but in general the question is whether the blood come directly from the stomach or the air-passages. The points involved in this differential diagnosis have been already presented in treating of bronchorrhagia.[1] Blood which has been vomited in most cases presents a dark, grumous appearance. Exceptionally it is florid, like arterial blood. It then comes from an artery, and is vomited as soon as it accumulates within the stomach.

Gastric hemorrhage in the vast majority of cases has no claim to be regarded as an individual affection. It may be a symptom of carcinoma of the stomach or of gastric ulcer, and as such has been already considered. It is an effect of portal congestion in cases of cirrhosis of the liver and of obstruction of the vena portæ from a thrombus or the pressure of a tumor. It may be caused by aneurisms or by varices of the gastric blood-vessels. It coexists with hemorrhage in other situations in cases of purpura hæmorrhagica and scorbutus. It occurs in cases of acute gastritis, in acute yellow atrophy of the liver, and in relapsing fever. It is a prominent event in the natural history of yellow fever, the so-called black vomit in this disease denoting neither more nor less than gastrorrhagia. It may occur in profound anæmias and in malaria. It may occur in place of the menses in cases of amenorrhœa. Instances in which gastrorrhagia is vicarious are by no means common; in the majority of the cases in which it follows suppression of the menses it proceeds from disease of the stomach; yet that it may take the place of the menses must be admitted. The cessation of habitual hemorrhage in some other situation, the arrest of purulent discharges of long standing, and the healing of old ulcers have been supposed to stand occasionally in a causative relation to gastrorrhagia, but this supposition does not rest on adequate clinical proof. Hemorrhage sometimes occurs from the stomach, as from the bronchial tubes, the Schneiderian membrane, and other situations, without any apparent pathological connections, neither following nor preceding any appreciable morbid conditions. It is then to be considered as idiopathic or as constituting an individual affection. Several cases have come under my observation, one of which, with the hemorrhage extremely profuse, I have reported in another work.[1] Examinations after death have shown the absence of ulceration, erosions, or of any appreciable solution of continuity, and the hemorrhage is then to be attributed to diapedesis. Hemorrhage from the stomach is an occasional event in pregnancy. It may be caused by a contusion received on the epigastrium. Irrespective of its occurrence as a symptom of gastritis, yellow fever, acute yellow atrophy of the liver, purpura hæmorrhagica, and scorbutus, gastrorrhagia occurs oftener in women than in men. It is a rare event in new-born children, occurring a few hours after birth, the blood being vomited and also passed from the bowels (melæna neonatorum). It proves fatal in some of these cases.

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[1] Vide p. 261.

The PROGNOSIS in cases of gastrorrhagia will depend upon its pathological import. The prognosis is of course unfavorable if it be associated with gastric carcinoma or yellow atrophy or cirrhosis of the liver, although under these circumstances death is rarely an immediate effect of the loss of blood. If, however, the hemorrhage be vicarious or not connected with any appreciable morbid conditions, the prognosis, as a rule, is favorable. The loss of blood in proportion as it is considerable or large induces debility, anæmia, and may prove the direct cause of death. The amount and rapidity of the hemorrhage may be such as to induce sudden death from syncope. On the other hand, a vicarious hemorrhage if moderate is not only without danger, but may conduce to the welfare of the system so long as the menstrual discharge remains suppressed. In determining the amount of hemorrhage it is to be borne in mind that the quantity of blood vomited is not always a criterion. The blood may accumulate and coagulate within the stomach without being vomited. The hemorrhage may be sufficient to destroy life, although the quantity vomited be not large. Death may take place in such cases as in cases of concealed uterine hemorrhage after labor. Evidence of hemorrhage being out of proportion to the amount of blood vomited is afforded by dulness on percussion over the distended stomach and symptoms denoting loss of blood—namely, feebleness of the pulse, coldness of the surface, pallor, faintness, etc. The blood thus accumulating within the stomach if not vomited passes into the intestines and is discharged by stool (melæna). Hemorrhage into the stomach has been known to occur and death to take place from the loss of blood without any vomiting.

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[1] Vide *Clinical Medicine*, p. 253.

The TREATMENT of gastrorrhagia has been already referred to in connection with gastric ulcer. The general principles of treatment, having reference to the arrest of hemorrhage and the prevention of its recurrence, are the same under whatever circumstances it occurs. With reference to these objects perfect rest of body and mind is important. If the hemorrhage be profuse or if it recur, rest of the stomach should be secured by withholding from this organ all nutriment and nourishing the patient exclusively by rectal alimentation. Alcoholics if indicated are to be given hypodermically or *per enema.* Opium is useful by quieting the peristaltic movements of the stomach. It should be given hypodermically. The direct refrigerant effect of iced water taken in small quantities at a time, or of swallowing small pieces of ice, is useful. In urgent cases, as a means of arresting the hemorrhage, ice may be applied to the epigastrium. In less urgent cases revulsive measures may be relied upon—namely, sinapisms and dry cups applied in different situations over the abdomen. Hot, stimulating foot-baths are useful by way of revulsion. Hæmostatic remedies, such as ergot, the acetate of lead, tannic acid, etc., if introduced into the stomach, are liable to excite vomiting and thereby do harm. Ergotin may be given by hypodermic injection. Purgatives are contraindicated.

Experience has shown that transfusion is likely to cause renewed hemorrhage, so that this procedure should be resorted to in gastrorrhagia only when there is imminent danger of a fatal termination from the loss of blood; and then only a small quantity should be transfused. The infusion of physiological salt solution is safer than the transfusion of blood, and is about as effective.

These measures are to be employed in individual cases according to the urgency with which the arrest or the prevention of hemorrhage is indicated. If there be ground for regarding the hemorrhage as vicarious, interference will be required only to keep it within proper limits. This statement will also apply to hemorrhage occurring in pregnancy.

The treatment in most cases of gastrorrhagia of course embraces other measures than those having reference to the arrest and the prevention of the hemorrhage—namely, measures addressed to the morbid conditions with which the hemorrhage is associated. The latter are considered under other heads. So also measures called for after the occurrence of gastric hemorrhage, having reference to debility and anæmia dependent on the loss of blood, need not be here considered. After a considerable hemorrhage the stomach, assuming the non-existence of cancer or ulcer, is enfeebled, and the liability to a recurrence of the hemorrhage is to be kept in view. Careful regulation of the diet, quietude of the body, and the avoidance of all disturbing agencies are important during convalescence. Although profuse or repeated hemorrhages involve more or less immediate danger, patients may recover even when the loss of blood occasions prolonged syncope and extreme anæmia. Striking illustrations of this fact have come under my observation.

Gastric hemorrhage is sometimes simulated by hysterical females and by malingerers among soldiers and prisoners. Blood obtained from animals, and even human blood, may be taken into the stomach, and vomiting excited by various means. This deception may be suspected in the case of a young woman who manifests a morbid disposition to impose on the credulity of those around her as regards her ailments, when the symptoms do not denote loss of blood, when there are no grounds, aside from hemorrhage, to suspect gastric ulcer, and when the menses are not suppressed. Among soldiers and prisoners it is to be suspected when the usual effects of hemorrhage are not apparent, and when the morbid conditions with which gastric hemorrhage is usually associated are wanting. The microscope may be of use in the detection of this kind of deception. Bennett cites a case in which a specimen of blood which it was pretended had been vomited was found to present the characteristic red corpuscles of the blood of a bird.

Leube mentions, as an occasional sequel of gastrorrhagia, double and permanent amaurosis, pallor of the papilla and attenuation of the retinal arteries being the only appearances revealed by the ophthalmoscope. That the amaurosis is simply an effect of anæmia is disproved by the fact that it persists after the anæmic condition disappears. He therefore infers the existence of some connection between the nerves and vessels of the stomach and certain parts of the brain—an inference sustained by the occurrence of gastric hemorrhage after injuries to certain parts of the brain.

Inanition.

In connection with the functional affections of the stomach, I shall offer a few remarks on a pathological condition which is not perhaps entitled to rank in the nosological catalogue as an individual disease, but which is an element of all diseases compromising alimentation and digestion. The pathological condition referred to is *inanition*. The importance of a due appreciation of this condition in medical practice renders it desirable to devote to it some attention, and it may with propriety be noticed in connection with the functional affections of the alimentary canal.

Health requires the assimilation of nutriment adequate in quantity and kind to the wants of nutrition. Every one is familiar with the mental and physical exhaustion felt when abstinence is protracted for only a few hours after the sense of hunger indicates the need of food. Every one knows that the deprivation of aliment for a period varying according to circumstances proves fatal. During the progress of starvation the body loses rapidly in weight, the fat disappears, the muscles diminish in size, exhaustion progressively increases, the heat of the body is lowered, vomiting and diarrhœa not infrequently occur, the mental powers are weakened, listlessness and hebetude are sometimes followed by delirium and coma, and, according to Chossat, if the deprivation of aliment be complete death takes place when one-fourth of the weight of the body at the time aliment was withheld has been lost. The mode of dying is a type of slow asthenia. It is worthy of note that during the progress of starvation hunger is not usually a prominent symptom. If felt for a time it is liable to disappear, and it may be followed by a loathing of food.

Inanition is a pathological condition entering into all diseases which interfere with the ingestion or the assimilation of aliment. The phenomena of inanition in cases of these diseases are combined with the symptoms belonging to the particular disease which exists. The inability of the system to support, resist, and overcome disease proceeds, in a greater or less degree, from this element. The immediate cause of death in many cases of disease proving fatal by slow asthenia is inanition; that is to say, starvation. These are facts of great practical importance, and they are not sufficiently appreciated by many practitioners of medicine.

An important practical bearing of these facts relates to alimentation in the management of diseases. Until recently for many years not only was the importance of alimentation not fully recognized in medical practice, but the dietetic treatment of most diseases contributed to inanition. The writings of Graves led to the employment of alimentation in fevers. It is not less important in all other diseases which tend to destroy life by asthenia.

Patients may be starved to death in other diseases as well as in fevers. As a practical rule, it is an object in any disease in which the danger lies in the direction of asthenia to meet, as far as practicable by alimentary supplies, the wants of nutrition. Harm may arise from the ingestion of food beyond the powers of digestion, but no harm can arise from an undue assimilation of food; on the contrary, the greater the assimilation the less the danger from inanition superadded to the disease. Let it be borne in mind that in all diseases tending to destroy life by asthenia there is danger from inanition, but never from hypernutrition! In the language of Chossat, "Inanition is a cause of death which marches silently in front with every disease in which alimentation falls below the normal standard. It reaches its natural termination sometimes sooner and sometimes later than the disease which it covertly accompanies, and it may supersede the disease of which at first it was merely an incidental element."

Alimentation is important in diseases not attended with immediate danger. In proportion as the assimilation of food can be made to approximate to the normal standard, the phenomena of inanition are forestalled, the vital powers are better enabled to tolerate and overcome the disease, the duration of the disease may be shortened, convalescence is hastened, and the recovery is more likely to be complete. As regards the selection of food and the modes of its preparation in different diseases, in addition to dietetic rules based on general experience, our knowledge of alimentary principles, etc., reliance may be placed upon the desires and tastes of the patient; but the absence of appetite is not to be a guide as regards alimentation. Loss of appetite is one of the symptoms of inanition. The wants of the system are not in disease, as in health, always expressed by hunger. The state of the mind dependent on the morbid condition of the nervous system, the coatings on the tongue, etc. interfere with the desire for food and the sense of taste. The practitioner is to exercise his judgment and tact in securing, as far as practicable, the assimilation of an adequate amount of nutriment, either with or without the co-operation of desire and taste on the part of the patient, and it is often better to incur risk of exceeding rather than falling below the quantity of food which the wants of the system seem to require. It may be laid down as a principle applicable to the management of most acute diseases that the assimilation of food to the fullest practicable extent is important; and it is a maxim of conservative medicine that under all circumstances a chronic affection is less likely to be prolonged, serious lesions of structure are less liable to take place, and a fatal termination is postponed, in proportion as the vital powers are maintained by a nutritious diet.[1]

Innutrition is not infrequently involved in the causation of diseases. The connection between scarcity of food and the prevalence of typhus has been repeatedly observed in epidemics in Ireland. The same connection in individual cases and families is observed frequently everywhere. Scorbutus is caused by a deficiency of certain alimentary principles or the want of a sufficient variety in food. Insufficient nourishment, which is sometimes involuntary and sometimes voluntary, is supposed to favor the development of phthisis in persons having the tuberculous predisposition. This cause may co-operate with other causes in giving rise to various diseases, whether epidemic or sporadic; and the ability of the system to bear up under disease is impaired in proportion as the powers of life are enfeebled by previous defective alimentation.

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[1] Vide "Essays on Conservative Medicine," by the author, *American Journal of the Medical Sciences*, January and October, 1863. Vide, also, essay on "Alimentation in Diseases," *New York Med. Journal*, 1867, and the work entitled *Essays on Conservative Medicine and Kindred Topics.*

Want of success in the treatment of diseases in hospitals, and often in private practice, especially among the poorer classes, is attributable in no small measure to the want of adequate alimentation.

Gastralgia.

Gastralgia, called frequently gastrodynia, is a neuralgic affection characterized by pain, more or less intense, referred to the region of the stomach. Pain is a symptom of gastritis, carcinoma of the stomach, and gastric ulcer; it also enters into the clinical history of dyspepsia, especially the variety called acute dyspepsia, and a peculiar character of pain constitutes the dyspeptic symptom called cardialgia or heartburn. But, considered as characterizing a distinct functional affection, the pain is not necessarily incidental to inflammation or any appreciable lesion, nor is it always, although frequently, associated with dyspeptic disorder; in short, gastralgia exists when pain and other circumstances denote an affection of the same pathological character as neuralgia in other situations.

Gastralgia is presented in practice in two forms: in one form a severe attack of pain occurs, and if not relieved by remedies it continues for several hours or even days. The pain is often excruciating, subduing the strongest resolution. The patient writhes and groans with intense suffering. A sense of constriction frequently accompanies the pain. Vomiting may occur, but this is rare. Generally, tenderness over the epigastrium is wanting, and some relief may be afforded by pressure. The paroxysm is not attended with pyrexia, and the skin may be bathed in perspiration from the intensity of pain. These attacks are generally attributed to spasm or cramp, and the affection has been called colic of the stomach. It is difficult to say how much of the suffering is due to spasmodic contraction and how much to neuralgic pain. Practically, it is not important to make this discrimination. This form of gastralgia may be distinguished as *acute*.

Such attacks in some persons are produced by certain ingesta. Strawberries, for example, have been known to prove in this way poisonous, and I have met with a patient who had always an attack after eating honey. He came under my observation when suffering from an attack thus produced. These, like other idiosyncrasies, are inexplicable. Attacks appear to be sometimes attributable to exposure to cold and to fatigue from over-exertion.

This form of gastralgia is readily discriminated from acute gastritis. The pain may be much more intense than in cases of the latter disease. As a rule, the tenderness over the epigastrium which belongs to the history of gastritis is wanting. Vomiting may be wanting, and if present is not as prominent a symptom as in gastritis. The absence of pyrexia is an important point in the exclusion of gastritis. The passage of gall-stones, or hepatic colic, offers many symptoms in common, and the discrimination is not at once readily made. The points involved in the differential diagnosis will be considered presently in treating of the passage of gall-stones. In the diagnosis of gastralgia attacks of angina pectoris are to be excluded. (Vide p. 360.)

Although, as a rule, acute gastralgia occurs irrespective of inflammation or any appreciable lesion, it is sometimes associated with either gastric or duodenal ulcer. The existence of ulceration is liable to be overlooked if the diagnostic symptoms be not marked. This statement is especially true of duodenal ulcer. In several cases which have come under my observation profuse hemorrhage has followed attacks of gastralgia.

Attacks of severe gastralgia, recurring after interruptions of variable duration and accompanied by vomiting, are described by Charcot, under the name *gastric crises*, as associated with locomotor ataxia. They may be associated with other affections of the spinal cord.[1]

This form of gastralgia is to be treated by opiates carried to the extent of procuring relief. A quarter or a half of a grain of a salt of morphia may be sprinkled on the tongue, and this dose repeated, if necessary, after the lapse of from half an hour to an hour, until the pain is relieved; or the same course may be pursued with other forms of opiate. If the opiate be rejected from the stomach, it may be given *per enema*. The hypodermic mode of administration is well suited to this affection, as to all affections in which it is desired to produce quickly and efficiently an anodyne effect. This plan of treatment is not to be deferred for the operation of a cathartic or an emetic even if the attack be attributable to dietetic cause. A sinapism and fomentations to the epigastrium are useful as auxiliary measures. An alcoholic stimulant may be added with advantage if not contraindicated by vomiting, especially if the pulse be feeble and the surface cold. Rest, regulated diet, and a mild cathartic or laxative, provided the bowels do not act spontaneously, will constitute the subsequent treatment.

Although the suffering is as great as in any other affection, there is little or no danger. It may be doubted whether this affection alone ever prove fatal; and the practitioner may count with confidence on procuring speedy relief by means of the plan of treatment just stated. The affection, therefore, is one of those which exemplify in a striking manner the resources of medicine.

Chronic gastralgia is another form of the affection. The pain is either limited to paroxysms which recur more or less frequently, or it is habitual, with exacerbations at variable intervals. The pain varies much in different cases as regards intensity, and also in different paroxysms or exacerbations in the same case. It may have so little intensity as to occasion only annoyance, or it may be so severe as to give rise to extreme suffering. The character of the pain is described by patients as burning, lancinating, or gnawing. Frequently the pain extends from the epigastrium in different directions—namely, to the back, into the chest, and laterally into the right and the left hypochondrium.

With the affection in some cases are associated dyspeptic ailments, and in other cases there is no disorder of digestion. Paroxysms or exacerbations sometimes appear to be provoked by the ingestion of food, but as a rule relief of pain is felt after eating. The desire for food is oftener increased than diminished. Generally, tenderness over the epigastrium is wanting, and pressure frequently affords relief. The decubitus during the suffering from pain is sometimes upon the belly. Constipation exists much oftener than looseness of the bowels. Flatulent distension of the stomach or intestine is sometimes associated. The pulse preserves its normal frequency and the temperature of the body is not increased. Patients who have suffered from the malady for some time are likely to become melancholic and hypochondriacal. I have known it to be associated with a tendency to suicide.

Gastralgia rarely if ever occurs before the age of puberty or in old age. It is generally considered that women, much more than men, are liable to it, but of 39 cases analyzed by Valleix only 20 were females. It affects those of good constitution and the robust as well as the feeble and delicate. Persons of sedentary habits are more likely to be affected than those engaged in active pursuits. Prolonged mental depression has been supposed to favor its occurrence. It appears in some cases to depend on the gouty diathesis. Like neuralgia in other situations, it may be an effect of malaria; and attacks of gastralgia may recur with the regularity of the paroxysms of intermittent fever. I have known gastralgia to take the place of the cold stage of the paroxysm, being followed by the hot and the sweating stage. Finally, causes which induce anæmia or lead to debility may be more or less involved in its production.

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[1] Vide article on "Gastric Ulcer" by Prof. Welch in the *System of Practical Medicine, by American Authors*, vol. ii. p. 517.

The DIAGNOSIS of the chronic form of gastralgia is to be based on certain distinctive characters—namely, the occurrence of pain, either in paroxysms or marked exacerbations; the relief frequently afforded by pressure and the ingestion of food; the persistence of appetite in many cases; and not infrequently the absence of disorder of digestion and of pyrexia. These points in the history are involved especially in the discrimination of gastralgia from subacute or chronic gastritis. The diagnosis involves the exclusion of gastric ulcer and cancer.[1] These affections are to be excluded by the absence of their diagnostic symptoms, irrespective of pain. In some cases of intercostal neuralgia pain is referred to the epigastrium. This affection is excluded by the absence of its diagnostic criterion—namely, tenderness on pressure over three points, posteriorly, laterally, and anteriorly.[2]

Chronic gastralgia is likely to be persisting. Its duration in different cases is extremely variable. In this respect it resembles other neuralgic affections. It may continue for many months, and it sometimes persists for many years. As regards prognosis, the worst to be apprehended is the persistence of the affection. It does not tend to destroy life either directly or by eventuating in some other more serious disease. It is not prudent for the practitioner to predict that the affection will end speedily or within any definite period, yet not infrequently it does not continue. In certain cases it is one of the affections most rebellious to therapeutical measures, but in other cases it is readily amenable to treatment.

In the TREATMENT of gastralgia clinical experience has abundantly shown certain measures formerly in vogue to be not only ineffectual, but prejudicial. This remark has reference to general and local bloodletting, emetics, mercurials, purgative remedies, and perhaps also vesication or other modes of active counter-irritation. Anodyne remedies are frequently called for to relieve pain. If the pain be severe, opium can hardly be dispensed with; but in this, as in other painful affections which are likely to be persisting, the liability to the formation of a habit of using opium is to be considered. Moreover, the use of opium, aside from the palliation of pain, is objectionable on account of its interference with the appetite and digestion, and in this way it may contribute to perpetuate the affection. Opium is therefore to be used with circumspection, and when practicable other palliative measures are to be substituted, such as belladonna, hyoscyamus, etc. Fomentations and embrocations containing opium, chloroform, aconite, etc. will sometimes succeed in affording relief. Cold applied to the epigastrium has been found effectual. Certain remedies appear in some cases to exert a curative influence. Bismuth is one of these. I have known large doses of this remedy to be promptly curative. Monneret advises it to be given to the extent of one, two, or three ounces daily. These enormous does are not required. Whatever curative power the remedy has will be exerted in doses of from a scruple to a drachm three or four times daily. Alum has been recommended in doses of from ten to twenty grains three or four times daily. Strychnia and nux vomica have been found useful, especially if the affection be associated with the formation of gas. A quarter of a grain of the extract of nux vomica may be given three or four times daily. Other remedies which may be employed with a view to a curative effect are the nitrate of silver and the subcarbonate of iron (precipitated carbonate or sesquioxide), given in drachm doses, and the iodide of potassium, as in other neuralgic affections. Another remedy is the purified oxide of manganese in doses of from five grains to half a drachm three times daily. Of curative remedies, in my experience, quinia in full doses is most likely to prove successful. The doses should be sufficient to produce cinchonism, which should be steadily maintained for about two weeks. If at the end of this time a curative effect be not exerted, it is useless to continue the remedy longer. Although it often succeeds, it not infrequently fails. After quinia, arsenic, given in small doses and continued without increase of dose, has been, in my experience, the most successful remedy. Salicin, a drachm or more given daily, is sometimes curative. In the hands of Leube, Beard and Rockwell, and others electricity has proved efficacious. The constant current is the form to be employed.

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[1] For the points involved in the differentiation from gastric ulcer, vide *System of Practical Medicine, by American Authors*, vol. ii. p. 516.

[2] Vide p. 183.

Measures of treatment other than the employment of direct curative remedies are of importance. Change of habits from those of sedentary to active life is of the first importance in certain cases. The moral influence of recreation, change of scene, etc. is often of more service than medicines. The diet should be nutritious. It should not be restricted. Alcoholic stimulants in moderation with meals may be advised for a time, exercising that reserve dictated by the danger of patients becoming addicted to their habitual use. Tonic remedies in most cases, continued steadily for a long period and varied from time to time, are advisable. The valerianate of zinc may be mentioned as a tonic remedy likely to prove useful. Preparations of iron are especially indicated if anæmia be present. Dyspeptic ailments which may or may not be associated are to receive appropriate treatment. In short, the object, aside from the employment of palliative and curative remedies, is to endeavor to place the digestive organs and the system in the best possible condition, and for this end hygienic and medicinal measures are to be adapted to the circumstances peculiar to individual cases.

CHAPTER V.

INFLAMMATORY DISEASES OF THE INTESTINE.

Anatomical Characters of Acute Dysentery.—Sporadic Dysentery.—Epidemic Dysentery.—Chronic Dysentery.—Inflammation and Perforation of the Cæcum.—Fecal Abscess.—Inflammation and Perforation of the Vermiform Appendix.—Colitis.—Proctitis.—Acute Enteritis.—Subacute and Chronic Enteritis.

Acute Dysentery.

THE term dysentery has long been in use to designate an inflammatory disease of the large intestine characterized by mucous and bloody dejections and by tenesmus. As a name for the disease it is not very distinctive, its etymology simply expressing intestinal difficulty; but it is not easy to substitute a term conformable to the nomenclature of inflammatory affections, as there is no anatomical name for the large intestine as a whole. The term dysentery has the advantage of not expressing anything erroneous or doubtful in regard to the pathological character of the disease. Acute and chronic dysentery claim separate consideration. Acute dysentery occurs as a sporadic and as an epidemic disease. This is a distinction of much clinical importance. After having considered the anatomical characters of sporadic and epidemic dysentery, these two forms of the disease will be treated of separately as regards clinical history, pathological character, etc.

ANATOMICAL CHARACTERS.—After death from acute dysentery the mucous membrane of the large intestine is in a condition either of simple muco-purulent or of diphtheritic inflammation. Anatomically, therefore, the disease is distinguished as simple inflammatory—or, as it is also called in accordance with German nomenclature, catarrhal dysentery—and as diphtheritic dysentery. Most of the cases of sporadic dysentery are of the simple inflammatory variety, which is the milder of the two varieties. The largest number of cases of diphtheritic dysentery belong to the epidemic form of the disease. The anatomical, however, cannot be used interchangeably with the clinical terms. Diphtheritic dysentery may occur sporadically, and epidemic dysentery usually includes a large number of cases of the simple inflammatory variety. There are, moreover, all grades of transition between the mildest cases of simple inflammatory dysentery and the most malignant cases of diphtheritic dysentery. It is not always possible during life to distinguish between simple and diphtheritic inflammation of the large intestine. For these and other reasons it is most convenient to treat of the clinical history of acute dysentery under the headings Sporadic and Epidemic Dysentery, but to describe the anatomical appearances first of simple and then of diphtheritic inflammation of the large intestine.

Dysentery in the *acute simple* (catarrhal) variety is a simple inflammation of the inner coats of the large intestine. The inflammatory process is usually more intense in certain situations than in others. The parts most involved are the cæcum, the hepatic and the splenic flexures of the colon, the sigmoid flexure and the rectum. These are the parts where the contents of the large intestine remain longest in contact with the mucous membrane. The inflammation often, although not invariably, increases in degree toward the lower end of the large intestine. The lower part of the ileum is also frequently involved in the inflammatory process.

The mucous membrane is reddened, softened, and swollen. The redness is usually in patches, and is most intense in the situations named. The color varies from light pink to dark, livid red. The redness is due chiefly to hyperæmia, as is evident from the arborescent appearance, especially marked when the intestine is examined by transmitted light. In addition to congestion, however, there are usually extravasations of blood in the mucous and submucous tissues in the form either of punctate or of diffuse ecchymoses. It may happen that a congested appearance is not evident after death, as may be the case in inflammation of other mucous membranes. The surface of the mucous membrane is covered with a layer of tenacious mucus, either colorless or of a yellowish or brownish-red hue from admixture of bile and blood. Sometimes the admixture of pus is sufficient to give an opaque, yellowish color to the mucus. The mucus contains, microscopically, pus-cells, red blood-corpuscles, and desquamated epithelial cells. Both the mucous membrane and the submucous tissue are swollen, the latter often in the greater degree. This swelling is due to congestion, together with the exudation of serum and of pus-cells. The solitary follicles are usually swollen, and they appear as grayish-white projections about the size of a pin's head, surrounded by a zone of congestion. They may become as large as a small pea. Sometimes the inflammation seems to be chiefly in the follicles. The disease is then sometimes called *follicular dysentery*. If there have been repeated attacks of acute dysentery, or if the acute passes into a chronic stage, the mucous membrane becomes more or less pigmented. Points of dark pigment appear in the centre of the solitary and of the agminated follicles. In Peyer's patches this pigmentation gives rise to the so-called shaven-beard appearance. The pigment is chiefly in the form of brown granules in the lymphoid cells. Diffuse patches and streaks of slate-colored or brownish pigmentation may appear outside of the follicles in the mucous membrane, especially in the large intestine. Small follicular ulcers, due to softening and breaking down of the follicles, are often observed in acute dysentery, especially in the follicular variety. The peritoneum over the parts most inflamed may be normal or it may be coated with a layer of fibrin.

Microscopical examination shows the small veins and capillaries of the mucous membrane and of the submucous tissue, particularly of its upper layers, distended with blood. In some places there may be only hyperæmia, but as a rule there is distinct evidence of inflammation. This is afforded by the presence of pus-cells. The pus-cells are particularly abundant near the muscularis mucosæ (Brücke's muscle), just below the glands of Lieberkühn, where there is normally a rich network of blood-vessels. The pus-cells are frequently observed in swarms around the small veins. There are also pus-cells in the mucous membrane between the glands of Lieberkühn; but as the connective tissue in this situation is of the variety called adenoid, which normally is densely infiltrated with lymphoid cells, it is difficult to determine here the presence of a moderate number of pus-cells, as these anatomically resemble the lymphoid cells. If the pus-cells accumulate in large number, they press apart and distort the glands of Lieberkühn. The enlargement of the solitary follicles is due to an increase in the number of lymphoid elements. These infiltrate the surrounding submucous tissue. In addition to the emigration of white corpuscles more or less red corpuscles escape by diapedesis. The larger hemorrhages are the result of rupture of the vessel-walls (rhexis). The spaces between the fibres of the submucous tissue are distended by the presence of serum (œdema). As already said, the accumulation of lymphoid cells may be sufficient to compress the tubules of Lieberkühn. In this way the tubules may be obstructed in certain parts. They may undergo cystic dilatation below the seat of obstruction. Most of the epithelial cells lining the tubules are distended with mucus, and they appear as so-called goblet or caliciform cells—forms which are normally very frequent in the large intestine. The fate of the columnar epithelium covering the inner surface of the mucous membrane is uncertain. As under normal circumstances this epithelium soon desquamates after death, its absence from its normal place in inflammation cannot be considered evidence of its separation during life. It is not probable that extensive desquamation of the epithelium occurs during life.

The description which has been given of the histological changes in acute simple inflammation of the intestine in dysentery is applicable to the same variety of inflammation in other conditions, as in acute enteritis. It is especially the involvement of the lower part of the large intestine in the inflammatory process which characterizes acute dysentery. As is evident from the description, there are no changes in acute simple dysentery which may not undergo resolution, with the exception of the follicular ulcerations, which are by no means always present, and which may readily cicatrize. More or less pigmentation and atrophy of the mucous membrane usually remains after an attack of dysentery.

Some writers describe a more severe form of purulent inflammation than that to which the name simple inflammatory dysentery has been given. In this, excessive suppuration occurs and ulcers are formed by destruction of the superficial layers of the mucous membrane. Follicular ulcers are present at the same time.

Dysentery with *diphtheritic* or *pseudo-membranous exudation* is a much graver disease than the simple inflammatory variety. It is called diphtheritic, not on account of any connection with diphtheria of the pharynx, but in consequence of the resemblance between its lesions and those of diphtheritic inflammation of the throat. It is also called fibrinous (croupous) dysentery, and corresponds in its more severe manifestations to the putrid and gangrenous dysentery of the old writers. Diphtheritic inflammation of the large intestine is characterized by the presence of an exudation containing fibrin, blood, and pus-cells upon the surface and in the substance of the mucous membrane, combined with sloughing and consequent loss of substance of the mucous membrane. A large number of cases of epidemic dysentery are of this type, but it may also occur sporadically.

According to Virchow, diphtheritic dysentery is always preceded by simple inflammation of the large intestine. Others believe that the inflammation may be diphtheritic from the onset. However this may be, diphtheritic inflammation is very frequently combined with simple inflammation. The morbid changes are usually, as in simple inflammation, most marked in the cæcum, the flexures, and the rectum, and they affect by preference the projecting folds of the mucous membrane. The inflammation is often most intense in the sigmoid flexure and the rectum, and it may be confined to these parts. Sometimes the rectum escapes. In other cases the inflammation is uninterrupted from the beginning to the end of the large intestine. In the lower part of the ileum simple inflammation is frequently present, but there is rarely diphtheritic inflammation. In the early stages of the diphtheritic process the inner surface of the large intestine presents here and there patches of grayish-red, yellow, or variously discolored material, of varying thickness and extent, usually soft, but sometimes firm. This pseudo-membranous deposit consists of an exudation of fibrin, pus, and blood involving the upper layers of the mucous membrane. It extends in depth and destroys the vitality of the invaded tissue. The mucous membrane and often the subjacent tissues slough. In this way the so-called diphtheritic ulcers are produced, which may extend only in part through the mucous membrane, or they may expose the submucous tissue and even the muscular coat of the intestine. By further extension of the sloughing process the wall of the intestine may be perforated and a fatal peritonitis induced. This occurrence is infrequent. The diphtheritic ulcers are irregular in shape, their walls being bevelled or abrupt, in some cases overhanging. Sometimes little openings appear in the bed of the ulcer, which lead into dilated tubules of Lieberkühn. The ulcers may be so extensive that the undestroyed mucous membrane appears in the form of scattered islands rising from the surrounding depressions. The mucous membrane may slough away in little shreds or in larger coherent masses, sometimes several inches in extent. In advanced cases the inner surface of the intestine —with the ulcerations, the pseudo-membranous deposits, the ridges of mucous membrane, the partially detached sloughs, all of various colors and consistence —presents a peculiarly variegated appearance. Enlargement of the follicles and follicular ulcerations may likewise be present. The submucous tissue, where preserved, is usually much swollen and infiltrated with blood and pus. Submucous abscesses may form and undermine the mucous membrane. The peritoneal covering of the inflamed portion of the intestine is congested and is frequently coated with fibrin. Thus, circumscribed and sometimes general peritonitis may occur, agglutinating the coils of intestine.

The microscopical examination shows sometimes distinctly fibrillated fibrin, enclosing red blood-corpuscles, pus, and epithelial cells, as a firm layer upon the surface of the mucous membrane, extending into the glands of Lieberkühn and infiltrating the substance of the mucous membrane. Frequently, however, the pultaceous deposits on the inner surface of the intestine present little evidence of structure, but consist of a mass of granular material, low vegetable organisms, and decomposed blood. In the sloughs of mucous membrane careful inspection may reveal traces of the glands of Lieberkühn. These glands, when preserved, often present constrictions and cystic dilatations. These cysts may invade the solitary follicles. In the submucous layer a network of fibrin appears in the dilated interstices of the connective tissue, especially in the lymphatic vessels. Hemorrhages and pus-cells are abundant. Bacteria are frequently observed in the coats of the intestine and in the pseudo-membranous deposits. It is difficult to assign to these and the other forms of low organisms observed here any pathological significance, as they are normally present in large number in the feces, and as decomposition rapidly attacks the sloughy masses in dysentery. Coagulation necrosis is probably an essential element in the pathology of diphtheritic dysentery, as of fibrinous inflammations of mucous membranes in general.

When the sloughing process involves the mucous membrane of most of the large intestine death is almost inevitable. Less extensive diphtheritic ulcerations may cicatrize. Granulation-tissue develops from the surface of the ulcer. (For the repair of ulcers, see Part I. p. 39.) The cicatrix is composed of dense fibrous tissue, covered with epithelium but without glandular elements. The cicatrix is usually puckered, and it causes some contraction of the surrounding tissue. Sometimes, but not frequently, stricture of the intestine, followed by grave and even fatal symptoms, is produced by the contraction of these cicatrices. The healing of these ulcers is slow and is attended by the symptoms of chronic dysentery.

Of changes in other organs of the body occasionally observed in diphtheritic dysentery, abscess of the liver deserves especial mention. This is a frequent complication in tropical climates. It is not frequent in this climate, but it seems to be oftener observed here than in Great Britain and on the continent of Europe. The abscesses are usually multiple. They are explained by the transportation of infectious emboli derived from thrombi in the mesenteric veins proceeding from the diseased intestine. The mesenteric glands are usually swollen, and they may suppurate. Pulmonary complications, such as pneumonitis and œdema, are frequent. Parenchymatous degeneration of the kidney and occasionally acute diffuse nephritis occur.[1]

The foregoing account of the anatomical characters is of course descriptive of appearances observed in fatal cases of sporadic and epidemic dysentery. It is to be added that in either form the disease is not infrequently so mild as to involve no danger, and hence in mild cases the opportunity of inspecting the parts after death is not offered. There are but few diseases in the nosology which offer a wider contrast than the mildest cases of sporadic and the gravest cases of epidemic dysentery.

Sporadic Dysentery.

CLINICAL HISTORY.—Dysentery is generally preceded by simple diarrhœa; that is, more or less frequent and loose dejections of a feculent character.

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[1] An exhaustive consideration of the pathology and of other subjects pertaining to the natural history of diarrhœa and dysentery is given by Woodward in the second medical volume of the *Medical and Surgical History of the War of the Rebellion*, Washington, 1879.

Of 33 recorded cases which I have analyzed with reference to this point, these dejections preceded the characteristic dysenteric evacuations in 30. The latter appeared after the former had existed for a period varying from twenty-four hours to seven days.[1] With this prodromic diarrhœa there are colic pains, the appetite is impaired, and there is a general feeling of malaise. A pronounced chill rarely occurs, and even chilly sensations are not common.

The various ingredients which may enter into the composition of dysenteric stools are—feculent matter, mucus, blood, pus, serum, pseudo-membranes, necrotic tissue, bacteria together with other low organisms, and undigested aliment. According to the preponderance of one or more of these substances the stools are described as mucous, muco-purulent, muco-sanguineous, sero-sanguineous, sanguineo-purulent, gangrenous, bloody, or purulent. The presence of pseudomembranes and of necrotic portions of the intestinal coats is characteristic of the diphtheritic form of inflammation. The other ingredients are common to both varieties of inflammation. The development of the disease is denoted by characteristic evacuations consisting of mucus, with which more or less blood is usually commingled. The quantity passed at each act of defecation is generally small, but the act is repeated at short intervals, slight evacuations often taking place every hour or two, and sometimes a few moments only intervening. The mucus is discharged in the form of clumps of a yellowish or reddish glairy material, sometimes without feces and sometimes around balls of feces. The quantity of mucus expelled in some cases is abundant, and forms a jelly-like mass not unlike a collection of the rusty sputa of pneumonitis. Microscopical examination of the mucous discharges shows cells resembling the white blood-corpuscles (called often, in this situation, mucous corpuscles, but in no way differing from pus-cells), red blood-corpuscles, bacteria, and sometimes cylindrical and flattened epithelial cells. The mucus itself is characterized by its viscid nature, and by its precipitation with acetic acid in the form of threads and flakes of mucin insoluble in an excess of the acid. When the mucous or pus-corpuscles are present in large number the substance acquires an opaque whitish or yellow appearance, and is called muco-pus. The blood may give a diffuse or a streaky red color to the discharges. After the characteristic dysenteric stools appear, feculent matters are usually entirely absent. These may, however, appear, at intervals, especially in the form of hard balls of feces called scybala. When improvement begins feculent matters appear in the stools. In mild cases the discharges may remain mucous throughout the whole course of the disease. In severe cases of sporadic (and frequently in epidemic) dysentery the discharge of a sero-sanguinolent liquid occurs. The serous fluid is very rich in albumen, of which large quantities may be precipitated, after filtration, by nitric acid. The amount of albumen is much greater than can be accounted for by the admixture of blood. It is the result of exudation of the blood-plasma. The characteristic stools of diphtheritic inflammation of the large intestine consist of a sero-sanguineous fluid containing small morsels of pseudo-membranous and necrotic tissue. These morsels have been compared to raw minced meat, and the yellowish-red fluid containing them was called by the older writers *lotura carnium.* The microscopical examination of these shreds and particles frequently shows only a mass of decomposing granular matter and bacteria mingled with blood-corpuscles and perhaps indistinctly fibrillated fibrin. In other specimens the fibrin may be more distinct. The examination of a sufficient number of specimens will usually bring to view some in which fragments of the tubules of Lieberkühn can be recognized, and thus is demonstrated the origin of these particles from necrosed intestinal mucous membrane. These stools emit a peculiarly offensive, cadaveric odor. Less frequent than the presence of these small shreds is the discharge of large fragments of necrosed mucous membrane. These may be several inches in length. The stools containing them are dark in color, have a putrid odor, and are called gangrenous. The tubular pseudo-membranous casts which have been described by some writers are composed of inspissated mucus. They are very rarely observed in dysentery, but are present in a rare form of enteritis called membranous enteritis, muco-colitis, or tubular diarrhœa. Bacteria are present in dysenteric stools in enormous number. Fungus-spores and infusoria are also observed. The stools may consist almost wholly of blood or they may be chiefly purulent. Little transparent clumps resembling granules of boiled sago or frogs' spawn have been seen in dysenteric stools. According to many writers, they consist of masses of mucus moulded into a round form in follicular ulcers. According to Virchow, they are colored blue with iodine, and are, in reality, undigested granules of starch. Woodward also considers them to be of a starchy nature. Dysenteric stools are usually alkaline in reaction, and are said to contain carbonate of ammonia.

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[1] *Clinical Report on Dysentery*, based on an analysis of 49 cases, by the author, 1853.

The inflammation of the rectum occasions a sensation as if this portion of the bowel were filled, and leads to the frequent desire to defecate, with as much straining as the soreness of the parts will allow. This disposition to strain ineffectually is called *tenesmus*. Sometimes the straining efforts produce prolapsus of the intestine. The evacuations are frequently preceded by griping or colic pains which are called *tormina*. The tormina and tenesmus are not proportionate to the gravity of the disease. They are sometimes wanting in fatal cases, and are prominent symptoms in some mild cases. They are, however, the chief sources of suffering. Tenderness on pressure is in some cases more or less marked over the descending colon and sometimes over the whole tract of the large intestine. It is rarely great, and not infrequently is wanting. Meteorism or tympanitic distension is rarely present. The abdomen is usually depressed. Strangury and retention of urine are occasional symptoms, and in the female leucorrhæa may be produced. Redness and excoriation around the anus may be produced by the irritation of the dejections. Perineal abscess is an occasional result of an extension of the inflammation. Intussusception, with the symptoms of ileus, sometimes occurs in children.

The pulse in the majority of cases is but little, and sometimes not at all, accelerated. The body-heat is but moderately or slightly increased. Exceptionally, high febrile movement exists. The thermometer may show an increase of temperature to 103° or 104°. Great frequency of the pulse always denotes gravity and danger, but the converse does not hold good. In some fatal cases the pulse is never much accelerated. The tongue may be frosted or more or less coated, or it may present a natural appearance. During the course of the disease the appetite is sometimes more or less diminished or is lost. Thirst is usually present, and is sometimes urgent. Vomiting occasionally occurs, and may be prominent as a symptom, the matter vomited being of a greenish color. The intellect is unaffected, as a rule, delirium occurring with great infrequency in cases which end in recovery, but occasionally, in cases which prove fatal, being present toward the close of life. The loss of strength varies greatly in different cases according to the intensity and extent of the local affection, depending also on the condition of the patient when attacked.

The duration of the disease from the date of the attack to convalescence varies from four to twenty-one days. These figures represent the minimum and maximum duration in thirty recorded cases.

PATHOLOGICAL CHARACTER.—Our present knowledge of the pathology of sporadic dysentery embraces only the intestinal affection, the local and general phenomena which make up the clinical history being symptomatic of this affection. The pathological character of the intestinal affection is the same in the sporadic as in the epidemic form of the disease.

Writers have described different varieties of the disease, distinguished as typhoid, bilious, intermittent and remittent, febrile and non-febrile, etc. These terms denote either variations in the phenomena of the disease or the coexistence of other diseases. Dysentery may occur as a complication of continued and of periodical fever. Rheumatism and dysentery are occasionally associated. These combinations are important in their practical relations, but it does not follow that the disease under these circumstances is specifically different from its ordinary form. Variations as regards febrile movement, supposed disorder of the liver, etc. do not affect the special character of the disease. Paralysis affecting certain muscles, especially the muscles of the lower limbs (paraplegia), has been observed in some cases to either accompany or follow dysentery; and different theories have been proposed to explain a supposed pathological connection between the paralytic and the dysenteric affection. The truth is, the affections are so rarely connected that their association is probably a mere coincidence.

Causation.—No age is exempt from liability to this disease, but in the majority of cases the patients are adults and less than thirty-five years of age. A larger number of males than females are attacked, but the preponderance is not sufficient to show that a marked predisposition relates to sex. The causation has a very obvious relation to climate. The disease is much more frequent, in the sporadic as well as in the epidemic form, in tropical than in cold and temperate climates. The causation has also a striking relation to season. Of 44 cases which I recorded, all save 1 case occurred in July, August, September, and October. The constitution and previous health were good in one-half of the cases which I have analyzed, and in the remaining half there was no uniformity in character as regards the antecedent affections or disordered health.

Writers have been accustomed to state that the disease may be produced by various obvious causes, such as atmospherical changes, excess in eating and drinking, indulgence in acid, unripe fruits, crude vegetables, etc.; but in the majority of cases its origin cannot be traced to these causes or to any obvious cause. This renders it probable that a special cause is involved in sporadic as well as in epidemic dysentery. The limitation of the occurrence of the disease to the summer and autumnal season, and its more frequent occurrence in tropical climates, point to this conclusion and to the agency of a high temperature in the evolution of the special cause. Persons who are in good health and vigorous are as likely to be affected as those who are feeble. Various circumstances standing in a causative relation to the disease in an epidemic form may also be involved in its production when it is sporadic. These causative circumstances will be noticed in treating of epidemic dysentery.

Diagnosis.—The diagnosis of dysentery involves no difficulty. The characteristic evacuations are pathognomonic. It is to be discriminated from cases of bloody evacuations and irritation of the rectum incident to hemorrhoids, intestinal hemorrhage or melæna, and the discharge of pus from an abscess opening into the intestine. The points of contrast with dysentery which these cases offer are sufficiently apparent.

Prognosis.—Sporadic dysentery in temperate climates tends to recovery. It is a distressing disease, but is attended with little danger to life. A fatal result is due either to an unusual intensity and extent of the local affection, to an inability of the system to resist the disease from feebleness of constitution, or to some coexisting affection. The prognosis in tropical climates is less favorable. The recovery from the disease in temperate climates is almost always complete; that is, it rarely eventuates in chronic dysentery; nor does it predispose to any other disease. In tropical climates abscess of the liver is not an infrequent sequel. This very rarely follows in temperate climates. So far as my experience goes, immediate relapses are not likely to take place, and the occurrence of the disease diminishes the liability to its occurrence at a subsequent period. With reference to the latter point, my analysis of cases, although the number was not large, developed an interesting result. Of the cases recorded during fourteen years, in not a single instance was the disease known to recur; and in 16 of these cases the patients were under observation after recovery during periods varying from one year to thirteen years. This result was the more striking from the fact that the patients for several years were within the sphere of an epidemic influence which in some instances affected other members of the same families.

TREATMENT.—Sporadic dysentery is a self-limited disease, and in the great majority of cases would end in recovery without medicinal treatment. In order to study its natural history I allowed the disease in 10 cases to pass through its course without any therapeutical interference. These were sporadic cases of average severity. They ended in recovery after an average duration of from eight to nine days. An analysis of 49 cases previously recorded, in which different methods of treatment were employed, gave about the same duration in the cases ending in recovery.[1] These observations are sufficient to establish the self-limitation of sporadic dysentery and its intrinsic tendency to recovery—facts which, so far as I know, had not been before demonstrated by withholding all medication in a series of cases; but there is reason to believe that the disease is sometimes arrested, that its duration may be frequently abridged, and that the distressing symptoms may be greatly relieved by judicious therapeutical measures.

It is desirable that as early as possible the fecal contents of the large intestine should be effectually removed, in order to prevent their continued passage over the inflamed surface, and to secure so far as may be that important end in the treatment of all inflammations—namely, rest of the inflamed part. It would seem that nature endeavors to relieve the bowels of their contents by the diarrhœa which precedes the dysenteric evacuations. The first point in entering on the treatment is to ascertain if the bowels have been spontaneously relieved. With reference to this point the size and character of the stools are to be ascertained and the abdomen examined by palpation. In general, it is advisable to render the effort of nature more complete by giving an effective purgative. Castor oil has been in much repute as an appropriate remedy, but, aside from the objection on the score of the disgust which many persons have for this remedy, the saline cathartics are to be preferred. The sulphate of magnesia or soda, the Rochelle salts, or the citrate of magnesia may be employed. The saline selected should be given in sufficient doses to promptly produce abundant dejections, and it is then to be discontinued. It will sometimes happen that after free purgation the dysenteric evacuations do not return and recovery at once ensues. This abortive effect takes place only in some cases in which the inflammation is not intense and is limited in its extent. Notable diminution of the frequency of the dysenteric evacuations and relief of the tormina and the tenesmus not infrequently follow the operation of the saline purgative. This treatment may be pursued except in cases in which it is contraindicated by great feebleness of the patient. In these cases castor oil is the purgative to be preferred.

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[1] Vide "A Contribution toward the Natural History of Acute Dysentery," in the *Am. Journ. of Med. Sciences*, July, 1875, and a clinical report based on analyses of 49 cases, in the *Buffalo Med. Journ.*, July, Aug., Sept., and Oct., 1853.

Another method of effecting the removal of the contents of the large intestine deserves mention—namely, the employment of large injections. More than a century ago O'Beirne demonstrated that a flexible tube can be passed into the sigmoid flexure, and in this way a large quantity of liquid injected into the colon. He advocated the employment of this method in dysentery and other affections. Under the name irrigation (Darmeingiessungen) this method has been recently revived, and it is recommended on the basis of clinical experience especially by two German writers, Hegar and Mosler.[1] By means of a tube carried into the sigmoid flexure several quarts of liquid may be injected. By the writers just named the injection is made through a funnel elevated sufficiently for the requisite force from gravitation. The object is to remove fecal accumulations and all irritating matters. By this method also medicated liquids may be brought into contact with the inflamed surface. The method claims trial sufficient to determine whether it can advantageously take the place of saline or other cathartics in the treatment of dysentery, and also to what extent the topical application of medicated liquids may be employed with advantage. The injections may be repeated two or three times daily. They are made more effectual if the patient be placed in the knee-and-elbow or the knee-and-shoulder posture, but if the patient be feeble the decubitus may be on the back or side. Davidson's syringe will probably answer as well as, if not better than, the elevated funnel. In order to introduce as much water as is practicable and desirable, it should be introduced slowly, intermitting the injection for a few moments from time to time.

After purgation, opium in some form is the remedy most to be relied upon. A full dose of opium administered by enema or as a suppository will sometimes succeed in arresting the disease. It often mitigates the severity of the disease and affords marked relief. Administered by the rectum, however, it is not always retained, and then reliance must be had upon its administration *per os* or hypodermically. It may be given in full doses repeated every four, six, or eight hours, or in smaller doses repeated at shorter intervals. As regards the form of opium selected, I believe this is chiefly important in respect to the preference or peculiarity of the patient. I have seen the opium in gum or powder, the various liquid preparations, and the salts of morphia equally effectual. The form found to be preferable is to be continued during the progress of the disease, the doses being regulated by the frequency of the evacuations and the suffering. Experience in each case is to be the guide as to the mode of administration. Generally, the administration by the mouth and rectum alternately is advisable. The opiate remedies are to be gradually diminished as the dysenteric evacuations become less frequent and the suffering from tormina and tenesmus is less. After two or three days, if the disease continue, the saline purgative may be repeated, if not contraindicated by the feebleness of the patient, and afterward the use of opium resumed.

In this plan of treatment the reliance is upon free purgation and opium. The chief reliance, I believe, should be upon these measures, but other measures may be conjoined. The subnitrate or the subcarbonate of bismuth in scruple or half-drachm doses, the acetate of lead, the sulphate of copper, gallic acid, and the various vegetable astringents, such as rhatania, kino, catechu, hæmatoxylon, etc., are useful as adjuvants. These are to be relied upon, to the exclusion of opium, in those cases only in which, from a constitutional peculiarity, the latter produces unpleasant effects sufficient to preclude its employment. It is undoubtedly true that some persons are affected so unpleasantly by opium that its use is, if possible, to be avoided; but it is often the case that it is well borne in this and some other diseases, although it may not be in trivial affections. Certain preparations of opium may be well borne, although others occasion unpleasant effects. An aqueous solution or extract is frequently taken without difficulty by those who cannot comfortably take other preparations. Codeia sometimes answers well as a substitute for the preparations of opium and morphia.

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[1] *Deutsches Archiv für klin. Med.*, 1875, Bd. 15, p. 233.

Dr. Q. C. Smith of Texas recommends belladonna as an adjunct to opium, and as a substitute when the latter remedy is not well borne. His recommendation is based on a large personal experience and on the testimony of physicians in his neighborhood. He has found belladonna especially useful in cases occurring in childhood, in view of the fact that opium must be given to children with more caution than to adults.

Ipecacuanha has been considered a valuable remedy in dysentery. Some have attributed to it a special curative influence, and have advocated its employment in as large doses as can be borne. Maclean states that the system of treating acute dysentery generally followed in India is as follows: Thirty drops of the tincture of opium may be at first given, but this may be sometimes omitted and the ipecacuanha given at once. If the opiate be given, in half an hour it is to be followed by from 25 to 30 grs. of ipecacuanha. The latter remedy should be given in as small a quantity of fluid as possible, and a little syrup of orange-peel covers the taste of the medicine. The patient after this dose should keep perfectly quiet and abstain from fluid for at least three hours. With these precautions nausea and vomiting are rarely troublesome. After eight or ten hours the ipecacuanha may be repeated in a reduced dose, observing the same precautions.[1] If required, the treatment is to be continued for several days, the remedy being given in diminished doses, with intervals sufficient to allow mild nourishment to be taken. He cites statistics showing the mortality in India under the use of ipecacuanha to be 28.87 in 1000, whereas under the old system the mortality was 88.2 in 1000. It is to be borne in mind, however, that the old system of treatment included bloodletting and the free use of mercury. I have resorted to this plan of treatment in a considerable number of cases at Bellevue Hospital, but without very satisfactory results. In a small proportion of cases the disease was either arrested or was favorably modified by it, but in the majority of cases it had no apparent influence on the disease.

Mercury has been heretofore much relied upon in the treatment of this disease, given either in large doses, united with opium in order to prevent a cathartic effect, or in small doses repeated at short intervals. The recovery in cases treated with mercury is of course no proof of its value, inasmuch as sporadic dysentery tends intrinsically to recovery. The duration of the disease, the relief of the symptoms, the rapidity of convalescence, and the subsequent condition of health are the points to be considered in estimating the influence of remedies in this disease. With reference to these points a fair estimate of the effects of mercury has led to its rejection by most practitioners in this country as a remedy for dysentery.

During the early part of the disease the diet should be restricted to a small quantity of the blandest articles of food. It is an object throughout the disease to keep the patient on a diet which will leave but little residuary matter to pass into the large intestine; in other words, a diet as purely nutritious as possible. After the first few days, however, it is important to provide for proper support of the system by nutritive supplies, and for this purpose milk and animal broths are best suited. If the symptoms denote failure of the vital powers, alcoholics are to be given, as in all diseases which destroy life by asthenia. Tonic remedies may be given with advantage during convalescence.

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[1] Vide article on dysentery in *A System of Medicine*, edited by Reynolds.

Patients should be instructed to refrain as much as possible from repeated acts of defecation and straining efforts. Cold applications to the anus sometimes relieve the tenesmus. A piece of ice may be wrapped in cloth and applied frequently in this situation. Enemas of cold water are often grateful. Relief in some cases is afforded by injecting astringent liquids. The nitrate of silver, a solution of tannin, acetate of lead, or the sulphate of zinc may be thus used.

Fomentations and mild revulsive applications over the abdomen are useful as palliatives in proportion to the amount of tormina and abdominal tenderness.

Epidemic Dysentery.

Epidemic dysentery is essentially the same disease as sporadic dysentery. Certain events occur much oftener in the former than in the latter, yet there is nothing pertaining to the epidemic which is not occasionally seen in the sporadic form of the disease. Epidemic dysentery is often very fatal, yet in the epidemics attended with the largest fatality cases occur which are as mild as the mildest sporadic cases, and on the other hand occasionally in sporadic cases life is quickly destroyed. Like other diseases which prevail epidemically, this differs greatly at different times and places as regards gravity, and at certain times and places it presents features which it does not present at other times and places. The differences between the epidemics of this disease are in a measure to be explained by the coexistence of other diseases, and by the conjoint operation of morbific causes not standing in a special relation to the production of this affection. In treating of epidemic dysentery the main object will be to point out the circumstances in which it is likely to differ from sporadic dysentery.

With respect to the ANATOMICAL CHARACTERS, they are essentially the same as in cases of sporadic dysentery. In fatal cases of either form the varied appearances which have been stated may be found. Extensive and disorganizing changes occur oftener in epidemic dysentery. As a rule, in the latter the inflammation extends over a larger portion of the large intestine, and it sometimes extends into the ileum. Ulcerations and diphtheritic exudation are much more likely to occur in epidemic dysentery. Perforation of the intestine is sometimes a result of ulceration, giving rise to peritonitis. Perforation of the cæcum may take place in this disease, leading to abscess in the areolar tissue beneath the peritoneum or fecal abscess. Perforation of the rectum and fecal abscess in that situation have been known to occur.

The CLINICAL HISTORY of epidemic differs from that of sporadic dysentery according to differences as regards the extent and severity of the local affection, and also according to various morbid conditions due to the action of accessory causes, the latter co-operating oftener in epidemic than in sporadic cases. Severe cases of epidemic dysentery are characterized by the occurrence of sero-sanguinolent dejections, the *lotura carnium* of the older writers, containing usually pseudo-membranous and necrotic particles in more or less abundance, and holding in solution a large quantity of albumen. Hence epidemics of dysentery are popularly known as *bloody flux*. Dejections of this character always denote gravity of disease. Tormina and tenesmus may be less prominent as symptoms in severe cases of epidemic than in most cases of sporadic dysentery. In some fatal cases these symptoms are entirely wanting. Tenderness over the tract of the colon may or may not be marked. The local symptoms in some cases of epidemic dysentery are comparatively slight, and in general, so far as my observation goes, patients suffer far less in fatal cases than in sporadic cases wholly devoid of danger. Nausea and vomiting occur oftener in the epidemic than in the sporadic disease. In epidemic dysentery albumen and casts in the urine are occasionally observed.

The differences, as regards the general symptoms, in different epidemics and in different cases are marked. Severe cases are characterized by prostration, feebleness of the circulation, coldness of the surface—in short, by symptoms denoting adynamia. The pulse may become frequent, small, soft, and vibratory but compressible, or it may be small, weak, and thready, with but little, and sometimes not any, increase of frequency. These characters of the pulse, denoting feebleness of the action of the heart together with other adynamic symptoms, are developed in some cases early in the disease, without having been preceded by active febrile movement; but in other cases the pulse for a time is full and strong, the skin hot, the adynamia succeeding these symptoms of high fever. In fatal cases a condition analogous to the stage of collapse in epidemic cholera may precede, for a variable period, the termination of the disease. In this condition the pulse is scarcely appreciable or it is extinct; the surface is cold, and either dry or bathed in perspiration; the teeth are sometimes covered with sordes; the quantity of urine is greatly diminished; cramps of the muscles of the lower extremities sometimes occur; and this collapsed condition may exist for one, two, or three days before death. The mode of dying is by slow asthenia.

The intellect generally either remains intact or slight mental aberration occurs only in the latter part of life; but some cases are characterized by delirium. The patient talks incoherently under the influence of transient delusions; he may attempt to get out of bed, and requires to be restrained. In a case which came under my observation the delirium was of a boisterous, mirthful character, the patient singing snatches of humorous songs, shouting, and attempting to get out of bed, after the pulse had become extinct and the surface of the body was cold like that of a cadaver. In this case the manifestations of delirium were so painfully incongruous that the patient was kept under the influence of chloroform for several hours before his death.

The delirium and other symptoms in some cases are analogous to those belonging to typhus and typhoid fever. The typhoid state may exist in cases of this disease as in various other affections. But it is to be borne in mind that dysentery sometimes occurs as a local complication of typhus and typhoid fever. Certain epidemics of these fevers are characterized by the occurrence of this complication. The distinction between dysentery with typhoid symptoms, or typhoid dysentery, and typhus or typhoid fever with a dysenteric affection developed secondarily, is the same as between typhoid pneumonitis and typhus or typhoid fever complicated with pneumonitis. The symptoms of scorbutus are associated with those of dysentery when, as is sometimes the case, the latter prevails among persons who are suffering from scorbutic deterioration of the blood. So in districts where periodical fevers prevail, epidemics of dysentery may be characterized by periodicity as regards febrile movement, etc., due to the union of dysentery with intermittent or remittent fever or the conjoined operation of the special cause producing the latter.

The duration of the disease in cases of epidemic dysentery is very variable. In malignant epidemics the disease sometimes runs very rapidly to a fatal issue, the collapsed condition occurring perhaps in the course of a few hours. On the other hand, cases may be protracted to the third and fourth week before either convalescence or death takes place. As already stated, epidemics differ widely as regards the severity of the disease. In some epidemics the majority of cases are mild and the rate of mortality is very small, while in other epidemics the proportion of deaths is very large. The latter are justly distinguished as malignant. There are few epidemics more to be dreaded than those of malignant dysentery. They occur much oftener in tropical than in temperate climates, but there is no part of the world exempt from their occasional occurrence. Epidemic dysentery is one of the scourges of camps in time of war, especially if to causes operating particularly on armies tropical influences be added. It prevailed to a very great extent in the late civil war in this country among the national and the insurgent forces, especially in the Southern States, and it was often associated with phenomena referable to malaria.[1]

Epidemic dysentery is an infectious disease. The special CAUSE is the same in epidemic and in sporadic dysentery. Reasoning analogically, this special cause is a specific micro-organism. The various agencies which appear to give rise to the disease, such as exposure to atmospheric vicissitudes, fatigue and hardships incident to army life, insufficiency of diet, overcrowding, malaria, etc., probably act as co-operating causes, rendering the system more susceptible to the dysenteric poison and giving rise to other morbid phenomena than those which the special cause would alone produce. Epidemics would perhaps not often occur were it not for the operation of accessory causes; hence the rationale of their occurrence in camps, in prisons, and in situations where numbers are exposed to hygienic influences conducive to the development of any disease. With respect to the source of the special cause of dysentery or the conditions under which it is generated and diffused we have at present no positive knowledge. The contagiousness of epidemic dysentery has been a much-mooted question. Without entering into any discussion of this question I shall content myself with saying that the evidence of communicability rests mainly on the successive occurrence of a greater or less number of cases among members of one household, or in persons who from proximity of residence are brought into contact with each other; and the diffusion of the disease under these circumstances may perhaps be satisfactorily accounted for by the fact that the persons attacked are alike exposed to the special cause together with the same co-operating causes. It is, however, proper to state that some distinguished authors suppose that the intestinal excreta contain a virus by means of which the disease may be communicated. That the virus is a *contagium vivum* has not as yet been proved by the discovery of a special micro-organism and the production of the disease by inoculation. Whether the disease be communicable or not, there are instances which seem to afford evidence that the special cause may be transported by means of clothing, etc.

The opinions of different writers and practitioners concerning the TREATMENT of dysentery, especially as an epidemic, show great diversity, and there is abundant testimony of the success of measures quite opposite in character. The fact just stated shows either that the recoveries were due mainly to an intrinsic tendency of the disease, or that the disease is presented at different times and places under different pathological phases, and the measures of treatment require to be correspondingly varied. Both explanations are doubtless measurably applicable. Sporadic dysentery as a rule, and epidemic dysentery in a certain proportion of cases, tend intrinsically to recovery. Recovery under these circumstances takes place, whatever be the treatment, provided it be not destructive. On the other hand, in a certain proportion of cases of epidemic dysentery the disease must, of necessity, end fatally. The extent of mucous surface affected, the loss of fluids by hemorrhage and transudation, and the destructive lesions which take place preclude recovery under any treatment; but it is also true that the different features which the disease assumes in different epidemics, and the various morbid conditions with which it may be associated, must more or less influence therapeutical measures.

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[1] Vide Woodward, second medical volume of *Med. and Surg. History of the War of the Rebellion*, Washington, 1879.

Purgatives, especially of the saline class, may be as appropriate in epidemic as in sporadic dysentery; but they should be given cautiously or omitted if sero-sanguinolent dejections occur or if the symptoms denote much tendency to adynamia. Theoretically, it would seem that the removal of the fecal contents of the intestine would be more safely effected by large injections (irrigation) than by saline purgatives. It is very desirable to arrest or moderate the dejections just named, for these doubtless conduce to the condition of collapse into which patients are liable to fall. For this purpose opium is to be given in as large doses as are required or as are well borne, in conjunction with astringent remedies, such as gallic acid, acetate of lead, the persulphate or pernitrate of iron, rhatany, etc. I believe the main reliance should be placed on opium in some form in these cases, other remedies being considered as auxiliary. The latter, therefore, are not to be employed as substitutes for opium except in cases in which, from a constitutional peculiarity, this remedy in any form produces such distressing effects as to contraindicate its use. It should not be decided that opium cannot be tolerated on the assertion of the patient, but only after a fair trial.

In this disease, as in some others, there is frequently a wonderful tolerance of opium—a tolerance not to be explained by the antagonizing influence of pain, since it is manifested in cases in which pain is not a prominent symptom. Christison, in stating this fact, adds that he has known from 24 to 30 grains of the gum of opium to be taken in the course of twenty-four hours. I have given 2 grains hourly in the case of a female affected with dysentery characterized by abundant sero-sanguinolent dejections, for the space of a week, without at any time symptoms denoting narcotism. I have met with a case exemplifying a still greater tolerance of this remedy. In this case, the patient not being habituated to the use of opium before his illness, the quantity of the sulphate of morphia given hourly was gradually increased to one grain, making 24 grains in the twenty-four hours, and this quantity was given for several consecutive days without any narcotism. Opium is not to be given as a matter of course in doses approaching these, but it is to be given with a view to the desired end—namely, the arrest of the sero-sanguinolent dejections; and if this effect be not obtained the doses are to be increased to the amount which will be borne without narcotism, be the amount never so large. It is hardly necessary to say that in increasing the dose the condition of the patient is to be carefully watched; and attention is to be directed to the pupils and to the number of respirations, as well as to the soporific state, in order to avoid a cumulation which may result unexpectedly in narcotism. The quantity of opium which may with prudence and propriety be given in dysentery will vary greatly according to the varying tolerance in different cases.

In proportion as the symptoms denote a tendency to death by asthenia, the powers of life are to be supported by alcoholics and nourishment. The general principles which should govern the employment of supporting measures are the same as in other affections which in like manner tend to destroy life by exhaustion. Alcoholics are sometimes tolerated in large quantity in severe cases of this disease. I have known over 40 ounces of brandy to be taken in twenty-four hours by a patient who in health was not accustomed to the use of ardent spirits, and this large quantity produced no excitation of the circulation or nervous system. This patient was saved apparently by the persevering use of alcohol with large doses of the sulphate of morphia. I have repeatedly seen patients recover under the vigorous employment of supporting measures, conjoined with opium in large doses, when, as judged by the symptoms, the condition appeared to be truly desperate; yet of the cases properly distinguished as malignant many must die, despite the faithful employment of the measures on which the chief reliance is to be placed.

Dysentery associated with phenomena denoting the operation of the special cause of periodical fever calls for treatment having reference to the latter, in addition to the measures addressed to the dysenteric affection. Quinia is a highly important remedy in these cases. Associated with scorbutus, the dietetic and remedial measures appropriate to this condition are indicated. Occurring as a complication of the continued fevers, it furnishes an additional indication for the supporting measures which these fevers claim irrespective of the dysenteric complication. Coexisting rheumatism calls for the addition of the remedies appropriate for this disease.

Chronic Dysentery.

The anatomical basis of chronic dysentery is chronic inflammation of the large intestine, with or without ulceration. The term, however, is sometimes applied to cases of functional diarrhœa, and on the other hand the term chronic diarrhœa is often applied to cases of chronic dysentery. The distinction between chronic diarrhœa and chronic dysentery is usually based upon the character of the stools. It is impossible, however, to draw a sharp boundary-line between the two processes. Woodward even takes the ground that no distinction is admissible. He says:[1] "The results of any attempt to subdivide these cases into chronic diarrhœa and chronic dysentery in accordance with the presence or absence of tenesmus or of mucus, muco-pus, pus, or blood in the stools, or any other clinical evidence with which I am acquainted, would not be found to correspond with any anatomical classification." He therefore includes under the name chronic diarrhœa all the chronic alvine fluxes with the exception of those resulting from tuberculous ulceration of the intestine; but, as in many other processes which cannot be sharply separated from each other, it is here convenient to retain the old names, although the assignment of certain cases to one class or to the other may be more or less arbitrary. Typical cases of chronic diarrhœa, however, are sufficiently distinguishable from typical cases of chronic dysentery.

Chronic dysentery very rarely occurs in this climate, save as a sequel of acute dysentery. Recovery from the latter does not take place, but the inflammation becomes chronic, or more commonly ulcerations which take place during the acute affection do not cicatrize, but continue for an indefinite period. Acute exacerbations often occur in the course of chronic dysentery. Acute dysentery, however, very rarely eventuates in the chronic form of the disease in temperate climates, and hence chronic dysentery is rare in these climates. It is otherwise in warm climates. Here the chronic often follows the acute affection. Chronic dysentery in tropical climates may also begin insidiously without an acute attack. Chronic dysentery thus prevails chiefly in tropical countries or among those who have contracted dysentery in these countries. In the Northern States of the Union the cases observed are, for the most part, imported from the South. Of the soldiers who returned from the war in Mexico, large numbers were affected with this disease, and during the late civil war cases were abundant among the soldiers sent to the Northern military hospitals from different points in the Southern States.

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[1] Woodward, second medical vol. of the *Med. and Surg. History of the War of the Rebellion*, Washington, 1879, p. 483.

The lesions observed in chronic dysentery are the result of inflammation of the mucous and submucous coats of the large intestine, with or without ulceration. Chronic inflammation without ulceration presents an appearance in many respects similar to that already described for acute simple inflammation of the large intestine. Instead of bright red, the color of the mucous membrane is more frequently bluish-red, or some hue due to the deposit of dark pigment, as brown, slate-color, or ash-color. The pigmentation may be both in the follicles and in the mucous membrane outside of the follicles. The mucous and submucous tissues are thickened, usually more in some places than in others. The wall of the intestine often feels stiff and hard. Its calibre may be diminished. The mucous membrane may present many small elevations lying close to each other and producing the so-called mammillated appearance. In rare cases large polyp-like excrescences, consisting of hyperplastic connective tissue containing glands and covered with columnar epithelium, are produced, and may be present in large number (enteritis polyposa). Sometimes little projecting cysts, varying in size from a pin's head to a pea and containing clear mucus, are observed beneath the mucous membrane. These are formed by the dilatation and coalescence of the tubules of Lieberkühn. They generally occupy the site of the solitary follicles. As pointed out by Cornil, and especially by Kelsch,[1] the solitary follicles in dysentery, particularly in cases with follicular enlargement and ulceration, are not infrequently invaded by distension and new growth of the adjoining tubules of Lieberkühn. The thickening of the mucous and submucous coats in chronic dysentery is due partly to infiltration with new cells and partly to new formation of connective tissue. There is, however, less tendency toward the development of new fibrillated connective tissue in chronic dysentery than in the chronic inflammation of most mucous membranes. Hence, complete recovery is possible after long duration of the disease. In some cases of chronic dysentery enlargement and ulceration of the solitary follicles are very prominent, and constitute almost the sole lesions. Such cases are called follicular inflammation or follicular dysentery. In many of these cases the growth of the glands of Lieberkühn into the follicles has been observed by Kelsch and by Woodward. Swelling and ulceration of the follicles, however, frequently occur without any such growth.[2]

The ulcers which may occur in dysentery are of three kinds: first, follicular; second, non-follicular or simple (catarrhal); and third, diphtheritic. These varieties are often combined. The follicular ulcerations are the result of softening and breaking down of the swollen solitary follicles, either in their centres or at their apices. The ulcer at first presents a little, round, constricted opening, as if punched out, leading into an irregular cavity in the mucous or submucous tissue. It extends peripherally more rapidly in the submucous than in the mucous coat, and hence often presents overhanging edges, even when of considerable size. By extension and coalescence of these follicular ulcers large losses of substance in the inner coats of the intestine are produced. Ulcers unconnected with the follicles are formed by dense infiltration of the mucous membrane with pus-cells. By softening and molecular disintegration of the intercellular substance, first in the upper layers, superficial ulcers are produced. These extend in depth and in superficies by continuance of the purulent infiltration and by liquefaction of the basement-substance. These ulcers possess no very distinctive characters. They resemble closely those produced by diphtheritic sloughing. The method of formation of diphtheritic ulcers has already been sufficiently described under the head of Acute Dysentery. When the fibrinous exudation and the sloughing process disappear, these ulcers extend by the same process of suppuration as the preceding. They usually present abrupt or sloping edges, but may have undermined walls. In fact, when dysenteric ulcers attain to a large size, it is generally impossible to tell in what way they have originated. Any of these ulcers may extend in depth so far as to perforate the intestinal wall, but this is a rare occurrence. Recovery from dysentery with ulceration is effected by cicatrization of the ulcers. Some ulcers may cicatrize while others are extending. There is nothing characteristic regarding these cicatrices except in the case of those resulting from small follicular ulcers. The cicatrices of these small follicular ulcers are stellate in shape. The radiating ridges extend into the substance of the surrounding mucous membrane, and enclose between them mucous membrane somewhat modified, but containing the glands of Lieberkühn (Woodward). Allusion has already been made to the occasional occurrence of stricture of the intestine from contraction of the cicatrices resulting from dysenteric ulcers. Waxy degeneration of the intestinal mucous membrane has been occasionally observed in cases of dysentery. Acute diphtheritic inflammation may occur at any time during the course of chronic dysentery, and may lead to a fatal issue. Of the diseases which may complicate chronic dysentery, peritonitis, hepatic abscess, and pneumonitis should be mentioned especially. Abscess of the liver is a frequent complication in tropical, but is rare in temperate, climates. It is generally in the form of small multiple abscesses.

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[1] *Archives de Phys. norm. et path.*, 1873, pp. 406 and 573.

[2] Kelsch (*loc. cit.*) describes a form of dysentery prevalent in tropical countries in which embryonic or granulation-tissue develops in the mucous membrane and obliterates most of the glands of Lieberkühn. Portions of these glands undergo cystic distension and invade the solitary follicles. Embryonic cells collect in especially large number about Brücke's muscle and infiltrate also the submucosa. The disease begins insidiously, and, unless acute attacks occur, no ulceration takes place If there be an acute attack, the accumulation of pus-cells about the vascular plexus beneath the mucous membrane becomes so great as to shut off the nutritive supply to the tissue above, and gives rise to sloughing of the mucous membrane. Kelsch denies that a fibrinous or diphtheritic exudation ever occurs in dysentery; but his observations are based upon an insufficient number of cases.

In chronic dysentery the evacuations vary in character. They contain at times thin or pultaceous feces, and occasionally scybala. Their most characteristic ingredients are mucus, pus, and blood; mucus, however, may be absent. Flakes of pseudo-membrane and of necrosed tissue are present only when an acute diphtheritic inflammation supervenes. Undigested particles of food are often present, especially when substances difficult of digestion are taken. Woodward applies the term yeasty to frothy stools containing bubbles of carbonic acid and the yeast-fungus. These may be observed when the digestion is greatly impaired and amylaceous articles of food are taken. The stools are usually accompanied with griping pains. Tenesmus is less frequent than in acute dysentery. In some cases the evacuations are unattended with any feeling of distress. The stools vary from three or four to twenty or more in the twenty-four hours. There may be intervals in which constipation takes the place of the frequent discharges. The accumulation of gas in the intestines and the escape of flatus may be annoying symptoms. In severe cases the stools are always loose and thin and contain mucus, pus, and blood. In these cases it is to be inferred that the inflammation extends over the whole or the greater part of the large intestine. In other cases dysenteric discharges occur more or less frequently in alternation with fecal dejections. In these cases the inflammation is less diffused. If the evacuations be generally or frequently solid or moulded, the dysenteric evacuations being superadded, the inflammation or lesions are circumscribed and situated near the anus. In these cases ulcerations may sometimes be seen if examination be made with the speculum. There is generally more or less tenderness along the line of the colon.

Chronic dysentery is one of the most intractable and hopeless of diseases. For a time, if the evacuations be held in check by palliative measures, the appetite and digestion not being greatly impaired, the general aspect and strength may not show much deterioration, but at length the appetite and digestion fail, and the continued irritation and loss of fluids induce progressive emaciation and debility. The duration of the disease extends usually over several months, and sometimes years. If not destroyed by some intercurrent affection, the patient becomes extremely emaciated and is reduced almost to a skeleton; the surface is usually dry, cool, or cold; the pulse becomes more and more feeble; the mental faculties are weakened, delirium rarely occurring, but the mind in certain cases falls into an apathetic state, the patient being indifferent to, and taking but little notice of, persons and things around him. Anorexia becomes complete, and vomiting in some cases is a prominent symptom; œdema of the lower limbs sometimes occurs; ulceration of the cornea is an occasional event, and I have known the cornea to be perforated, with loss of the humors of both eyes. The mode in which a fatal termination takes place is generally typical of dying by slow asthenia. The immediate cause of death is sometimes general peritonitis from perforation of the intestine. Parenchymatous nephritis is not an infrequent complication, but uræmic coma and convulsions are rare. Woodward states that "sudden death was a not infrequent occurrence, and attracted considerable attention during the war."[1]

The DIAGNOSIS of chronic dysentery is not always easy. The disease is to be discriminated from functional diarrhœa, and this is not always readily done. The presence of pus, mucus, and of blood at times, if not frequently or constantly, is the important point in the differential diagnosis. It is also to be discriminated from ulceration of the large intestine due to tuberculosis, carcinoma, or other cause. Tuberculosis may be excluded if the lungs be free from the deposit of tubercle. In carcinomatous disease frequently a tumor is to be felt through the abdominal walls, or if seated in the rectum it is accessible to examination by means of the touch and speculum. The fact that in the great majority of cases chronic is preceded by acute dysentery is highly important in the diagnosis. We may conclude at once that the disease is chronic dysentery if it have followed the acute disease; and the latter fact may generally be ascertained without difficulty, owing to the well-marked diagnostic symptoms of acute dysentery. In temperate climates the affections with which chronic dysentery is liable to be confounded are most likely to exist in cases of doubt, owing to the infrequency of the latter.

The TREATMENT of chronic dysentery relates *first* to the local affection. Remedies to allay irritation and to promote the healing of ulcerations are indicated; but, unhappily, in the great majority of cases there is very little probability that a cure will be effected, and all that can be hoped for from judicious treatment are palliation of symptoms and prolongation of life.

Certain remedies have been employed with a view to their direct action upon the affected portions of the intestines. The nitrate of silver is one of these; but it is absurd to suppose that this remedy, given as largely as prudence will allow, passes through the stomach and small intestines unchanged. Whatever benefit may be derived from it, therefore, is not to be explained on the ground of its topical application to the diseased surface. The balsamic medicines—particularly the balsam of copaiba—have been given for the same purpose, and with more reason; but clinical observation shows not much benefit to be derived from them. The subnitrate or the subcarbonate of bismuth may be included among the remedies supposed to act by coming in contact with the affected parts. This remedy is often beneficial as a palliative, the dejections becoming less frequent, with relief of pain, etc., under its use. It should be given in doses of from 20 to 30 grains three or four times daily. Some have recommended much larger doses, and, given almost without limit, it produces no deleterious effects; but probably all the benefit to be derived from it is secured by the doses just indicated. Its administration in enemas has been advocated in this disease. The various astringent remedies are to be tried in succession. Of those most likely to be useful the following may be mentioned: tannic or gallic acid, alumen, rhatany, rubus villosus (blackberry-root), and the persulphate or pernitrate of iron. The oxide of zinc is considered by many as a useful remedy.

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[1] *Op. cit.*

In so far as the dejections are controllable by the foregoing remedies, they should be relied upon to the exclusion of opium, because they will interfere less with appetite and digestion. Opiates, however, will be required to a greater or less extent in conjunction with other remedies. So far as practicable, they should be administered by the rectum, either in enemas (a salt of morphia or some liquid preparation contained in an ounce of mucilage or thin starch-water) or in suppositories. The lower part of the rectum should be examined with the speculum, and appropriate topical applications made if ulcers in this situation be discovered. Dr. Maury and Prof. T. G. Thomas have reported cases in which remarkable benefit followed the application of nitric acid to ulcerations within the rectum; and they call attention to the fact that by means of a speculum the interior of the bowel may be brought into view as far as the sigmoid flexure.[1] The removal of fecal collections and of other irritating material, by means of large injections given through a flexible tube introduced into the sigmoid flexure, deserves trial in chronic as in acute dysentery. Prudence, however, dictates caution in the introduction of the tube and as regards the quantity of water introduced, lest rupture at an ulcerated spot may be produced.

The treatment relates, *secondly*, to the system. In relative importance the general take precedence of the local measures. The surface of the body should be well protected against atmospheric changes. The diet should be nutritious but bland, articles of food being selected which do not leave much indigestible matter to pass into the large intestine. Milk, eggs, and farinaceous food are generally best suited, but in some cases animal food is found to agree. The plan of diet is to be governed by experimental trials. Tonic remedies and alcoholic stimulants are indicated by defective appetite and digestion. Of tonic remedies, quinia, the chalybeate preparations, the mineral acids, and the bitter infusions are to be employed in succession. The use of alcoholic stimulants is to be regulated by their effects in individual cases. Moderate out-of-door exercise and mental recreation are important. Change of climate is sometimes effectual when other measures prove unavailing. The experience of the late war showed the happiest effect of transferring soldiers from the South to Northern hospitals in salubrious rural situations.

Inflammation and Perforation of the Cæcum—Fecal Abscess.

Inflammation of the cæcum, the inflammation limited to this portion of the large intestine, constitutes an affection called *typhlitis*, *typhlo-enteritis*, or

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[1] Vide *New York Med. Journ.*, Jan., 1876, and the author's *Clinical Medicine*, p. 292 *et seq.*

cæcitis. Acute inflammation confined to the cæcum is not of frequent occurrence. The symptoms are—pain and tenderness within a circumscribed space corresponding to the situation of the cæcum, with vomiting, sometimes constipation and sometimes diarrhœa, and febrile movement. It is liable to be confounded with phlegmonous inflammation connected with disease of the vertebræ, renal calculus, circumscribed peritonitis from perforation of the vermiform appendix, and inflammation of the right ovary. Peritoneal inflammation, limited to the portion of peritoneum covering the cæcum, may accompany the affection. The pain and tenderness are then more marked, and the cæcum is distended by an accumulation of gas, the peritoneal inflammation producing paralysis of the muscular tunic. The distension sometimes is so great that a circumscribed prominence corresponding to the area occupied by this portion of the large intestine is manifest to the eye and may be felt through the abdominal walls. Extension of the limbs may occasion pain, as in cases of general peritonitis. The cases which I have seen have terminated in recovery. I have not met with perforation followed by either general peritonitis or fecal abscess in a case of acute inflammation of the cæcum. The probable explanation is that in these acute cases circumscribed peritonitis gives rise to an agglutination of peritoneal surfaces, which is protective against perforation. Subacute inflammation gives rise to symptoms less distinctive. It may occur, and recovery take place, without the development of symptoms denoting an affection of importance; but it becomes a very important affection when it leads to ulceration and perforation of the intestinal coats.

If the perforation take place in that portion of the cæcum not invested with the peritoneum, the gaseous and other contents of the intestine, escaping into the areolar tissue, give rise to inflammation which may be either diffused or circumscribed, in the latter case forming an abscess called a fecal abscess. If diffused, the inflammation may extend more or less over the abdominal walls, which become swelled, pitting on pressure as in œdema, with a feeling of crepitation due to the permeation of intestinal gas. If an incision be made, a dark, sanious liquid escapes with gas emitting an intestinal odor. Under these circumstances a fatal result is inevitable. If, however, the perforation lead to a circumscribed abscess which opens externally, gas and fecal matter, with pus, escape; a fistulous communication with the intestine is established; and this may remain permanently, or it may gradually close and recovery take place.

Perforation at a point where the intestine is covered with the peritoneum may give rise to diffuse peritonitis, which in most cases, if not invariably, is fatal. Often, however, the escape of the intestinal contents into the peritoneal cavity is prevented by a prior circumscribed peritonitis resulting in protective adhesion of the peritoneal surfaces. Under these circumstances a perityphlitic abscess follows, and this is liable to rupture into the peritoneal cavity if timely surgical interference be not resorted to.

The causes of inflammation of the cæcum, whether it be acute or subacute, are probably the detention in this situation of the feces, and the presence of irritating matters either received with the ingesta or arising from chemical changes within the intestine. Over-exercise and exposure to cold may be auxiliary causes.

Complete rest is an essential part of the treatment. This should be rigidly enforced when the inflammation is subacute not less than when it is acute. The importance of this part of the treatment relates to the prevention of perforation. It is desirable to effect the removal of the contents of the cæcum, but active purgatives are fraught with danger. Mild laxatives only are admissible, such as castor oil or small doses of Epsom salts. It is perhaps safest to rely upon enemata given through a long flexible tube carried upward to the sigmoid flexure. These may be repeated at short intervals; that is, three or four times daily. A warm poultice or the water-dressing should be applied over the site of the cæcum. Blisters and other counter-irritant applications are of doubtful utility. The ingesta should be restricted to animal broths and milk with the addition of lime-water. Opiates should be given in doses sufficient to relieve pain. After the disappearance of the symptoms denoting inflammation, the diet for some time should be regulated with reference to a small quantity of fecal residue. Constipation should be avoided by means of mild laxatives, and physical exercise should be interdicted.

Perforation of the cæcum from an ulcerative process may take place without having been preceded by local symptoms denoting any important affection. It occurs perhaps when patients are engaged in their usual avocations. A localized sharp pain is usually felt at the time of the perforation, followed by circumscribed tenderness. These symptoms should at once excite suspicion. Within the area of the pain and tenderness, situated in the right iliac fossa, a sense of resistance is shortly felt by the touch, the boundaries of which may not be sharply defined. On percussion there may be either flatness or a tympanitic resonance, the latter, if limited to the area of tenderness, being due to gas escaping through the perforation. In the course of a few days fluctuation is appreciable.

As regards DIAGNOSIS, fecal or perityphlitic abscesses are to be discriminated from psoas abscess in Pott's disease, abscess with caries of pelvic bones, cellulitis, abscess originating in the abdominal wall, perinephritic abscess, and disease of the hip-joint. Tympanitic resonance on percussion and crepitation from the presence of intestinal gas are diagnostic points.[1] The tympanitic resonance, if ascertained not to be transmitted from the cæcum, distinguishes this abscess from phlegmonous suppuration, which is primarily extra-cæcal; that is, not secondary to perforation. An abscess, however, originating in either the connective tissue behind the cæcum or in the abdominal wall may lead to perforation of the intestine. In the latter case, if the abscess do not discharge its contents into the intestine and a fluctuating tumor remain, the differential diagnosis is not important practically, since the treatment indicated is the same as if the perforation preceded the abscess.

An abscess connected with perforation of the cæcum if treated only by rest, fomentations, and anodyne remedies may open externally, as already stated, and recovery follow. The recovery may be rapid, as in an instance which I have reported.[2] The discharge may take place into the intestine, and recovery may follow closure of the perforation. An opening may take place into the bladder and fecal matter be passed with the urine. I have met with an instance of this kind. There is, however, danger of the discharge of pus into the peritoneal cavity, and consequent fatal peritonitis. The pus is rarely absorbed. It may migrate in different directions for a considerable distance. In a case which I saw with Dr. Damainville of New York, it made its way above the diaphragm into the bronchial tubes, and was discharged by expectoration. The case ended in recovery.

The TREATMENT which has proved successful in the hands of many surgeons in this country is that inaugurated by Prof. Willard Parker in 1867. It consists in removing the pus, either by aspiration or by a free opening, as soon as fluctuation is felt and the presence of pus demonstrated by an exploratory puncture. This method is a highly important improvement in the treatment of perityphlitic or fecal abscess. (Further details belong to surgery.[1])

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[1] Vide an analysis of 32 cases, by Prof. Roberts Bartholow, in the *Amer. Journ. of Med. Sciences*, Oct., 1866.

[2] Vide *Clinical Medicine.*

Absolute rest is to be enjoined until recovery; cathartics are to be avoided, and the patient should be restricted to a purely nutritious diet.

Inflammation and Perforation of the Vermiform Appendix.

The appendix is, in fact, a part of the cæcum, but inflammation and perforation here may occur independently of any affection of the cæcum proper. The term typhlitis is applied to inflammation of the vermiform appendix as well as to cæcitis. Perforation of the cæcum is much more infrequent than of the appendix.

Inflammation of the appendix is caused either by the entrance of irritating substances within its cavity or by distension from fecal matter. The communication of its cavity with that of the cæcum may become closed, and dilatation may result from the accumulation of inflammatory products. It may be dilated so as to form a tumor of considerable size. My colleague, Prof. Janeway, has seen a case in which it formed a tumor as large as a child's head, and another case in which it was as large as a Bologna sausage.[2] Whenever moderately enlarged it may be felt as a tumor through the abdominal walls. The tumor in cases reported by James Jackson, in his *Letters to a Young Physician*, under the title "A Painful Tumor near the Cæcum," was doubtless an enlarged appendix. I have met with a case in which five previous attacks had occurred. In this case the tumor and all the symptoms disappeared after the discharge of pus from the bowels. At the meeting of the International Medical Congress in Copenhagen in 1884 the late Dr. Mahomed related a case in which, after repeated attacks, an abdominal incision was made and a calculus removed from the appendix, the patient recovering and no subsequent attacks taking place.

Perityphlitis and fecal abscess may arise from inflammation and perforation of the appendix. Indeed, these affections are more likely to be connected with inflammation and perforation of the appendix to the cæcum than with diseases of the latter. The treatment is that already considered in connection with inflammation and perforation of the cæcum.

An important question is, whether in cases of intestinal perforation laparotomy be not admissible with a view to closure of the opening into the intestine and the removal of the irritating matter within the peritoneal cavity. In the light of the knowledge recently acquired respecting abdominal sections this operation appears not only warrantable, but advisable as a possible life-saving procedure under circumstances which without it render the prognosis almost of necessity fatal.

Perforation of the appendix, followed by diffuse peritonitis, is often not preceded by symptoms denoting acute inflammation. In cases which have come under my observation the patients generally had had uncomfortable sensations in the iliac region for two or three days, and in many instances had taken some cathartic medicine, thinking that they were "bilious" or needed "clearing out." Suddenly a sharp, lancinating pain was followed by the symptoms of diffuse peritonitis, which proved fatal. At the autopsy, generally within the appendix hard bodies are found to which it is customary to attribute the perforation. These bodies are usually of small size (from a pea to a date-stone), and consist of hard feces, a calcareous material, the seeds or stones of fruit, etc. If nothing be found, the supposition is that the bodies have escaped through the perforation.

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[1] For a table of 60 cases, with operations and results, collected by Dr. William C. Wey, vide *Trans. of Med. Soc. of the State of New York*, 1880.

[2] *N. Y. Med. Record*, April 12, 1884.

Perforation of intestine in cases of diffuse peritonitis is referable, as a rule. to the appendix, if not preceded by notable intestinal or gastric symptoms. The evidence for and against the occurrence of perforation as the cause of diffuse peritonitis will be considered in connection with the latter affection.

In the exceptional instances of recovery when diffuse peritonitis has been thus induced the appendix quickly becomes adherent to adjacent parts, and the perforation is in this way closed. I have met with fatal cases in which the appearances after death showed more or less progress toward this result. In the examination of bodies dead with other diseases old adhesions are not infrequently found. They are probably results of circumscribed peritonitis secondary to disease of the appendix. Adhesion, taking place before perforation occurs, protects against diffuse peritonitis; but fecal abscess may follow, as in cases of perforation of the cæcum. I have seen a specimen in which the appendix was attached to the bladder and perforation had taken place, causing a vesico-intestinal fistula. The first evidence of this was the appearance of a lumbricoid worm at the end of the penis, the patient then being eight years of age. Subsequently, other worms were discharged with the urine, and repeatedly the seeds of fruit. Most of the urine was discharged by the bowels. The fistula for many years gave but little inconvenience. The patient served as a soldier in the late civil war. He was operated upon for stone in the bladder, and died five days after the operation, the fistulous communication of the bladder with the appendix having existed for twenty years.

Pain or uneasiness referred to the right iliac fossa, without other symptoms of disease, should always excite apprehension in the mind of the physician. Rest should be enjoined and cathartics avoided. By these precautions, if patients seek advice prior to perforation, its occurrence may perhaps be prevented.

Colitis—Proctitis.

Inflammation of the colon (colitis) and of the rectum (proctitis) furnishes the anatomical characters of dysentery. But in the latter the inflammation involves a special cause; hence dysentery is with propriety included among the infectious diseases, using the term infectious in the sense now generally adopted. (Vide p. 84.) The terms colitis and proctitis may be applied to inflammation in the colon and in the rectum when it occurs irrespective of a special causation. These terms will then correspond to the ordinary catarrhal inflammation of many authors.

Colitis, in this scope of the term, is rarely acute, but as a subacute affection it is not rare in the adult, and is common in infancy. It enters into the disease known as cholera infantum or the ileo-colitis of childhood. It may or may not be associated with inflammation seated in the small intestine (enteritis). In the cases among soldiers grouped by Woodward under the name of Acute Diarrhœa, inflammation affected especially the large intestine. The local symptoms are colicky pains, diarrhœa, the dejections being loose or watery and containing gelatinous mucus in greater or less quantity, with absence of tenesmus. Tenderness may be present, either limited to or most marked in the situation of the colon. The diagnosis is to be based on these symptoms. Fever, anorexia, and general debility are associated with these symptoms, and are proportionate to the degree and extent of inflammation.

The causes are the accumulation of feces, and especially the presence of undigested irritating matters received from the small intestine. General causes, such as over-fatigue, exposure to cold, insufficient alimentation, intemperance, etc., co-operate with the local causes, increasing the susceptibility to the latter and impairing tolerance. Hence the greater frequency and the greater severity of the disease in military campaigns than in civil life.

The general principles of the treatment are essentially the same as in cases of sporadic dysentery.

There is no practical advantage in endeavoring to distinguish chronic colitis from chronic dysentery, although the distinction may be admissible on anatomical and pathological grounds.

Proctitis occurs from the action of local causes, and it may simulate dysentery as regards the presence of mucus and blood in the dejections, together with tenesmus. Diarrhœa is wanting, and the clinical history in other respects lacks the features of a truly dysenteric disease. Perforation of the rectum sometimes gives rise to periproctitis and fecal abscess. These purely local affections of the rectum are properly surgical.

Reference may be made here to the occasional expulsion, *per rectum*, of membraniform casts of the intestinal tube. These have been observed a foot and more in length. They have been supposed to be the mucous membrane itself exfoliated, but oftener they have been mistaken for fibrinous exudation. They consist of inspissated mucus, and usually do not contain fibrin or coagulable lymph. In a case which came under my observation a number of casts were expelled, one of which was fifteen inches in length, one and a half inches in breadth, and from one-fourth to one-eighth of an inch in thickness. This specimen, examined by Professor Wm. H. Welch, was found to contain fibrillated fibrin, a large number of pus-cells, mucous corpuscles, with bacteria, red blood-corpuscles, and a few cylindrical epithelial cells. Their expulsion is preceded and accompanied by colicky pains and diarrhœa, the dejections containing blood and muco-pus. In most instances the casts are expelled repeatedly after variable intervals. The patients are generally women who manifest hysterical phenomena, and uterine troubles are often associated.

The casts are probably formed in the large intestine, and in connection with other symptoms they denote inflammation. The affection is therefore a variety of colitis. It has received a variety of names. DaCosta, who has reported a number of cases,[1] calls it *membranous enteritis*. The name *tubular diarrhœa* was proposed by John Mason Good, and to this name Woodward gives the preference, as implying no theory. (The reader is referred to the *Medical History of the War of the Rebellion*, p. 363, for a full reference to the literature of this curious intestinal affection.)

Acute Enteritis.

The term *enteritis* signifies, literally, intestinal inflammation. Various significations have been attached to it by different writers. The older writers described under this name inflammation of the serous coat of the intestine, but modern authors, almost without exception, apply the term to inflammation of only the mucous and submucous layers. Many pathologists understand by enteritis inflammation of any part of the intestinal tract, and consider as subdivisions of enteritis duodenitis, jejunitis, ileitis, typhlitis, colitis, and proctitis. There are clinical grounds for distinguishing as separate affections duodenitis (usually in conjunction with gastritis), typhlitis, and proctitis, but it is impossible to distinguish, clinically, jejunitis and ileitis. It has already been remarked that diffuse inflammation of the large intestine is liable to involve the lower part of the ileum; and it is likewise true that inflammation of the small intestine frequently extends into the large intestine. As we already have in the words dysentery and colitis names for inflammation of the large intestine, it has become a conventional usage in this country, and to an extent elsewhere, to apply the term enteritis particularly to inflammation of the small intestine. The fact, however, is recognized that as in dysentery the inflammation often involves the ileum, so in enteritis, with even greater frequency, the inflammation extends to the upper part of the large intestine. It will be found that in most of the cases here described as enteritis the inflammation is most intense in the ileum, the cæcum, and the upper parts of the colon. The inflammation may be, however, nearly or quite confined to the small intestine.

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[1] *American Journal of Medical Sciences*, Oct., 1871.

Anatomical Characters.—As the disease in itself rarely proves fatal, the opportunity of inspecting the appearances after death is not often offered. The changes are essentially the same as those described for acute simple inflammation of the large intestine (p. 464). The mucous membrane presents an arborescent redness, either diffuse or in patches. Ecchymoses within and beneath this membrane may be observed. Its surface is coated with mucus and muco-pus. An increased transudation of serum occurs, which, combined with rapid peristole, causes fluid stools. The mucous and submucous tissues are swollen in consequence of œdema, congestion, and cell-infiltration. The enlargement of the villi gives a plush-like appearance to the surface of the mucous membrane. The solitary and agminated follicles usually are swollen and appear as grayish-white elevations surrounded by a red, congested zone. Microscopical examination shows a variable number of emigrated white blood-corpuscles in the submucous and mucous coats. The same remarks are applicable to the desquamation of the epithelium here as in acute simple inflammation of the large intestine.

Clinical History.—The local symptoms in cases of acute enteritis are pain, tenderness on pressure over the abdomen, especially over the lower part of the ileum and the cæcum, and frequently diarrhœa, with nausea and vomiting. The pain is not intense, and is of a dull, aching character, with exacerbations, in which it resembles the pain of colic. The pain is referred to the umbilical region. The tenderness is not great. Slight pressure is well borne, but deep, firm pressure occasions more or less suffering. Diarrhœa is often, but not always, present. If present, the dejections are more or less frequent, loose, watery, and gelatinous. Tenesmus and the characteristic dejections of dysentery are wanting. I have met with fatal cases in which diarrhœa had not occurred, the inflammation having been limited to the small intestine, as shown by the autopsy. If diarrhœa be a prominent symptom, it probably denotes an extension of the inflammation into the cæcum and colon. Vomiting is generally more or less prominent as a symptom. It may be excited sympathetically, or it may denote an extension of the inflammation to the duodenum and stomach. If jaundice occur it denotes duodenitis. The general symptoms vary considerably in different cases, the variations probably corresponding to the extent as well as the degree of the inflammation. The febrile temperature is in most cases moderate or slight, and the prostration is not great. The intellect is unaffected, except in severe cases, when delirium may occur, together with tympanites, hiccough, and symptoms denoting adynamia. The duration in favorable cases is brief, convalescence being declared in from a week to ten days.

Pathological Character.—Excluding enteritis occurring in connection with typhoid fever, tuberculosis, and the diseases of infancy, the disease has no special pathological character. It is a simple inflammation of a mucous membrane (intestinal catarrh). It occurs only as a sporadic disease, in this respect differing from dysentery. It differs also from dysentery in not leading to destructive lesions of the affected membrane, like those which occur especially in the epidemic form of that disease.

CAUSATION.—The susceptibility of the mucous membrane of the small intestine to acute inflammation after infancy is slight. It may be produced by dietetic excesses, the use of stimulating food and alcoholic drinks, poisoning with acrid substances, and the use of drastic purgatives. It is sometimes attributable to the action of cold upon the body when heated and perspiring, local causes being conjoined therewith. Fatigue and debility may co-operate with these causes.

DIAGNOSIS.—Acute enteritis is to be discriminated from dysentery, gastritis, colic, peritonitis, and typhoid fever. Assuming that the local symptoms are sufficient to denote inflammation somewhere within the abdomen, the absence of the characteristic dysenteric evacuations, of tenesmus, and of tenderness in the tract of the colon suffices to exclude dysentery. The microscopical examination of the stools may aid in determining the seat of the inflammation, as has been shown by Nothnagel. Under normal conditions Gmelin's reaction for bile-pigment with nitroso-nitric acid cannot be obtained in the feces or in the contents of the large intestine. If yellowish balls and clumps of mucus, or yellowish cylindrical epithelial cells which respond to Gmelin's test, be found in the feces, it is evidence of increased peristole, and often of inflammation in the small intestine. The presence of much undigested food, such as abundant starch-granules, in the feces is also evidence of increased peristole in the small intestine.

Nausea and vomiting, if unusually prominent, may suggest acute gastritis. But the symptoms of acute gastritis point distinctly to the stomach as the seat of disease. This disease may generally be excluded without difficulty. The two diseases, however, may be combined. The inflammation in some cases perhaps extends from the small intestine to the stomach; and in cases of poisoning with acrid substances the local effects of the latter may be produced within the small intestine as well as in the stomach.

Colic is a functional affection characterized by intense paroxysmal pain, without tenderness or febrile movement, and is oftener accompanied by constipation than by diarrhœa. These points are sufficient for its exclusion.

As regards the local symptoms, acute enteritis approximates to acute peritonitis more closely than to any other affection; but as a rule there is a marked disparity in the intensity of the symptoms in these two affections. The pain, tenderness, and tympanites common to both are, in the great majority of cases, notably greater in peritonitis. Diarrhœa, which is the rule in enteritis, occurs exceptionally in peritonitis. Rigidity of the abdominal muscles, which is one of the diagnostic symptoms of peritonitis, is wanting in enteritis. Moreover, the constitutional disturbance, as denoted by frequency of the pulse, prostration, etc., is much greater in peritonitis. Attention to these points renders the differential diagnosis easy in most cases.

The abdominal symptoms in typhoid fever may lead the physician to suppose that disease to be simply enteritis, and on the other hand a primary enteritis may be supposed to be typhoid fever. Typhoid fever is to be excluded by the absence of the diagnostic symptoms which attend its development and progress—namely, the characteristic rise of temperature, together with cephalalgia, epistaxis, bronchitis, the rose eruption, and delirium. Moreover, the adynamic symptoms in typhoid fever are generally greater than in simple acute enteritis, while the enteric symptoms are less marked.

PROGNOSIS.—Acute enteritis after infancy in the majority of cases is not a disease of great gravity. If it be not associated with any other important affection, and the patient have not a feeble constitution, a favorable prognosis may generally be entertained. The disease, however, sometimes ends fatally. This is to be apprehended when the pulse becomes feeble and frequent, the prostration marked, and delirium is developed, the local symptoms persisting. The mode of dying is by asthenia, as in cases of dysentery.

TREATMENT.—The indications for treatment may be embraced in a few words. An efficient but non-irritating purgative is generally at first advisable. Afterward opium in some form is to be given sufficiently to relieve pain and diarrhœa. It may be administered by either the mouth or rectum. or alternately in both modes, or by hypodermic injection. It is to be given in doses proportionate to the urgency of the symptoms, and graduated according to its effects. Fomentations over the abdomen by means of poultices. the water-dressing, or spongio-piline are useful. Moderate stimulation of the surface of the abdomen by sinapisms or liniments is appropriate. General or local depletion by bloodletting is very rarely if ever called for. Mercury as an alterative remedy, or given to "improve the secretions," is not to be recommended. The supporting treatment is indicated in proportion to the duration of the disease and the tendency to failure of the vital powers. The diet at first should be restricted and bland, but alimentation is of course important if supporting measures be indicated. The question of irrigation does not enter into the consideration of the treatment of enteritis, inasmuch as the injected liquid does not readily ascend beyond the ileo-cæcal valve.

Subacute and Chronic Enteritis.

Subacute enteritis is of frequent occurrence, arising either from dietetic errors or an arrest of digestion by exposure to cold, over-exertion, etc. It is not easy to distinguish, practically, cases of transient subacute enteritis from cases of functional diarrhœa; nor is it of much practical consequence to make this discrimination, since the indications for treatment in either case do not materially differ, embracing mild purgation, followed by anodyne remedies and regulation of the ingesta.

Enteritis, acute, subacute, and chronic, is described by some authors under the name of *intestinal catarrh.* Here, as in other connections, the term catarrh is not adopted in this work. Authors who treat at length of intestinal catarrh embrace under this head cases which I describe in connection with diarrhœa and intestinal dyspepsia.

Chronic enteritis is usually referable to some prior morbid condition. It very rarely follows acute enteritis. Chronic passive congestion in the portal circulation is a cause of chronic enteritis. It may in this way attend diseases of the liver (such as cirrhosis), of the lungs (such as emphysema, chronic pleuritis), and of the heart (valvular lesions.) All of these affections lead to chronic intestinal inflammation by obstructing the flow of blood through the portal system. The pathological condition is at first chronic passive congestion of the intestines. Bright's disease may be accompanied by chronic enteritis. It is a frequent attendant of tuberculous ulcerations of the intestine, and it may occur in chronic tuberculosis without the presence of intestinal ulcers. Chronic enteritis occurs also as a circumscribed inflammation about other forms of intestinal ulcer, about new growths in the intestine, and above strictures of the gut. The intestinal mucous membrane in chronic enteritis is coated with mucus, and is usually of a slate or ash color from the deposition of pigment. The follicles are often enlarged and pigmented, and they may be the seat of ulcers.

The SYMPTOMS are the same as those in acute enteritis, but they are less marked. Constipation may alternate with diarrhœa. As regards treatment the cause should be, as far as possible, removed. This involves in many cases the treatment of a prior morbid state.

CHAPTER VI.

STRUCTURAL DISEASES OF THE INTESTINE.

Intestinal Ulcers.—Duodenal Ulcer.—Embolism of the Superior Mesenteric Artery.—Carcinoma of the Intestines.—Waxy Degeneration of the Intestines.—Structural Lesions causing Intestinal Obstruction.—Intussusception.—Strangulated Hernia within the Abdomen.—Rotation or Twisting of Intestine.—Compression and Stricture of the Intestine.—Obstruction from Impaction of Feces, Enteroliths, and Foreign Bodies.—Functional Obstruction.

Intestinal Ulcers.

THE important forms of intestinal ulcers are the following: simple inflammatory (including follicular), digestive, dysenteric, typhoid, tuberculous, and syphilitic. Certain rare varieties, as the embolic, the amyloid, the corrosive (from swallowing corrosive poisons), the variolous, and the mycotic (especially in malignant pustule), will receive no further attention.

Ulcers may occupy any part of the intestinal tract. In general it may be said that their favorite seats are the ileum, the cæcum, the sigmoid flexure, and the rectum. Simple inflammatory ulcers, also called catarrhal, have been treated of in connection with dysentery and with enteritis. They include follicular and non-follicular forms. Ulcers produced by pressure of impacted feces are sometimes called stercoral. Similar ulcers may be caused by foreign bodies. These ulcers from pressure are most frequent in the cæcum, vermiform appendix, and sigmoid flexure. They have been considered in connection with typhlitis (see p. 482 *et seq.*). Digestive ulcers occur only in the upper part of the intestine. They are of the same nature as digestive or simple gastric ulcer, and will be considered in the next article, under the head of Duodenal Ulcer. Dysenteric ulcers have already been described. The so-called uræmic ulcers which occur in Bright's disease, and which are sometimes attributed to the irritation of carbonate of ammonia produced from urea in the intestinal tract, are in some cases the result of a dysenteric process. Dickinson has observed small ulcers in the lower part of the ileum in connection with small contracted kidney. Typhoid ulcers will be described subsequently in connection with Typhoid Fever.

Tuberculous intestinal ulcers are very frequently present in cases of pulmonary tuberculosis. They very rarely, if ever, occur independently of tuberculous processes in the lungs. The hypothesis that the intestinal ulcers of phthisical patients are caused by swallowing tuberculous sputa is probable, in view of the fact that these ulcers have been produced by feeding animals with tuberculous substances. Tuberculous ulceration affects by preference the lower part of the small intestine. The solitary follicles and Peyer's patches are usually first involved, but tubercles may develop independently of these structures. The process often extends to the remainder of the small intestine and to the large intestine. The vermiform appendix is very often the seat of tuberculous ulceration. The solitary follicles and the individual follicles of Peyer's patches become swollen, and they undergo, first in their centre, cheesy degeneration. The cheesy mass softens and a little ulcer is produced. This ulcer extends partly by an inflammatory process, but chiefly by the development of tubercles in its edges and base. These tubercles undergo cheesy metamorphosis and softening. The shape of the ulcer may correspond at first to that of a Peyer's patch, but its tendency is to extend transversely in the course of the lymphatics and blood-vessels. This direction is explained by the fact that the tubercles, to whose development the ulcer owes its progress, form chiefly in the walls of these vessels. The appearance of a fully-formed tuberculous ulcer is characteristic. Its long axis is transverse to the axis of the intestine; but this rule is not without exceptions. The ulcer may nearly encircle the lumen of the intestine. Tuberculous ulcers constitute the most frequent and important of the so-called annular or girdle ulcers. The edges of the ulcer are thickened and sinuous, and present an appearance often described as worm-eaten, from the presence of little excavations due to softened tubercles. Tuberculous granulations are usually recognized without difficulty both in the edges and in the base of the ulcer. Tubercles are developed in the vicinity of the ulcers. Patches of little white tuberculous nodules seated in the subserous tissue can generally be seen on the peritoneal surface covering the site of the tubercle. These peritoneal tubercles are of great assistance in the recognition of both the position and the nature of the ulcer. Upon microscopical examination tubercles are found in the neighborhood of the ulcer in the muscular and other coats of the intestine. They are formed chiefly in the outer sheaths of the arteries and in the lymphatic vessels. As a rule, they present the typical giant-celled structure (p. 48). Woodward describes a transverse puckering of the mucous membrane in the neighborhood of tuberculous ulcers, due to a tendency in the ulcer to longitudinal contraction. Partial cicatrization of tuberculous ulcers, resulting even in stenosis, has been observed in several instances. That the tuberculous ulcers ever completely cicatrize is improbable. These ulcers do not often lead to perforation of the intestine, and very rarely indeed to general peritonitis from perforation. Circumscribed peritonitis over the seat of the ulcer is common. Tuberculous ulcers are usually accompanied by more or less inflammation of the surrounding mucous membrane. The mesenteric glands are frequently swollen and tuberculous.

Syphilitic intestinal ulcers are infrequent. In only a small number of cases has their origin from gummata been demonstrated. Syphilitic ulcers of the intestine are either round or transverse. They may form annular or girdle ulcers. Their edges are generally grayish-white, indurated, and fibrous; the base is also indurated and usually without much purulent secretion; and the serous coat over the ulcer is often thickened, and may present cicatricial tissue containing little fibrous nodules. In the thickened walls and base cheesy nodules, which are doubtless gummata, are occasionally detected. The hard fibrous tissue forming the floor and edges of the ulcer is generally densely infiltrated with small round cells. These ulcers may be found in both the large and the small intestine. Their cicatrization leads to more or less contraction of the calibre of the intestine. Submucous gummata have been found with and without ulceration of the intestine, but they are extremely rare. They appear to be more frequent in congenital than in acquired syphilis. Syphilitic ulcers and strictures of the rectum are considered fully in works devoted to venereal diseases. According to Fournier,[1] syphilitic stricture of the rectum may be due not only to the cicatrization of ulcers, but also to a diffuse hyperplastic infiltration of the mucous and submucous coats. To this syphilitic infiltration he gives the name of ano-rectal syphiloma. Birch-Hirschfeld[2] has described as an occasional lesion of congenital syphilis diffuse sclerosis of the intestinal mucous membrane, which was found thickened by an infiltration of fusiform cells.

The SYMPTOMS of intestinal ulceration cannot well be considered apart from the morbid conditions on which the ulcers depend. In general, the symptoms are not characteristic of ulceration. There may be tenderness on pressure if the ulceration be extensive, especially if it be seated in the large intestine. The nutrition of the patient is usually impaired. The character of the stools is often abnormal. There may be diarrhœa, with liquid and often bloody evacuations. On the other hand, constipation may be present, particularly if the ulcers be high up in the bowels. Symptoms are often referable to a coincident enteritis. The hemorrhage from intestinal ulcers may be so profuse or persistent as to prove the immediate cause of a fatal termination, but instances of this are rare. Tubercle bacilli are frequently present in the fecal discharges in cases of tuberculous ulceration of the intestines. In rare instances tuberculous ulcers of the rectum are situated so that they can be explored during life. Ulcers may give rise to perforation and fatal peritonitis. They may cause fistulous communications. If cicatrization take place, constriction of the intestinal canal may be thereby produced, and this stenosis may be the cause of death. Syphilitic ulcers are particularly likely to cause stricture of the intestine, but the same result may follow dysenteric, simple inflammatory, and even in rare instances tuberculous ulcers. Typhoid ulcers do not lead to stenosis. Not very infrequently ulcers are found unexpectedly in post-mortem examinations, there having been no symptoms referable to them during life. The prognosis depends upon the primary morbid condition. The treatment also has reference to this state. In general, the treatment indicated in chronic dysentery is applicable. Ulcers and strictures seated in the lower part of the rectum are open to inspection, and are to be treated by local applications and surgical procedures.

Duodenal Ulcer.

Chronic ulcer has the same anatomical characters in the duodenum as in the stomach, and is probably, like the latter, caused by the solvent action of the gastric juice. It is far less frequent than gastric ulcer, the proportion being as 1 to 30 of the latter. The situation is usually near the pyloric opening. In contrast with gastric ulcer the ages of patients are generally between thirty and forty, and duodenal ulcer occurs much oftener in men than in women. These are points to be taken into account in the diagnosis. The fact of its occurrence in some cases after severe burns of the skin is also to be borne in mind.

The local symptoms by which it is to be differentiated from gastric ulcer are as follows: Vomiting is often wanting, and when it occurs the intervals between its occurrence and the ingestion of food are longer. The ingestion of food does not occasion the same degree of gastric disturbance and consequent distress. Paroxysms of pain often occur having the severity and character of gastralgia. The pain, as in gastralgia, is sometimes relieved by the taking of food. The pain is referred to the right hypochondrium. Jaundice sometimes, but rarely, is associated with duodenal ulcer. From the occurrence of this symptom either duodenitis or the situation of the ulcer near the opening of the ductus communis is to be inferred. Hemorrhage is as liable to occur as in cases of gastric ulcer. Blood may be vomited, but it is passed in larger quantity by the bowels. These points relating to hemorrhage are significant as regards the diagnosis. Cancer is excluded by the absence of tumor, of the gastric symptoms usually connected with that affection, and of the appearance characteristic of the cancerous cachexia. Moreover, cancer usually occurs at a later period of life—namely, between forty and sixty years of age.

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[1] *Leçons rédigées par Ch. Porak*, Paris, 1875.
[2] *Wiener Med. Presse*, 1878, No. 41.

Duodenal ulcers may cicatrize, giving rise to stenosis and consequent dilatation of the stomach; but cicatrization is rare. Perforation may take place and give rise to fatal peritonitis. This result is sometimes prevented by adhesions of the duodenum over the site of the ulcer prior to the perforation. These adhesions may take place and rupture afterward occur, as in a case which was under my observation. Duodenal like gastric ulcer may remain latent until perforation occurs.

The TREATMENT is essentially the same as in cases of gastric ulcer.

Embolism of the Superior Mesenteric Artery.

Embolism of the superior mesenteric artery is followed by hemorrhagic infarction of the small intestine. Although this artery anastomoses freely with the pancreatico-duodenal and with the inferior mesenteric, it has been shown by the experiments of Litten[1] that its branches are so little tolerant of an insufficient supply of blood that their coats undergo a molecular alteration which suffices for the production of a hemorrhagic infarction before a complete collateral circulation can be established. The wide extent of territory over which this vessel is distributed is an impediment to the rapid development of a sufficient collateral circulation, which, even in arteries more favorably situated, often requires considerable time. About thirty cases of embolism of the main trunk or of the branches of the superior mesenteric artery have been recorded. The embolus itself can usually be detected post-mortem without difficulty. The parts affected by embolism of the main trunk are the greater portion of the small intestine and the upper part of the large intestine. The extent of intestine involved of course depends upon the size of the branch obstructed. The wall of the affected portion of intestine is swollen, œdematous, and more or less infiltrated with blood. There may be in addition large and small circumscribed hemorrhages in the intestinal coats. The mucous membrane especially is swollen, soft, and infiltrated with blood. Its surface is coated with a layer of blood or of blood-stained mucus. In certain places there may be loss of substance from necrosis of the mucosa. The solitary follicles, Peyer's patches, and the mesenteric glands are frequently swollen. When the main trunk of the artery is occluded there are hemorrhages also in the mesentery. At first circumscribed peritonitis and subsequently general peritonitis result. If the artery become slowly obstructed, as by pressure, no effects follow.

The SYMPTOMS have been studied chiefly by Gerhardt and by Kussmaul.[2] Those to which they attribute diagnostic importance are the following: 1. A source for an embolus (organic disease of the heart was usually present). 2. Abundant intestinal hemorrhages, which cannot be explained by an independent disease of the intestine or by portal obstruction. 3. Rapid fall of temperature. 4. Pain in the abdomen, often resembling colic and often violent. 5. After a time development of tympanites, and perhaps of a recognizable exudation in the peritoneal cavity. 6. The evidence of embolism of other arteries of the body. 7. In rare instances the detection by palpation of sacs containing blood between the folds of the mesentery. Vomiting was present in many of the cases reported. The bloody stools often had a fetid odor. The blood was sometimes of a tarry consistence, and at other times it was fluid or in clots. Occasionally no hemorrhage occurred. In that case the diagnosis is impossible. The symptoms develop suddenly, and death usually soon follows, preceded by collapse. It does not seem to be absolutely fatal. In a case reported by Moos which recovered the diagnosis of this affection was rendered very probable. The diagnosis in the favorable cases of Lereboullet was not so well established.

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[1] *Virchow's Archiv*, 1875, Bd. 63, S. 289.

[2] *Würzburger medic. Zeitschrift*, 1864, Bd. 4 u. 5.

The objects of TREATMENT, as stated by Gerhardt, are to check the hemorrhage, to prevent necrotic processes in the intestine, to repair the loss of blood, and to favor the re-establishment of a collateral circulation. He says: "In what way these indications are to be best fulfilled future experience must decide." Moos treated his case which recovered by rest, lukewarm nourishing broths, small doses of wine, and by the internal and external application of ice. A certain degree of doubt necessarily attaches to the diagnosis of this case.

Embolism of the inferior mesenteric artery is sometimes unattended by symptoms; at other times symptoms similar to those described were present. Only about eight cases have been recorded.

Carcinoma of the Intestine.

Cancer of the intestine presents the same varieties as when seated in the stomach. The most frequent situation by far is the rectum. Here it is open to discovery and examination by the touch and vision. Next to the rectum, it is most frequently situated at the sigmoid flexure of the colon; and next in the transverse colon. It is situated in the duodenum or jejunum, the lower portion of the ileum, the descending colon, the ascending colon, and the middle of the ileum, as regards relative frequency in the order in which these parts are now enumerated. In these different situations it often produces obstruction by compression and stricture.

The cancerous growth, when it has reached a certain extent, if situated above the rectum, forms a tumor which may be felt through the abdominal walls. The problem is then whether or not the tumor be carcinoma. The diagnosis is to be based on the existence of radiating, lancinating pains, the age of the patient (over forty years), the frequently nodulated character of the tumor, the occurrence of bloody stools, the evidence of hereditary predisposition, and the symptoms which denote the cancerous cachexia. Fecal tumors, enteroliths, and foreign bodies are to be excluded; also, sarcomas and the benign neoplastic growths—namely, fibromas, myomas, lipomas, cysts, and adenomas. These as well as cancerous growths may produce obstruction at different situations in the intestinal canal. They cannot in cases of obstruction be excluded with certainty unless they be situated in the rectum. They cannot be differentiated from each other during life, and hence they are of pathological rather than of clinical interest. This brief reference to them, therefore, is all that is called for. Duodenal cancer is not easily differentiated from cancer of the stomach. The differentiation is not practically of importance. The association of jaundice points to the former.

Waxy Degeneration of the Intestine.

The character of the lesion known as waxy, lardaceous, and amyloid has been considered in the first part of this work. (Vide p. 55). The intestine as well as the stomach is not infrequently the seat of this structural change. The alteration is generally present throughout both the large and the small intestine, but it is usually most intense in the lower part of the ileum. The mucous membrane may be the coat chiefly affected, or in addition the other coats may be involved. Usually the solitary follicles and Peyer's patches escape or are but slightly affected. In certain cases, however, the waxy change is most marked in these structures (Hayem). Neumann observed cases in which the muscularis mucosæ was predominantly affected. Waxy degeneration is not to be positively recognized post-mortem without the application of iodine or other appropriate reagents. Simple inflammation of the mucosa is generally present in cases of waxy degeneration. Kyber denies that there is any tendency in this alteration to the production of ulcers. Tuberculous ulcerations are often present when the waxy metamorphosis is secondary to phthisis.

Waxy degeneration may be inferred when diarrhœa is a prominent and persistent symptom in connection with waxy degeneration of the liver, spleen, or kidneys, these organs being affected either singly or collectively, the latter being often the case. The lesion is rarely confined to the alimentary canal, and if so confined the diagnosis is impossible.

The lesion is a permanent one, and admits of only palliative treatment.

Certain structural affections of the intestine are important as causing mechanical obstruction to the passage of the intestinal contents. Hence, we may consider *obstruction of the bowels* or *obstipation* as caused, *first*, by structural affections; and *second*, as dependent on causes other than lesions of structure—namely, on the impaction of feces, the formation of concretions called enteroliths, the introduction of foreign bodies, and on merely functional disorder. The term ileus or iliac passion and other names have been applied to cases of obstipation with the ejection from the mouth of the intestinal contents, or so-called stercoraceous vomiting. As the latter symptom occurs in connection with obstruction from various causes, the names based upon it have no special pathological significance, and they tend to produce confusion.

Obstruction from structural affections is not of very frequent occurrence. The statistical researches by Brinton, the whole number of promiscuous necropsies being 12,000, showed obstructions from various causes, exclusive of hernia, in the proportion of 1 in 280 deaths.[1] The lesions involving obstruction are the following: Intussusception or invagination; strangulation from hernia within the abdomen; rotation of a portion of intestine upon its own axis or upon the mesentery, and twisting of a portion of intestine around another coil of intestine; compression of one portion of intestine by another portion or by a tumor situated exterior to the canal; and stricture produced by morbid growths within the canal or by the contraction following the cicatrization of ulcers. In these several affections obstipation is a common feature; but they differ in an important point—namely, certain of them causing only mechanical obstruction, and others involving in addition strangulation. This point of difference is to be taken into account with reference to diagnosis, prognosis, and treatment. The structural affections giving rise to both obstruction and strangulation will be first considered, and afterward obstruction by causes which do not occasion strangulation.

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[1] *British and Foreign Medico-Chirurg. Review*, October, 1857.

Intussusception.

Intussusception, or intestinal invagination, is the reception of one portion of the intestine into another portion. Generally, the invagination is from above downward, but in rare instances the lower portion is received into the upper. The latter, called retrograde invagination, according to Leichtenstern, never becomes incarcerated and strangulated. It is certain that invagination may occur transiently, giving rise to no symptoms. In post-mortem examinations, especially in children, innocuous invaginations of the small intestine are not infrequently found; the invaginated portion is restored without difficulty, and, aside from the displacement, there are no morbid appearances. I counted as many as fifteen such invaginations in the body of a child dead with typhoid fever. They have been observed especially after death from cerebral diseases, and they are supposed to occur during the last moments of life. It is only when the invaginated portion becomes incarcerated, giving rise to congestion, inflammation, etc., that obstruction and the symptoms of strangulation are produced. The lesion is then one of very great gravity, in the great majority of cases ending fatally. This is the most frequent of the lesions causing fatal obstruction. Of 169 cases of obstruction from various lesions collected by Phillips, 63 were of this class. An analysis of 600 cases by Brinton showed in 43 per cent. the obstruction to be from intussusception; and of 1541 cases of obstruction from all causes analyzed by Leichtenstern, 442 were due to this cause.[1] The greater relative frequency of the affection, however, relates to childhood. In adults it is less frequent than other causes of obstruction.

Intussusception brings into apposition three layers of intestine—namely, an entering, a returning, and a receiving layer. The receiving layer is called the intussuscipiens, and the middle and inner layer the intussusceptum. In the relation of the returning and receiving layers mucous surfaces are in contact, and these do not become adherent; but serous surfaces are in contact in the relation of the entering and returning layers, and inflammation, excited at the point of entrance, leads to adhesion of these surfaces extending more or less beyond this point. In this way the invagination becomes incarcerated. The vessels of the portion of the mesentery connected with the invaginated intestine are obstructed by tension. Congestion and swelling of the invaginated intestine ensue; it is dark-colored and ecchymotic, and at length it becomes gangrenous and sloughs away if life be sufficiently prolonged. The obstruction is due to the swelling from congestion and the exudation of inflammatory products. Usually, the obstruction is complete, but in rare cases the intestinal passage remains pervious although much contracted. In these cases the intussusception is usually in the large intestine. Peritonitis may extend more or less around the seat of the invagination, leading to morbid attachment to the adjacent parts.

The invagination may occur at any point in the intestinal tract. Its most frequent seat is at the junction of the ileum and the cæcum. This is shown in cases of children by the statistical researches of J. Lewis Smith.[2] Smith's account of the mode in which it generally occurs is as follows: "The intussusception not infrequently begins in the prolapse of the ileum through the ileo-cæcal valve, in the same way that prolapse of the rectum occurs through the sphincter ani. If death take place early, only a small portion of the ileum may have passed the valve. If the case be protracted, the tenesmus brings down more and more of the ileum with its accompanying mesentery. The constriction of the valve, which acts as a ligature, prevents the further descent of the ileum, and, the tenesmus continuing, the next step is the inversion of the caput coli, which is drawn into the colon by the descending mass, and unless the case terminate by sloughing or death the ascending and transverse portions of the colon are successively invaginated. Not infrequently the cæcum is the part primarily inverted and invaginated, and, descending along the colon, it draws after it the ileum, which sustains its natural relation to the ileo-cæcal valve. These two forms of invagination—that in which the ileum, passing through the ileo-cæcal valve, successively inverts and draws after it the caput coli and the divisions of the colon, and that in which the caput coli is primarily invaginated, and, descending along the large intestine, inverts the latter and draws after it the ileum—constitute the vast majority of cases of this disease in childhood." The invaginated portion may descend so low as to be felt and seen at the anus, and it may even protrude from the body. The invagination, however, may occur at any point in either the large or the small intestine. Of the cases analyzed by Brinton, the intussusception was ileo-cæcal in 56 per cent., iliac in 28 per cent., jejunal in 4 per cent., and in the colon in 12 per cent. Leichtenstern's statistics of 479 cases gave 52 per cent. of ileo-cæcal and ileo-colic, 30 per cent. of iliac, and 18 per cent. of colic invaginations.

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[1] Vide *Ziemssen's Cyclopædia*, Am. ed., vol. vii.

[2] "Statistical Researches relative to the Seat, Symptoms, Pathological Anatomy, etc. of Intussusception in Children," *Am. Journ. of Med. Sciences*, Jan., 1862. Vide also *Smith on the Diseases of Children*.

The probable causation in the majority of cases is formularized in the following quotation from Leichtenstern: "Paresis of a limited portion of the intestine, associated with vigorous peristaltic action excited by any cause whatever, offers suitable conditions for invagination." In adults the most frequent cause is a tumor of the intestine, particularly a polyp-shaped tumor, projecting within the intestinal canal. Such a tumor by its weight drags down a portion of the intestine and thus mechanically causes intussusception.

Intussusception causing obstruction and strangulation, as already stated, proves fatal in the great majority of cases; yet the affection is by no means absolutely hopeless. The manner in which recovery usually takes place is of importance in its bearing on the treatment. The invaginated portion of intestine sloughs away and is evacuated, the entering and receiving portion at the point of entrance remaining adherent and the perviousness of the canal being restored. Cases have been reported in which a large portion of invaginated intestine was thrown off, and recovery followed. Prof. Van Buren reported a case to the New York Pathological Society in which five feet of intestine were passed per anum, the patient recovering. Prof. Peaslee exhibited at a meeting of the New York Academy of Medicine, in 1865, five feet of intestine which had been passed per anum four months before the death of the patient. He also exhibited the intestines removed from the body of this patient after death. The small intestine was only sixteen feet in length, the length of the large intestine being five feet and ten inches. The invagination and sloughing had taken place in the small intestine at a distance of six feet from the duodenum. A stricture existed at this point; the intestine above was much dilated, and that below was greatly reduced in size. The patient died from inanition dependent on the stricture of the intestine.[1] Dr. William Thompson of Edinburgh collected 43 cases ending in recovery.[2] Of Dr. Haven's 59 cases, in 12 the invaginated portion was passed per anum, and of these 12 cases 10 ended in recovery. This is the mode of recovery which is to be hoped for after incarceration and strangulation of the invaginated intestine have taken place.

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[1] Vide *Bulletin of the New York Academy of Medicine*, vol. ii. Nos. 25–29.

[2] Vide article on "Internal Strangulated Intestine," by Elisha Harris, M. D., in *New York Journal of Medicine*, 1853.

The prominent local symptoms which enter into the clinical history of intussusception are the following: Pain is more or less prominent, at first paroxysmal as in colic, and after a time becoming constant, with frequent exacerbations. The pain, if the patient be old enough to describe it, appears to emanate from a certain fixed point. Tenderness at first may not be marked, but becomes developed, being either limited to or greatest at the point whence the pain emanates. The tenderness and constant pain denote the occurrence of peritonitis at the seat of the invagination. Vomiting soon becomes a prominent and persistent symptom, with very few exceptions. The vomited matter after a time may have the odor of feces, and is then said to be stercoraceous. The accumulation of ingesta and gas above the obstruction occasions more or less abdominal distension, provided the invagination be not situated in the upper portion of the small intestine. Frequently, the accumulation of intestinal contents at the seat of the invagination gives rise to a tumor appreciable by the eye and touch, and to dulness on percussion. The tumor is elongated, "sausage-shaped," corresponding to the size and direction of the dilated intestine above the seat of the obstruction. After the contents of the intestinal canal below the seat of the obstruction have been evacuated there is persisting obstipation, save in a few exceptional cases. Previously there is usually diarrhœa, followed by the discharge of bloody mucus, and if the invaginated portion of intestine descend to the rectum, tenesmus is felt and the patient is led to make straining efferts. Under these circumstances the affection is liable to be mistaken for dysentery.

The general symptoms at first may not be marked, but soon they denote a grave affection. The pulse becomes accelerated, its frequency progressively increases, and it is proportionally feeble or compressible. Progressive prostration, hiccough, coolness or coldness of the surface, and an anxious or haggard expression, denote the progress toward a fatal termination. The mode of dying is by asthenia, the pain and tenderness frequently diminishing or ceasing for some time before death.

A favorable progress is denoted by the occurrence of free evacuations from the bowels, the discharge of the invaginated portion of intestine, together with improvement in the local and in the general symptoms.

Invagination is to be distinguished from functional colic, acute peritonitis, and obstruction from other causes. The symptoms at first may denote nothing more than colic. The persistency of the pain, the development of tenderness, the acceleration of the pulse, the vomiting, etc., however, soon point to an affection of greater gravity than colic. Acute peritonitis is to be excluded by the gradual development of the local and general symptoms; by the absence of diffused tenderness over the abdomen and of rigidity of the abdominal muscles; by the localization of pain and tenderness within a circumscribed space; by stercoraceous vomiting; and by the presence of a characteristic tumor. Acute peritonitis sometimes supervenes in cases of invagination from rupture of the intestine above the obstruction. From obstruction caused by hernia within the abdomen or by rotation and twisting of the intestine the discrimination is more difficult, and, indeed, cannot be made with positiveness. The age of the patient has a bearing on the diagnosis. Invagination occurs most frequently in infancy. According to Smith's statistics, it is most liable to occur between the second and third months of infantile life. Of 47 cases, only 18 occurred between the ages of one year and twelve years. In Haven's cases the mean age was eighteen years; the youngest age was three months, and the oldest sixty-five years. In Leichtenstern's statistics half of the whole number of cases (473) occurred during the first ten years of life; and one-fourth occurred after three months and within the first year of life. Male children are somewhat more liable to it than female, the relative proportion in Smith's cases being 32 to 22, and in Haven's 59 cases 34 were males. On the other hand, hernial obstruction occurs oftener after infancy and childhood, and cases occur oftener in females than in males.

A point in diagnosis is to determine the probable seat of the invagination. The chances are that it is seated at the junction of the large and small intestine, especially in childhood. The probability of this being the seat is increased if the pain, tenderness, and swelling are situated in the neighborhood of the right iliac fossa. Tenesmus, with the discharge of blood and mucus, shows that the large intestine is involved. The invaginated portion may sometimes be felt and seen within the rectum. If seated in the small intestine, the constitutional disturbance is greater, and if the point of obstruction be toward the upper portion of the small intestine, there will be little or no abdominal distension. Indican is found in abnormal quantity in the urine in case of intestinal obstruction from intussusception and other causes, but also in other affections of the alimentary canal.[1]

The PROGNOSIS is extremely unfavorable. The usual mode of recovery in the exceptional cases in which the affection does not end fatally has been stated—namely, by sloughing away of the invaginated portion of intestine, the adhesions at the point of entrance being permanent. Possibly the invaginated portion is spontaneously restored in some cases after symptoms denoting the affection are developed, but such instances must be rare; and it is also rare for the invaginated portion of intestine to remain, the canal becoming or continuing pervious. Of the 50 cases analyzed by Dr. Smith, in 7 recovery took place, in each case by sloughing. In all these 7 cases the ages were between five and twelve years, Dr. Smith's researches being limited to cases occurring in childhood. The constitutional powers in infancy do not seem to be adequate to support the affection for a sufficient period for the sloughing process to be completed. The separation of the invaginated portion of intestine takes place, according to Leichtenstern, in the majority of cases between the eleventh and the twenty-first day. In fatal cases the duration rarely extends beyond eight days. Death may take place within twenty-four hours, the symptoms denoting "shock." In the majority of cases life is not prolonged beyond the third day. In rare instances, the obstruction being incomplete and the invaginated intestine not strangulated, the affection is tolerated for many weeks and months. There are cases, therefore, of chronic intussusception. Death sometimes is hastened by peritonitis caused by rupture of the intestine above the seat of the obstruction. In young children the duration is sometimes shortened by the occurrence of convulsions. Generally, the mode of dying is by slow asthenia.

If the existence of invagination could be ascertained prior to the development of symptoms denoting incarceration, it is probable that measures for restoration might be successfully employed; but generally a day or two elapses before the character of the affection is even suspected. In the mean time cathartics are likely to increase the invagination and diminish the practicability of restoration. The liability to the existence of this affection, especially in children, should be borne in mind in cases of apparent colic, and should dictate reserve in the employment of cathartics. As soon as the character of the affection is rendered probable by the symptoms measures to effect restoration may be tried. These measures are the injection of water or of air in considerable quantity into the rectum. If practicable, the injection should be made through a long flexible tube carried into the intestine as far as it can be made to pass without undue force. The object is to effect the restoration by the upward pressure of the air or water. The injections are not to be pushed beyond the limit at which they are borne without much suffering, and if they do not succeed after a fair trial they are not to be persisted in. They will very rarely succeed after the invaginated portion of intestine has become swollen by congestion and the peritoneal surfaces in contact have become adherent. If pushed too far, rupture of the intestine below the seat of the obstruction may be produced. I have known rupture to result from the injection successively of an alkaline carbonate and an acid, giving rise to the evolution of gas within the intestine. This method of employing pressure is highly objectionable, because the amount of pressure cannot be regulated. Cases have been repeatedly reported in which the injection of air or water has apparently effected restoration in cases of supposed invagination even after obstruction had existed for several days; but it is probable that in a certain proportion of these cases invagination did not really exist. These measures for reduction are of no avail if the seat of the invagination be above the ileo-cæcal valve.

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[1] Vide article by Heineman, *Archives of Medicine*, New York, August, 1880.

Exclusive of measures for reduction, the objects of treatment are to secure as much quietude of the intestinal canal as possible, to palliate suffering, and to support the powers of life, under the hope that sloughing and recovery may take place. The avoidance of cathartics is essential. The practitioner is not to be tempted to employ them with the hope of overcoming the obstruction. They interfere with the objects just named, and are destructive. Not only the active cathartics, but even mild purgatives and laxatives, are contra-indicated. To quiet the peristaltic movements opium is to be given in sufficient doses to secure relief of pain without inducing narcotism. Fomentations over the abdomen are useful. The strength of the patient is to be supported by concentrated and purely nutritious food given in small quantities at a time, together with alcohol. It is to be borne in mind that recovery will depend on life being sufficiently prolonged for the sloughing away of the invaginated portion of intestine. Bloodletting, counter-irritation, or any measures which tend to impair the vital forces are never indicated, and they cannot fail to do harm. After sloughing has taken place and the obstruction has been removed, purgatives are not to be employed, lest by exciting unduly peristaltic movements the adhesions at the point of the invagination may be broken up and peritonitis ensue from the escape of the contents of the intestine into the peritoneal sac.

Important practical questions relate to the operation of abdominal section, or laparotomy, in cases of invagination. Heretofore this operation has been resorted to as a last resource, and for this reason its merits were not fairly represented by its results. The first object of the operation is to restore the parts if practicable, and, if this be not practicable, a second object is to perform enterotomy and establish an artificial anus. The chances for restoration are greater in proportion as the operation is resorted to early. After strangulation has existed for some time it would not be advisable to restore the invaginated intestine, even were this practicable. After the time has elapsed for the practicability or safety of the restoration, the chances of recovery by sloughing away of the invaginated intestine are lessened by the operation, and it would have been better to have trusted to Nature's method. Taking into view this consideration, together with more or less uncertainty as regards the diagnosis, surgeons have not been disposed to regard the operation with favor, and in the early editions of this work doubts concerning its propriety were expressed. Within the past few years, however, experience has shown that laying open the peritoneal cavity is not attended with the danger formerly attributed thereto, and a number of cases have been reported in which the operation for invagination has been resorted to with success. Professor Sands, in 1877, operated successfully in the case of an infant six months old, eighteen hours after the occurrence of symptoms denoting invagination.

In connection with his report of this case he gives an analysis of 20 cases collected from different sources, in 7 of which the operation was successful.[1] The important points for decision respecting the operation in individual cases are the correctness of the diagnosis and the probability or otherwise that the invaginated intestine has not become gangrenous. The probable seat of the invagination is of importance with reference to enterotomy.

As a palliative measure, if the distension of the abdomen from tympanites become excessive, interfering with respiration, it has been recommended to pass an aspirating needle into the intestine and remove the gas. This procedure is not devoid of danger. Prof. Welch has met with an instance in which fatal peritonitis was thereby induced, fecal gas and thin feces escaping into the peritoneal cavity.

Reference has been already made to cases in which invagination may not involve complete obstruction and is tolerated for several months. These are, of course, cases of invagination without strangulation. A remarkable instance has been reported by Dr. C. A. Leale. The patient was a woman seventy-nine years of age. An attack of severe abdominal pain was followed by jaundice, bloody stools, tenesmus, complete fecal obstruction, and from time to time vomiting of intestinal contents. Five weeks afterward, on exploration of the rectum, an invaginated portion of intestine was felt. The finger, after considerable effort, was passed into the latter, and two quarts of pasty feces were subsequently voided. Eleven days afterward a solid body was felt at the opening of the invaginated bowel. This was broken into two pieces, and with considerable difficulty it was removed. It proved to be an enterolith four and a quarter inches in circumference and two and a half inches in length, formed by concentric layers of stercoraceous matter around a biliary calculus three and a half inches in circumference. Restoration of the bowel was speedily effected by the injection of carbonic-acid water in large quantity. This patient was seen by me in consultation.[2] Jonathan Hutchinson relates the case of a child in whom the invaginated bowel extended several inches beyond the anus. The case had been treated as one of prolapsus ani. Abdominal section was made and the bowel was restored with ease. Rapid recovery took place. The invagination in this case began at the cæcum.

Strangulated Hernia within the Abdomen—Rotation or Twisting of Intestine.

Several abnormal conditions lead to *hernia* within the abdomen. Fissures sometimes exist in the mesentery, meso-colon, omentum, and diaphragm, into which a portion of intestine passes and is liable to become incarcerated and strangulated. The vermiform appendix of the cæcum sometimes becomes adherent to an adjacent part, leaving a loop through which intestine may pass and become ligated. The same may happen in connection with diverticula of the intestinal tube, which are not very infrequent. The most frequent and important of these is that known as Meckel's diverticulum. This represents a portion of the unobliterated omphalo-mesenteric canal, by which in the embryo the umbilical vesicle communicates with the intestine. It is connected with the ileum usually from a foot to three feet above the ileocæcal valve. From its extremity a solid cord containing the remains of the omphalo-mesenteric vessels may pass to the umbilicus. Bands of adventitious membrane, resulting from peritonitis, may leave spaces for hernial protrusions. Various other forms of internal hernias have been described, some of which, being very rare, are to be regarded as curiosities rather than of practical importance. Much has been written concerning the so-called duodeno-jejunal hernia, of which between fifty and sixty cases have been reported. In this form of hernia, first accurately described by Treitz, the small intestine makes its way into the duodeno-jejunal fossa, which normally is a small pouch situated in front of the vertebræ at the beginning of the mesentery. A crescent-shaped fold of peritoneum, passing over the duodeno-jejunal flexure, bounds the free opening of this fossa, which is directed upward. All of the small intestine has been known to push its way into this fossa, which then becomes enormously distended. The intestine lies behind the peritoneum in this form of hernia. A very rare form of internal hernia is produced by the passage of portions of the small intestine through the foramen of Winslow into the cavity of the lesser omentum. Diaphragmatic hernia is more frequent on the left than on the right side. The intestines pass more frequently through the muscular than through the tendinous part of the diaphragm, and oftener through the posterior than the anterior part. The organs usually involved in diaphragmatic hernia are, in the order of frequency, the stomach, the transverse colon, the omentum, and the small intestine. The hernia may be due to a congenital defect in the diaphragm. Acquired diaphragmatic hernia is most frequently referable to traumatism, as to a stab or gunshot wound. Rare forms of hernia which are not, strictly speaking, internal, but which are often difficult to detect by external examination, are the ischiatic, the obturator, the perineal, the lumbar. As regards the frequency of the different varieties of internal hernia, of Leichtenstern's 1541 cases, embracing obstruction from all causes, the strangulation was caused by false ligaments in 111, by the omentum in 58, by diverticulæ in 66, by the appendix vermiformis in 34, by the diaphragm in 215, and in other ways in 60. The cases collectively amount to 544.

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[1] Vide *New York Medical Journal*, June, 1877; vide also article by Dr. John Ashhurst, Jr., in the *Amer. Journ. Med. Sciences*, July, 1874; also article by Jonathan Hutchinson, in the *London Med. Times and Gazette*, No. 29, 1873, and in the *New York Med. Record*, January 15, 1874. Peyrot has collected 23 cases, of which 9 recovered: *De l'Intervention chirurgicale dans l'Obstruction intestinale*, Paris, 1880. This work contains an analysis of 125 laparotomies for intestinal obstruction.

[2] For the report of Dr. Leale's case, vide *New York Med. Record*, November 22, 1879.

Whatever be the mode in which the strangulation is produced, the symptoms are the same. They are, in fact, identical with the symptoms of ordinary strangulated hernia occurring at the femoral or the inguinal outlet, and they are for the most part the same as in cases of intussusception. The existence of tenesmus with bloody and mucous evacuations in certain cases of the latter constitute the chief point of distinction. Irrespective of the symptoms just named, the differential diagnosis can hardly be made with positiveness, but the chances are that in the young child invagination exists, whereas in after-life obstruction from other causes is more liable to occur. The small intestine is far more likely to be the seat of hernia within the abdomen than the large intestine. Sloughing of the strangulated portion of the intestine is destructive, whereas in cases of invagination it is the mode of cure. The only hope of recovery is in the spontaneous or operative reduction of the hernia before gangrene takes place. Distending the large intestine with air or water will probably be useless if the seat of the hernia be in the small intestine. Cathartics do harm. Opiates to palliate pain, fomentations to the abdomen, and supporting measures are indicated.

Rotation, or twisting of a portion of intestine (volvulus), so as to induce strangulation, is most liable to occur at the sigmoid flexure of the colon. An unusually small root of the sigmoid meso-colon favors torsion of this portion of the intestine. The root of the meso-colon may be contracted by cicatricial tissue from an old peritonitis. Similar contractions may occur elsewhere. Torsion may occur at the cæcum, this portion of the large intestine being exceptionally mobile. Twisting of a portion of intestine around an axis formed of mesentery or around another coil of intestine occurs in the small intestine, or a coil of intestine may become looped around the sigmoid flexure, and *vice versâ*. These accidents are infrequent. Their symptoms are those of strangulation, and it is impossible to discriminate them from hernia within the abdomen. In the analysis of 600 fatal cases of obstruction from various causes exclusive of hernia, rotation or twisting of intestine occurred in 8 per cent. In Leichtenstern's 1541 cases, 54 were in this category. In cases of obstruction and strangulation exclusive of invagination the propriety of resorting to laparotomy is to be considered. Ashhurst (*op. cit.*) collected 57 cases in which this operation was performed. Of these cases, 18 terminated successfully, giving a mortality of a little over 68 per cent. It is in favor of resorting to the operation in these cases that the prospect of recovery without it is much less than in cases of intussusception. Ashhurst's conclusion embodies a proper rule of practice in these cases. It is as follows: "In cases of acute intestinal obstruction from other causes than intussusception, should milder measures fail to give relief in the course of three, or at most four, days, laparotomy should be unhesitatingly recommended, and may under such circumstances be resorted to with a reasonable hope of success."

A highly important injunction is to be added to the consideration of obstruction dependent on the foregoing structural affections: It is, that the practitioner in all cases which present the symptoms of these affections examine carefully for inguinal or femoral hernia. The hernial tumor may be so small as not to excite the attention of the patient, and in females motives of delicacy may prevent the patient from suggesting an examination even when the presence of a tumor is known to her. Instances have repeatedly occurred of patients dying with a strangulated femoral or inguinal hernia which was overlooked, the symptoms being attributed to some inaccessible lesion or even to functional disorder.

Compression and Stricture of Intestine.

Obstruction due to closure or diminished calibre of the intestinal tube by morbid growths within the tube, the pressure of tumors situated exterior to the tube, or constriction following the cicatrization of ulcers, differs from obstruction caused by the structural affections already considered in not involving strangulation. The obstruction under these circumstances is developed gradually, having been preceded by progressively increasing constipation. Above the seat of the obstruction the intestine is dilated and filled with accumulated fecal matter. The muscular coat is hypertrophied. Below the obstruction the intestine is small, collapsed, and often atrophied. The obstruction is rarely complete. The local and constitutional symptoms incident to inflammation, sphacelation, and sloughing of the intestine are wanting. The symptoms are those arising from the gradual accumulation of the contents of the intestine above the point of obstruction. The affection progresses more slowly toward a fatal termination after the obstruction becomes nearly or quite complete than the affections which involve strangulation.

Cases of obstruction falling under this head form a considerable proportion of the cases of obstruction from all causes. Stricture of the intestine may be due to cicatrization of dysenteric, of syphilitic, of digestive, and of simple inflammatory ulcers, and in rare instances to partial cicatrization of tuberculous ulcers, to cicatrization of ulcers produced by irritant poisons, and of ulcers caused by sloughing of an invaginated portion of intestine. In exceptional cases cicatrization of unusually extensive and deep-seated typhoid ulcers may be followed by stenosis. The contraction of newly-formed connective tissue resulting from circumscribed or from general peritonitis may cause constriction of the intestine. Both benign and malignant tumors may produce intestinal stenosis, either by pressure or by invading in their growth the calibre of the intestine. The consideration of syphilitic and other strictures of the rectum belongs to surgery. In the majority of cases stricture is due to cancer of the intestines. According to the statistics of Coupland and Morris (1878), in three-fourths of the cases of stricture the seat is in either the rectum or the sigmoid flexure. The transverse and descending colon are more frequently affected than the ascending colon and the cæcum.

The discrimination of the cases of obstruction caused by compression or stricture from the cases involving strangulation can generally be made clinically by means of the differential points which have just been stated.

The fact of the obstruction being seated in the lower part of the large intestine can generally be ascertained. If seated in the rectum, it is accessible to direct exploration. If seated at the sigmoid flexure, the fact is shown by the evidence, afforded by manual exploration, of distension of the transverse and ascending colon and by the inability to inject liquid beyond the rectum. *Per contra*, the situation at the upper part of the large intestine or in the small intestine is shown by the absence of distension of the large intestine with intestinal contents, and by the ability to fill the colon with injected liquid. The obstruction may be complete, but in general a certain quantity of liquefied fecal matter passes the point of obstruction and is evacuated. The injection of liquids as a means of determining the seat of stricture is open to fallacies. Not only may the obstacle be due to other causes, such as a fecal collection, but liquids may pass beyond a stricture from below, although the occlusion be complete as regards the passage downward of intestinal contents. Simon's method of exploration by the passage of the hand into the bowel may be resorted to, giving due consideration to the care to be exercised in the operation and the possible danger of producing rupture of the intestine.

The distension from accumulation of the intestinal contents may lead to rupture of the intestine. The distension may gradually go on and lead to this accident without pain or other symptoms sufficient to direct attention to the existence of any serious obstruction. This fact was illustrated by a case under my observation at Bellevue Hospital, in which nothing more than ordinary constipation had been suspected, when peritonitis suddenly became developed, ending fatally in a short time. Obstruction was produced in this case by a cancerous tumor at the sigmoid flexure; the colon and cæcum were enormously distended with feces, and the peritonitis was due to rupture of the cæcum. This case illustrates the ability of the ileo-cæcal valve to resist an amount of pressure sufficient to lead to rupture of the cæcal walls.

In the treatment of obstruction from compression or stricture active purgatives are to be avoided, but saline laxatives in small doses, in order to liquefy the intestinal contents, are indicated. Colic pains are to be relieved by opiates. The diet should be nutritious and concentrated, in order to leave as little fecal residue as possible. If the obstruction be situated at the lower part of the large intestine, careful efforts to pass a flexible tube beyond the constricted portion may be employed, and if successful liquid may be injected above the obstruction.

In cases of obstruction from compression or stricture at the lower part of the large intestine, colotomy with a view to the establishment of an artificial anus is warrantable, provided other measures to free the intestine from an amount of accumulation likely to lead to rupture be not successful. Cases have been reported in which life has been prolonged and comfortable health obtained by means of surgical interference. Cæsar Hawkins reported the results of the operation for artificial anus in 44 cases. In 10 of these cases death took place within forty-eight hours, in 21 within five weeks, and in 13 the operation was successful. Of these 13 cases, 6 died in six months, and 7 survived more than a year. Professor Erskine Mason, in connection with 6 cases in which he had performed this operation, collected and tabulated 80 cases, exclusive of the 44 cases reported by Hawkins. The reader is referred to Mason's article for a full consideration of the claims of colotomy.[1]

Obstruction from Impaction of Feces, Gall-Stones, Enteroliths, and Foreign Bodies.

Obstipation may be produced by the causes named in the above heading, but in general they induce only more or less constipation, and in treating of the latter affection they will again be referred to. An accumulation of a mass or of masses of hardened feces sufficient to produce obstruction may take place in different parts of the large intestine. The rectum in aged persons not infrequently is the seat of such an accumulation. It is to be suspected when enemas cannot be given and perhaps the pipe of the syringe is found to penetrate a fecal mass. Examination shows the seat and the character of the obstruction. It may be necessary to break down and remove the accumulation by a scoop or the handle of a spoon. The accumulation may be at the sigmoid flexure, and then resistance to the passage of liquid injected and to the introduction of a flexible tube is found to be at that point. The accumulation may be at any point above the sigmoid flexure, but is oftenest in the cæcum or ascending colon.

Masses of feces above the sigmoid flexure may give rise to a tumor or to tumors appreciable by manual exploration through the abdominal walls. These fecal tumors are liable to be mistaken for others of a different character. A woman in the country presented an abdominal tumor of the character of which her attending physician was not satisfied, and an eminent professor of anatomy, now deceased, was requested to see the case. He was led to regard the tumor as malignant, and of course to form a very unfavorable prognosis. Shortly after the consultation a profuse diarrhœa set in, and, much to the astonishment of the patient and her physician, the tumor suddenly disappeared. It was a fecal tumor. I could cite other instances of a similar error. In view of the liability to error of diagnosis, and in order that appropriate measures of treatment may be pursued, the distinctive features of the tumors are to be borne in mind. One point in the diagnosis is their situation in the tract of the colon. Generally, they are not tender on pressure, but may be handled freely without giving pain; but to this rule there are some exceptions. By firm and continued pressure they may sometimes be moved backward or forward in the direction of the colon. By firm and continued pressure also the form of the tumor may be altered.

The absence of pain, tenderness, vomiting, and of the constitutional symptoms accompanying invagination, internal hernia, and twisting of the intestine suffices to exclude these affections, but it cannot be at once determined that the obstruction is not due to compression or stricture. The effect of measures of treatment will be likely to settle the diagnosis. The measures to be employed are mild purgatives in small doses, repeated after short intervals, conjoined with large enemas given through a stomach-tube carried upward to the sigmoid flexure. Active cathartics do not accomplish the object, and are liable to do harm. The object is to soften the harder collections, at the same time gently increasing the peristaltic movements. An emulsion of castor oil and a solution of the sulphate of magnesia or of soda are eligible purgative remedies. The bitter waters (Friedrichshall, Pulna, and Hunyadi Janos) may be found effective. The injections should consist of lukewarm water, to which syrup or mucilage should be added to obviate rapid absorption by the bowels. Some attach importance to the addition of ox-gall to the enemas. These measures, judiciously employed, will generally prove successful without much delay in relieving the obstipation and causing the fecal tumors to disappear. Mild purgatives may be required for some time after the obstruction is removed; and the treatment then will resolve itself into that adapted to habitual constipation.

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[1] Vide *Am. Journ. of Med. Sciences*, October, 1873. A considerable number of cases has been reported in different medical journals since the date of Professor Mason's article.

Obstruction may be due to the presence of large gall-stones or of intestinal calculi or enteroliths. Gall-stones of sufficient size to produce obstruction usually escape into the intestine after fistulous communications have been formed between the gall-bladder and some part of the intestinal canal, as the duodenum. Large gall-stones may, however, pass through the greatly-distended ducts into the duodenum. Obstruction produced by gall-stones is rare, and is almost invariably situated in the small intestine.[1] Intestinal concretions or enteroliths rarely exceed in man the size of a hazelnut. In the horse and other herbivorous animals they are more common, and may attain an enormous size, weighing sometimes twenty pounds. These intestinal calculi in animals constitute a variety of the so-called bezoars, to which were formerly attributed remarkable medicinal virtues. The nucleus of these concretions in man is often formed by a gall-stone, by a hard mass of feces, or some foreign body, as fruit-seeds, eggs of ascarides, indigestible aliment, etc. The concretions frequently present concentric layers. They are composed for the most part of phosphate of lime and of magnesia, mixed with carbonate of lime, biliary coloring matter, and intestinal mucus. Concretions occasionally form in the intestine in consequence of the prolonged use of certain mineral drugs. In this way have been formed concretions composed of calcium carbonate, of magnesium carbonate, of magnesia, and of iron. Indigestible constituents of the food, especially the fibres and husks of vegetables, may form a conglomerate mass of sufficient size to give rise to obstipation. All these varieties of concretions are most frequently formed in the cæcum and large intestine. They rarely attain sufficient size to produce any considerable obstruction. They may, however, prove serious obstacles in case a stricture of the intestine already exists. These obstructing bodies may be situated within the cæcum, at the sigmoid flexure, or in the rectum. They may sometimes be felt through the abdominal walls. They may lead to ulceration and perforation of the intestine, or, if not discharged and if the obstruction be complete, vomiting of so-called stercoraceous matter ensues, and death may take place from exhaustion. An intestinal concretion, cuboid in form, the vertical and transverse diameter one and a half inches, its weight fifteen drachms, was sent to me by Dr. O. T. Jameson, which was found after death in the ileum just above the cæcum. It was distinctly felt through the abdominal walls during life, and had existed for more than twenty years. The patient was a shoemaker, and he experienced notable inconvenience from the concretion only at times after overwork or after violent muscular exertion. He died of pneumonia at the age of forty-seven.

Finally, foreign bodies—that is, by way of distinction, bodies not contained in the aliment or derived from the body—may pass into the intestinal canal from the stomach or be introduced into the rectum. In the latter case their presence is readily ascertained by exploration, and they are removed by appropriate surgical measures. When introduced into the stomach they have been swallowed either deliberately, as is done by jugglers, or inadvertently. Prof.

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[1] Vide case reported by Dr. Draper, in *New York Med. Journal*, July, 1882, and case reported by Dr. Johnson, in *Medical News*, June 27, 1885.

Gross cites the case of a man who swallowed a bar of lead weighing a pound, which was removed from the stomach by an opening made through the abdominal walls, and the patient recovered. The same author refers to another case in which a large teaspoon was swallowed in a fit of delirium, and this was extracted from the ileum by the operation of enterotomy.

Foreign bodies, not sufficient in number or not large enough to occasion obstruction, are often swallowed, especially by children. The practitioner is frequently summoned in haste, and great anxiety is felt when bodies such as pins, coins, portions of glass, etc. are known to have been swallowed. They may give rise to more or less irritation of the mucous membrane, and they may even occasion ulceration and perforation of the stomach or intestines. In general, however, they pass through the alimentary canal without any appreciable inconvenience, and the physician is warranted in giving assurances of the absence of danger. It is not judicious in such cases to give cathartics or laxatives. The mucous membrane is best protected against injury if a sharp-pointed or irritating body be contained in the solid contents of the bowels. Moreover, the increased peristaltic movements caused by a purgative increase the risk of injury. The desire to have anxiety relieved as quickly as possible, by ascertaining that the foreign body has passed, is an inducement to resort to purgation, which is to be resisted.

Functional Obstruction.

Persisting obstipation, accompanied with vomiting of so-called stercoraceous matter, colic pains, etc., may occur as a purely functional disorder, constituting an affection which has been called *idiopathic ileus.* A person is seized with abdominal pain, increased paroxysmally; vomiting soon takes place, and after a time the matter vomited has the odor of feces; measures to procure evacuations from the bowels are ineffectual; the pulse becomes frequent, and there is more or less prostration. After the lapse of a week or more these symptoms subside, free evacuations from the bowels ensue, and the patient recovers. These are the prominent features of a case which I reported many years ago.[1]

Cases of functional obstruction may simulate those of obstruction from the structural affections which have been considered, and there is ground for the belief that in a certain proportion of the reported cases of the successful treatment of invagination[2] or of strangulated hernia within the abdomen the affection was purely functional. The regurgitation of the contents of the small intestine, which have a stercoraceous odor from the diffusion of the gases contained in the large intestine, in these as in other cases of obstruction, has been attributed to an inversion of the peristaltic movement. Experiments on animals do not favor this explanation. Brinton explains the regurgitation by supposing that in the centre of the intestinal tube a retrograde current is produced by the normal peristaltic movements if there be an obstruction. The obstruction, when functional, may be caused by spasmodic contraction of the tube, or it may be due to paralysis of a portion of the tube, and consequent failure of this portion to propel its contents.

The clinical discrimination of functional obstipation from obstruction caused by structural affections cannot always be made at once with confidence. The absence of tumor and of pain and tenderness localized at a particular point tends to the exclusion of the latter. The functional character of the affection may be surmised if the patient be a female subject to hysteria and suffering from the nervous symptoms associated with so-called spinal irritation. The probability of the affection being functional is increased in proportion as the obstipation, with vomiting, etc., continues, without the supervention of the symptoms which occur after sphacelation of intestine has taken place—namely, frequency and feebleness of the pulse, coldness of the surface, extreme prostration, and great tympanites; in short, the symptoms denoting impending death by asthenia.

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[1] *Buffalo Med. Journal*, 1851, vol. vii. p. 530.

[2] *Ibid.*, 1852, vol. vii. p. 383.

In the TREATMENT of functional obstipation opium and other measures to relieve pain and allay spasmodic action are indicated. Cathartics may be cautiously tried, but if ineffectual they should not be persisted in. Enemas administered by means of a long flexible tube are to be employed.

The importance of examining for the evidence of femoral and inguinal hernia is to be enjoined in connection with the subject of functional obstipation.

CHAPTER VII.

FUNCTIONAL DISEASES OF THE INTESTINE.

Diarrhœa.—Enterorrhagia.—Constipation.—Intestinal Colic.—Enteralgia.—Lead Colic.—Colic from Copper.—Passage of Gall-Stones.

Diarrhœa.

THE term diarrhœa is used to denote morbid frequency of intestinal dejections which are also liquid or morbidly soft, and often otherwise altered in character. Diarrhœa is a symptom of inflammation of the large and the small intestine and of certain structural lesions, such as those incident to tuberculosis of the intestine and to typhoid fever. It is a functional affection when not dependent on inflammation or on any appreciable lesion of structure. It is an element of other functional affections—namely, sporadic cholera, intestinal indigestion, and certain cases of colic. As a functional affection it may be either transient or chronic.

Writers have described several varieties of diarrhœa, based on diversities as regards the dejections. It is sufficient to enumerate the more important of these diversities without considering each as constituting a separate variety of the affection. The following enumeration answers all practical purposes:

A diarrhœa is said to be *fecal*, *feculent*, *stercoraceous*, or *simple* when the dejections consist of the feces not changed in character, but simply morbidly soft or liquid. This form of diarrhœa is represented by the operation of cathartics, which increase the peristaltic movements and give rise to a certain amount of transudation. The dejections in this form of diarrhœa have the normal fecal odor. They are called *bilious* when, from their yellow or green color, bile is supposed to be present in larger quantity than usual. The green color which has been considered as characteristic of the action of mercury upon the liver, according to Thudichum is due to the presence of subsulphide of mercury.[1] Robin states that the microscope shows the coloring matter to be biliverdin.[2] Diarrhœa is *serous* or *watery* when there is an abundant discharge of liquid; that is, when there is copious transudation or enterorrhœa. This is represented by the operation of hydragogue cathartics, such as elaterium, jalap, and the bitartrate of potassa. The dejections are called *lienteric* when they contain undigested aliment. This form of diarrhœa is met with especially in children. A diarrhœa is *mucous* or *dysenteric* when mucus is apparent in the dejections. An abnormal amount of mucus may be secreted from irritation alone without inflammation, but if the mucus be abundant it denotes an inflammatory condition. This form of diarrhœa is represented by the operation of drastic cathartics, such as croton oil. Finally, in a rare form, called *adipose* or *fatty diarrhœa*, free fat or oil in greater or less abundance is found in the evacuations. This last form claims distinct consideration.

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[1] *The Lancet*, Oct., 1860.

[2] *Sur les Humeurs.*

In cases of adipose or fatty diarrhœa the fat is generally liquid when passed, resembling melted butter or grease, having when cold the consistence of butter, beef tallow, or wax. In some cases it is passed in the form of globular concretions of a waxy consistency, varying in size, which melt with heat and burn like tallow. The fat is often passed by itself, and it may be passed involuntarily in a small quantity at a time. When passed with the feces it separates and collects on the surface. It has usually an extremely offensive odor. The quantity passed *per diem* in cases which have been reported varies from two or three ounces to half a pound or more.

Assuming all the fat passed from the bowels to have been ingested as fat, the rationale involves simply the non-digestion of this alimentary substance; but in some reported cases it is stated that the quantity of fat passed was not affected by the amount of fatty food. In a case reported by Wells the patient abstained for several weeks from fatty food, and as far as convenient from articles convertible into fat, without any diminution in the quantity of fat passed, nor was the latter affected by indulging freely in fatty food.[1] In two cases reported by Bright no change was produced by excluding as far as possible fatty articles from the diet. That the non-digestion of fat is in a great measure involved in the pathological explanation is, however, not to be doubted. Physiological researches have shown that the pancreatic secretion, the bile, and the intestinal juice effect the digestion of fat, and hence we are to look to the sources of these fluids in tracing the causation of fatty diarrhœa. It is an interesting fact that Bright, long before the discovery of the particular function of the pancreas by Bernard, was led by an analysis of three cases which came under his observation to consider fatty diarrhœa as a symptom of disease of the pancreas. In these three cases the only lesions found after death which were common to all were of the pancreas, duodenum, and liver. He excluded the lesions in the two latter situations because they occur in these organs so often without fatty diarrhœa; but he admits that disease of the pancreas is not always accompanied by this symptom.[2] The pancreas has been found diseased in a large majority of the reported fatal cases in which this organ was examined after death. Of 25 cases tabulated by Griscom,[3] in 10 the bodies were examined after death; of these 10 cases, in 8 the pancreas was diseased, and in the remaining 2 cases the absence of disease of the pancreas is not stated. Yet fatty diarrhœa occurs in only a small proportion of the cases of disease of the pancreas. Thus, it was observed in only 3 of 37 cases of cancer affecting this organ tabulated by DaCosta.[4] Hence it is to be inferred that there are generally involved morbid conditions which influence the digestion of fat, irrespective of the pancreatic secretion. Of the fatal cases among those tabulated by Griscom in which the morbid appearances were observed, the liver in several was either diseased or its ducts engorged; but in some of the cases this organ appeared to be healthy. The fact of complete recovery taking place in a certain proportion of cases goes to show that the non-digestion of fat may occur as a functional disorder.

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[1] *New York Medical Times*, February, 1854.

[2] "Cases and Observations connected with the Diseases of the Pancreas and Duodenum," by Richard Bright, M. D., *Medico-Chirurg. Transactions*, vol. xviii. Vide review in *British and Foreign Medico-Chirurgical Review*, July, 1853.

[3] *Transactions of the American Medical Association* for 1863.

[4] "On the Morbid Anatomy and Symptoms of Cancer of the Pancreas," extracted from the *Proceedings of the Pathological Society of Philadelphia*, 1858.

A larger number of cases than now available is required in order to establish the clinical history, as well as the causation and pathological relations, of this form of diarrhœa. Jaundice was noted in several (six) of Griscom's cases. Diabetes mellitus coexisted in two cases. Hemorrhage from the bowels, vomiting, and pains in the abdomen sometimes resembling those caused by the passage of gall-stones, are among the symptoms noted in these cases. The duration of the affection varies much in different cases. In one case it had existed at frequent intervals for six years, in another case for two years, and in one case it disappeared after a few weeks. It is evident that the prognosis is to be based on circumstances other than the discharge of fat —namely, enlargement of the pancreas and liver, persisting jaundice, progressive emaciation, etc.; in other words, the danger depends on the lesions or morbid conditions on which the discharge of fat may be dependent or with which it is associated. Of the 25 cases collected by Griscom, 14 died, 8 recovered, and in 3 the termination was not noted.

The TREATMENT must have reference more to the circumstances connected with the fatty diarrhœa than to the latter. With our present knowledge, palliative measures according to the symptoms in individual cases, together with measures to improve digestion and invigorate the general health, comprise the treatment. In one of Griscom's cases recovery took place under the use of olive oil in large quantity, and Watson states that a case was successfully treated by Elliotson in the same way. In a case observed by Griscom at the New York Hospital the discharge of fat invariably ceased whenever the patient took from six to eight ounces of whiskey *per diem*. In the case reported by Wells, the patient, having had a daily discharge of three gills of fat for two years or more, recovered in a few weeks after giving up the occupation of a carpenter and adopting the life of a farmer.

Exclusive of the form just considered, diarrhœa involves different pathological elements—namely, increased peristaltic movements, increased transudation, increased secretion from the mucous follicles, and perhaps increased flow of bile. If the diarrhœa be purely functional, of course there is no inflammation of the mucous membrane. Practically, it is not always easy to determine whether diarrhœa be purely functional or dependent on subacute inflammation; but with reference to the treatment to determine this point is not of great importance. It is not improbable that, of the cases considered as functional in a certain proportion there is a slight grade of inflammation, such as exists in coryza or a mild bronchitis. In making the diagnosis, acute inflammation is to be excluded by the absence of diagnostic symptoms—namely, pain, tenderness, febrile movement, and anorexia. Lesions of the mucous membrane are also to be excluded. In cases of chronic diarrhœa this is not always easy. Lesions are to be suspected if the diarrhœa follow an attack of dysentery or enteritis, if it persist in spite of judicious management, if blood be sometimes present in the dejections, and if tubercles exist in the lungs. In the latter case tuberculosis of the intestine is to be suspected; and on the other hand persistent or frequently recurring diarrhœa, conjoined with cough, be the latter never so slight, should excite suspicion of pulmonary phthisis.

Transient diarrhœa due to arrested or defective intestinal digestion is of frequent occurrence. With the diarrhœa which follows over-indulgence at the table or the ingestion of certain articles of food every one is practically familiar. This is the *diarrhœa crapulosa* of the old writers. It may proceed from excesses in quantity or from particular kinds of food, the indigestibility of the latter perhaps showing an idiosyncrasy of the person affected; thus, ices, salads, shellfish, etc. in some persons always, and occasionally in all, give rise to transient diarrhœa. The undigested aliment, passing into the large intestine, having undergone chemical changes which the processes of digestion in health prevent, produces irritation and acts like a cathartic. Frequently the diarrhœa is preceded and accompanied by griping pains, and it may be under these circumstances an element of another functional affection—namely, colic. These attacks of diarrhœa cease spontaneously, as a rule, after the expulsion of the undigested contents of the intestine, and they are rarely considered of sufficient importance to require medical advice.

An arrest of intestinal digestion may take place from causes other than errors of diet and give rise to diarrhœa. A strong mental emotion may have this effect. I was present at an operation for hernia when the surgeon, from the mental anxiety incident to his sense of responsibility, was obliged to relinquish the scalpel and precipitately retire to evacuate the bowels. A gentleman in business, receiving suddenly unexpected information which led him to know that he was a bankrupt, was immediately seized with diarrhœa. In like manner, diarrhœa may arise from an arrest of digestion from an exposure to cold or from over-exertion. In these cases the mechanism is the same as when the diarrhœa depends on dietetic errors, the undigested aliment in the large intestine acting like a cathartic.

Diarrhœa not transient, but continuing more or less, perhaps becoming chronic or recurring at short intervals, also proceeds in a large proportion of cases from indigestion. Habitual or frequent indigestion leads to diarrhœa in the same way as when it is transient—namely, the undigested aliment undergoes chemical changes and causes irritation of the large intestine. The dejections, as in transient diarrhœa, may be feculent, bilious, mucous, lienteric, or, more rarely, serous. The diarrhœa in these cases is an element or symptom of indigestion, but not infrequently, the disorder of digestion being confined to the small intestine, gastric ailments are not present. The indigestion thus giving rise to diarrhœa may be produced and kept up by various causes independent of excess or errors of diet.

It has been customary to attribute diarrhœa in certain cases to an excess or to a vitiated quality of bile. This causation is inferred when the dejections are notably bilious. This view is not improbable, although not based on positive knowledge. The bile may not be reabsorbed from the alimentary canal as in health, and hence it accumulates in excess without a morbid increase in the secretion; or it may be secreted in undue quantity or it may have a morbidly irritating property.

Diarrhœa is most liable to occur during the summer season. This may be explained, in part, by the larger proportion of fruit and vegetables in the ingesta during this season and by the effect of heat in weakening digestion. It may also in part be due to a more abundant secretion or to a lessened absorption of bile. It is a much more frequent affection in tropical than in cold or temperate climates. It occurs in travelling, especially in the summer season. Its occurrence is generally attributed to the drinking-water, but it is more rational to refer it to indigestion caused by dietetic errors, over-exertion, and disturbance of the regular habits of life. Children are far more subject to diarrhœa than adults, especially during dentition; and in children the dejections are often manifestly lienteric, showing its dependence on indigestion. It is the most prominent feature of the affection known as *cholera infantum*, or popularly, in this country, *summer complaint*. Among soldiers, especially in campaigns during the summer season or in warm climates, diarrhœa is extremely common, and is attributable to irregular habits as regards diet, conjoined with the exposure, excitement, and fatigue incident to active service. Diarrhœa occurs in persons exhausted from the want of food or from other deprivations, and in females enfeebled and anæmic from protracted lactation, due in these cases to intestinal indigestion. The same explanation probably applies to the diarrhœa which occurs at a late period in cases of pulmonary phthisis and other chronic affections when not dependent on intestinal lesions. Under these circumstances it has been called *colliquative diarrhœa.*

The accumulation of urinary principles in the blood as a consequence of renal disease occasions diarrhœa. The diarrhœa and vomiting which occur under these circumstances appear to be for the purpose of eliminating urea vicariously, and they are therefore conservative. They occur when the kidneys are removed in inferior animals, and, as shown by the experiments of Bernard and Barreswill, the liquid transudation into the alimentary canal contains either urea or the carbonate of ammonia. The dejections in uræmic diarrhœa are serous or watery.

Another pathological condition occasionally giving rise to diarrhœa is congestion of the portal vessels when the circulation in this portion of the vascular system is obstructed by hepatic disease. It thus is a symptom in some cases of cirrhosis of the liver, and it sometimes appears to limit the dropsical effusion into the peritoneal sac which belongs to the history of that disease. The diarrhœa under these circumstances is serous, transudation taking place from the pressure of the blood within the congested veins.

Crapulous diarrhœa, or transient diarrhœa due to indigestion, in general calls for little or no treatment. If the contents of the large intestine be not spontaneously expelled, an evacuant remedy may be given. A saline purgative is best adapted to this end. The propriety of this measure is to be determined by ascertaining the quantity and character of the dejections which have occurred. If these have been abundant and feculent, a cathartic is not required. If, after the bowels have been freely evacuated, diarrhœa, pain, or uneasiness continue, the irritation may be quieted by a mild anodyne; for example, a drachm or two of the camphorated tincture of opium for an adult in chalk mixture; a grain of opium; a sixth or a quarter of a grain of a salt of morphia in mint-water; or five grains of Dover's powder combined with two or three grains of aromatic powder. The remedy chosen is to be repeated after six or eight hours, if required. The diet for a day or two should be simple and somewhat restricted. Subsequently a laxative may be required if the bowels do not act spontaneously. The treatment of cases of diarrhœa tending to continuance or frequently recurring and dependent on indigestion embraces, in the first place, remedies to relieve the irritation of the large intestine. Opium in some form may be used for this purpose, but only temporarily, because its continued use tends still more to impair digestion. A mild purgative may often be prescribed with advantage, for frequent and loose dejections are not incompatible with retention of hardened feces—in fact, with constipation. The subcarbonate or the subnitrate of bismuth is an excellent remedy to relieve intestinal irritation. It is often effective, and is not open to the objections of opium. It should be given in doses of from a scruple to half a drachm to adult patients. If not effective alone, a small quantity of a salt of morphia may be added to it. Of a variety of remedies tried in cases of chronic diarrhœa among soldiers returning from the campaigns in Virginia in the summers of 1862 and 1863, bismuth, in my hands, proved the most effective, and this, I believe, was the result of the experience of others. Opiates are sometimes most effective and least objectionable when given *per enema.* Pepsin or the rennet wine is sometimes useful temporarily as a means of artificial digestion, given as already recommended in cases of dyspepsia.

This part of the treatment, however, is merely palliative. The curative treatment embraces regulation of diet and measures to render digestion complete. The diet in quantity and quality should be adapted to the digestive powers. Chickens, eggs, and tender meats plainly cooked are most likely to be digested, but in some cases a milk and farinaceous diet is found to be preferable. Experience in individual cases is to be the guide. Crude vegetables are to be interdicted, but ripe fruits in moderation may frequently be taken without inconvenience and with advantage. A moderate quantity of food taken at short intervals is generally advisable, rather than a full meal once or twice daily. A sea-voyage, a visit, removal from the city to the country, or the change of a warm, variable, humid, relaxing climate for an atmosphere cool, uniform, dry, and bracing, will be likely to prove highly beneficial, if not curative, in cases which resist dietetic and medicinal treatment. The efficacy of these measures is strikingly illustrated in cases of the so-called summer complaint in children.

The remedies, other than those merely palliative, which are useful in the cases of diarrhœa under consideration belong among the tonics and the astringents; and the latter may be both palliative and curative. Of the various vegetable astringents, krameria, hæmatoxylon, kino, catechu, and rubus villosus are eligible articles. The tannic acid, if well borne by the stomach, is sometimes efficient. As purely tonic remedies, quinia or the non-officinal preparations of calisaya-bark so much in vogue, and other bitter infusions, are often useful. Salicin, in doses of from ten to twenty grains three times daily, is an excellent tonic remedy. Preparations of iron are especially useful when the patient is anæmic, as in cases of diarrhœa occurring in females during lactation. The tincture of the chloride of iron is one of the best of the chalybeates. The persulphate or pernitrate of iron is sometimes efficacious as an astringent, as well as a tonic remedy. Of mineral tonics and astringents, the acetate of lead, nitrate of silver, and the sulphate of copper have been found useful. The subcarbonate or the subnitrate of bismuth is of great value in chronic diarrhœa, not only as a palliative, but as a curative remedy, given in large doses—namely, from a scruple to a drachm three or four times daily, sometimes in combination with small doses of a salt of morphia, as recommended above in connection with palliative measures of treatment. The mineral acids are efficacious in some cases. The preparation known as Hope's mixture has been much in use. This mixture is composed of four drops of nitric acid and from forty to sixty drops of laudanum in four ounces of camphor-water, a tablespoonful to be taken every two, three, or four hours. In rebellious cases of chronic diarrhœa the various remedies which have been named are to be given in succession and in different combinations. The curative and palliative remedies are to be combined to meet the indications in individual cases.

Diarrhœa dependent on uræmia and on cirrhosis of the liver does not admit of curative treatment, and, being conservative, is only to be kept within certain limits by palliative remedies.

Enterorrhagia.

Hemorrhage into the intestinal canal is properly called enterorrhagia. The term *melæna* has been used to denote dark-colored or black dejections consisting of blood which has passed from the stomach into the intestinal canal. Partial digestion of the blood or the action upon it of the gastric and intestinal secretions renders it tarry or pitch-like in appearance and generally very offensive to the smell. Dejections of this character often follow gastrorrhagia. The term melæna has been also applied to cases of gastrorrhagia in which the vomited blood presents a similar appearance. Moreover, the term has been loosely applied to denote dark-colored or black evacuations not consisting of blood. The term is one of those which it is desirable should become obsolete. Cases in which blood evacuated from the bowels comes from the stomach or from any of the abdominal viscera other than the intestinal canal are not cases of enterorrhagia; and cases of hemorrhoidal or other hemorrhages occurring near the anus are not properly embraced under this head. The source of the hemorrhage must be the mucous membrane between the stomach and rectum. The tarry or pitch-like appearance of blood in the stools is evidence of its source in the stomach or upper part of the intestine; but this appearance is not positive proof that the matter evacuated is blood, as a dark color may be imparted to the stools by the administration of iron or of bismuth. It probably is blood if the stools have been preceded by the vomiting of blood; but in some cases of gastrorrhagia vomiting does not take place, all the blood passing into the intestinal canal. In doubtful cases dilution in water of some of the matter evacuated renders the appearance of blood more evident, or recourse may be had to the microscope or to the spectroscope. When the source of hemorrhage is the rectum, the blood is fresh in appearance, and is passed alone, in greater or less abundance, either after a fecal evacuation or with straining efforts to procure a stool. Examination with the eye or touch will generally show the existence of hemorrhoids, but in some cases it reveals other local affections, such as ulcers, carcinoma, a polypus, or intussusception.

In cases of enterorrhagia the hemorrhage takes place in different situations along the tract of the small and large intestine. The blood is less changed in appearance the nearer its source is to the rectum and the more quickly it is evacuated. If the hemorrhage be at the upper part of the intestinal tube and if the blood pass slowly along the tube, it becomes dark or blackish, its appearance resembling that of the melænic stools in cases of gastrorrhagia, and there may be a similar difficulty in determining whether the appearance be due to the presence of blood. The quantity of blood passed varies in different cases of enterorrhagia. It is frequently large, amounting in some cases to several pints and even quarts. Evacuations may consist wholly of blood or the blood may be mixed with fecal matter. The blood may be entirely liquid or the evacuations may contain clots in greater or less abundance.

Intestinal like gastric hemorrhage has rarely any claim to be regarded as an individual affection. Like gastric hemorrhage, it is incident to a variety of morbid conditions. It is an important occasional event in typhoid fever, and will be referred to in treating of that disease. It enters into the characteristic dejections of dysentery. It is an effect also of thrombosis of the portal vein and of the mesenteric vein.[1] It is one of the symptoms of embolism of the superior mesenteric artery. It occurs in cases of carcinoma of the bowels. It is a rare occurrence in tuberculous ulceration of the small or large intestine. It belongs to the natural history of scorbutus, purpura hæmorrhagica, and yellow fever. Like gastric hemorrhage, it may be vicarious of menstruation. It is not an uncommon effect of the portal congestion occasioned by cirrhosis of the liver. It occurs, however, when not connected with these or any other discoverable morbid conditions. I have met with several examples of enterorrhagia not connected with any of the several causative conditions which exist in the larger proportion of cases. While, therefore, the chances are that some of these conditions exist, the occurrence of hemorrhage is not proof of the existence of any of them. Symptoms attributable to the loss of blood, and marked in proportion to the amount of hemorrhage, are debility, pallor, feebleness of the pulse, perspiration, sense of faintness, etc. The loss of blood may be sufficiently great and rapid to occasion sudden death, but this is rare. The repetitions of the hemorrhage may lead to a degree of exhaustion and anæmia from which recovery is difficult, and a fatal result may take place by slow asthenia. In general, however, if the hemorrhage be not connected with morbid conditions which involve danger, the prognosis is favorable. Hemorrhage sufficient to occasion fatal syncope may take place and no blood be discharged by stool. In such a case the diagnosis of enterorrhagia is impracticable.

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[1] Sir William Gull has reported a case of probable thrombosis of the superior mesenteric vein and the renal veins, recovery taking place. (Vide *Guy's Hospital Reports*, vol. xlii.) Profuse intestinal hemorrhage was associated with severe pain, albuminuria, and a condition of nearly complete collapse. He cites a fatal case reported by Dr. Hilton Fagge.

The TREATMENT of the different affections or morbid conditions with which intestinal hemorrhage is usually connected need not be here repeated. It will suffice to refer to measures having reference to the hemorrhage. These are essentially the same as in cases of gastric hemorrhage. Rest in the recumbent position and quietude of mind are important. The peristaltic movements are to be quieted by opium. Cold applications to the abdomen should be made if the arrest of the hemorrhage be urgently indicated. The most effective mode of applying cold is by means of the ice-bag. In cases in which this indication is less urgent, dry cupping, sinapisms, and warm stimulating pediluvia will suffice without the application of cold. Food and drinks should be taken cold. The diet should be restricted and bland. Purgatives are contraindicated. The mineral acids are generally thought to be useful in cases of intestinal as well as in gastric hemorrhage. The sulphuric-acid lemonade has the advantage of being a grateful beverage. The various hæmostatic remedies given in cases of gastrorrhagia—namely, tannic acid, gallic acid, acetate of lead, ergot, the astringent preparations of iron, etc.—are to be resorted to in proportion to the importance of arresting the hemorrhage.

Constipation.

The terms *constipation* and *costiveness* are commonly used as synonyms, denoting insufficiency of evacuations from the bowels. The latter of the two terms is sometimes used to denote a less degree of insufficiency than the former, the number of dejections being normal, but the quantity deficient and the act of defecation labored. The term *obstipation* has been already defined to denote a greater amount of difficulty than constipation; that is, obstruction of the bowels, either as a functional disorder or dependent on the various lesions which have been considered in a preceding chapter. Constipation exists as a functional disorder and it is incident to various affections. As a functional disorder it is extremely frequent, and, although not a serious affection, it claims attention on account of the inconvenience which it occasions, its importance as leading to other ailments, and the difficulty often of its removal.

The affection is seated in the large intestine. The anatomical arrangements of this portion of the alimentary canal show it to be intended to serve as a temporary dépôt for fecal matter, thus providing against the need of frequent acts of defecation. The contents of the alimentary canal are propelled more slowly in the large than in the small intestine—*first*, because the circumference of the former is larger in proportion to its muscular power; *second*, in the ascending part of the colon and of the sigmoid flexure the contents are propelled in opposition to gravitation; and *third*, the liquid portion of the contents is absorbed in their passage through the small intestine. Experience shows that one free evacuation from the bowels daily is the rule in health; but this rule is not without exceptions. Some persons have, habitually, two or three evacuations daily, and on the other hand some have an evacuation regularly every second or third day without any of the inconveniences of constipation; in fact, persons of the latter habit usually experience discomfort if temporarily evacuations take place daily. In determining, then, the existence of this affection, the habit in health is of course to be taken into account. In some cases the act of defecation is delayed for one, two, or three days after the period when it should have taken place, but the evacuation when it occurs is ample. In other cases the act occurs daily, but it is incomplete, and in these cases the act is usually labored and painful, the feces being dry, compact, and hard. The affection may be occasional and transient or it may be habitual.

Constipation gives rise to various local morbid effects, such as a feeling of pressure or weight in the perineum, a sense of abdominal distension or uneasiness, flatulency, diarrhœa, and colic pains. Hemorrhoids are often attributable to this affection. It gives rise also to pain in the head, dulness of the mind, flushing of the face, palpitation, and general malaise. If the act of defecation require violent straining efforts, these sometimes occasion hemorrhage into the brain if the cerebral arteries be diseased, and hernial protrusions. The contractility of the muscular tunic of the large intestine may be permanently impaired by long-continued distension, so that the affection is necessarily permanent.

Occasional constipation is not generally considered of sufficient importance to require medical advice, and persons resort of their own accord to a purgative or an enema. It is when the affection has become habitual or chronic that cases come under the cognizance of the physician; and, in order to understand the pathological character of the affection in these cases and its causation, certain points pertaining to the function of defecation in health are to be borne in mind. In its normal condition the rectum is empty, as shown by the researches of O'Beirne.[1] This portion of the large intestine is endowed with a peculiar sensibility which in health gives notice of the presence of feces and occasions the desire to defecate. The ability to perform the act involves a certain contractile power in the large intestine, and also in the abdominal and other muscles which co-operate in the performance of the act. In habitual constipation the contractile power of the intestine is impaired by distension. The muscular tunic is more or less paralyzed from the accumulation of the intestinal contents, in the same way as the bladder becomes paralyzed from over-distension. The accumulation giving rise to the distension may be owing to the large quantity of excrement, but it is generally a result of habitual neglect of the calls of nature. The desire to defecate is resisted, or, the mind being preoccupied, is unheeded, and the act is postponed, until at length the sensibility no longer gives notice of fecal accumulation; hence the accumulation goes on, the rectum and other portions of the large intestine become distended, and paresis follows. This is the manner in which constipation as a chronic affection is produced in a large majority of cases. The hurried performance of the act of defecation, the evacuation being in consequence incomplete, has measurably the same result as the neglect of the calls of nature. Instead of receiving adequate attention as important to health, defecation is considered an annoyance to be escaped from as quickly as possible. The uncomfortable provisions for defecation, especially in the country, contribute to constipation by rendering it disagreeable to devote to the act more time than seems absolutely necessary.

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[1] *New Views of Defecation, etc.*, by James O'Beirne, Am. ed., 1834.

Various circumstances may contribute to this affection. The abdominal muscles play an important part in the act of defecation. These muscles become weakened by obesity, and in females as a result of pregnancy. The muscular tunic of the intestine and the auxiliary voluntary muscles concerned in defecation lose more or less of their contractile power, in common with the whole muscular system, in anæmia and in other enfeebling conditions. Deficiency of bile and of the intestinal secretions may enter into the causation in some cases, but probably the importance of this source of constipation has been over-estimated. Constipation is not always present when no bile enters the alimentary canal in certain cases of jaundice. Inadequate alimentation, and especially the ingestion of purely nutritious food, leaving but little excrementitious residue, contribute to constipation. Sedentary habits are supposed to favor the affection, but it is probable that other causes are generally more operative, and especially inattention to the calls of nature. On the other hand, active exercise induces constipation by rendering assimilation more active, the liquid contents of the small intestine being more fully absorbed. Loss of fluids by abundant perspiration or by diuresis is another cause. The change of habits, with, generally, an increased assimilation, incident to a sea-voyage, as is well known, induces in most cases constipation.

Occasional constipation, if slight, is relieved by a laxative pill, repeated if necessary, or by a small quantity of Epsom or Rochelle salts dissolved in a tumbler of simple or carbonated water and taken upon an empty stomach. The Congress or the Kissingen water and the stronger bitter waters—namely, the Friedrichshall, Pullna, or Hunyadi-Janos—in small doses may be taken in lieu of the salts just named. A preferable method, which will generally suffice, is to excite the action of the large intestine by a simple enema of cold water. By means of Davidson's syringe the patient can resort to this measure without any difficulty. If the constipation be more than slight, a few grains of blue mass or a purgative pill may be taken at bedtime, followed, if necessary, by a saline draught or an enema in the morning. Persons often have recourse to purgatives without consulting physicians, under an impression that such remedies are always harmless and generally useful aside from the relief of constipation. Hence it is common to take active purgatives for slight constipation, and often when nothing is required. If the constipation give rise to little or no inconvenience, it is better to wait for spontaneous relief, and a little delay will often show that medicinal interference would have been unnecessary.

The management of habitual constipation often requires much care and perseverance on the part of the physician and patient. The object is to secure regularity and sufficiency in the evacuations. The means which may be employed are various. They may be arranged into dietetical, medicinal, and mechanical. The dietetical means consist in using freely articles which leave, after digestion, a bulky residuum—namely, cabbage, lettuce, the various vegetables known in the country as greens; articles having a laxative property, such as molasses, prunes, figs, etc.; or articles with indigestible constituents which stimulate the alimentary canal—namely, bran bread, corn meal, groats, or cracked wheat. A diet consisting in part of the foregoing articles will sometimes succeed in relieving habitual constipation; but with regard to the choice of this class of means the following practical rule is to be adopted: They are not to be preferred if they disorder digestion or occasion more disturbance than medicines. More harm sometimes results from overloading the digestive organs with articles of diet difficult of digestion or subjecting the canal to the irritation of unbolted flour than the continuance of constipation would occasion. A tumbler of simple water or of carbonated water taken in the morning before breakfast is not infrequently efficacious; if not, a teaspoonful of table-salt added will often render it sufficient.

The medicinal means are laxative remedies. With regard to these, a general rule is that the remedy chosen should be mild and the quantity as small as will suffice for the object. Purgation is to be avoided. Some patients fall into the custom of allowing the constipation to continue for several days, and then resorting to a free purgative. The constipation is of course relieved for the time, but the constipated habit becomes more and more fixed by this course. Another general rule may be stated: If more than one *small* dose of laxative be required, the remedy is to be given in small doses repeated twice or thrice daily, rather than in a single, larger dose. In this way the object is effected with a less amount of medicinal impression, and there is less risk of purgation.

As regards the particular remedy, aloes is specially suited to the desired object. It may generally with advantage be combined in a pill with a tonic, either the sulphate of quinia or a preparation of iron, and a small quantity of hyoscyamus or belladonna may be added. A very small proportion of aloes, a small fraction of a grain in each pill, will often suffice. The old preparation known as the elixir proprietatis (tincture of aloes and myrrh) is well suited to the object, a drachm given at bedtime, and repeated, if necessary, once or twice during the day. This preparation loses its disagreeable bitterness with age, and it is not generally repulsive when given with a little water and an abundance of sugar. The extract of nux vomica combined with belladonna or hyoscyamus sometimes answers well. A small piece of rhubarb taken repeatedly during the day sometimes effects the object satisfactorily, and this remedy to many persons is not disagreeable. I have known a few drops of the tincture of colchicum, repeated shortly after each meal, to answer admirably. The syrup of buckthorn, the extract of butternut, and the compound licorice powder are good laxative remedies. There are various modes of rendering the daily use of a laxative remedy not unpleasant to the taste. Prunes stewed in an infusion of senna answer well, and are not unpalatable. The confection of senna and medicated figs are also suited to persons who take a pill or potion with repugnance. Trousseau claimed for his favorite medicine, belladonna, that it acts as an efficient laxative in cases of habitual constipation. It has failed to secure this action in my experience, and I have frequently made trial of it. Here, as in other instances, experience in individual cases is to be the guide. In the majority of cases in which medicinal means are relied upon, some form of the so-called dinner-pills, or, as they have been appropriately termed, "peristaltic persuaders," will be found most convenient and satisfactory; and for the fastidious they may be silvered or sugar-coated. The white mustard-seed was formerly a popular remedy much in vogue, but this is objectionable on account of a liability to accumulate and become impacted within the intestine. Laxative remedies are not to be continued for a longer period than is required to secure an habitual action of the bowels. A minute dose of strychnia or nux vomica added to a laxative renders the operation of the latter more successful.

The mechanical means consist in the use of enemas and suppositories. The regular use of an enema of cold water at the same hour daily is a simple and sometimes an effective measure. This measure, however, is better suited to occasional than to habitual constipation. Habitually resorted to, it is likely to fail after a time; and the rectum becoming accustomed, as it were, to the stimulus of distension, it ceases to excite the peristaltic movements. In some cases a suppository of soap answers the purpose of provoking a regular and sufficient evacuation. The cocoanut butter may be used for this purpose.

Common molasses candy answers equally well, an oval mass of the size of a pigeon's egg being introduced within the rectum. This, however, is only a temporary expedient, ceasing usually to be effective after a time.

The most important part of the management in cases of habitual constipation is not embraced in the foregoing measures. It is the adoption of a rule to solicit an evacuation at the same hour daily. The importance of this rule is to be enjoined, but of course its success will depend on the perseverance of the patient. The time of the day most convenient for the act of defecation is to be selected, and in general the most favorable time is in the morning after breakfast. At the time fixed upon the patient should devote a reasonable period to the function of defecation, but without persisting in violent fruitless efforts. This should be considered in the light of a duty, not to be omitted a single day except from necessity. It may be long before the desired object is accomplished, but sooner or later, with the aid of some of the means which have been indicated, the desire will be felt at the appointed hour, and the ability to defecate at that time will be acquired in the great majority of cases. It is impossible to secure regularity and sufficiency of the evacuations without perseverance in this part of the management. The management also includes a rule not less important—namely, never to neglect or postpone the call to defecate when it occurs either after or before the time for the habitual performance of the act of defecation. The importance of waiting for a certain period after the contents of the rectum have been expelled is to be enjoined. Often the expulsion of the contents of the rectum is immediately followed by the passage of fecal matter from the colon into the rectum, which, remaining, blunts the sensibility of the bowel and renders it accustomed to distension. The objects of the management are to be understood and kept in view—namely, 1st, To preserve or restore the normal animal sensibility of the rectum by which the presence of fecal matter is felt; and 2d, to prevent paresis from distension, and to restore the muscular power which has been lost from this cause.

If this plan were early inculcated and carried out in health, habitual constipation would be as rare as it is now frequent. The prevention is not less sure than simple. This fact should be generally understood, especially in the training of girls, most of whom become affected with habitual constipation and suffer from it all their lives. Unfortunately, the existence of the function of defecation is too often ignored from notions of false delicacy, and the affection becomes established because parents and teachers are either themselves ignorant of this simple method of prevention or consider the subject as belonging exclusively to the physician.

Cases in which the intervals between fecal evacuations are many weeks and even many months, the accumulation of intestinal contents being tolerated with more or less inconvenience during these long periods, are among the curiosities of medical experience. Of the cases in medical literature, the one in which the longest interval occurred, with a single exception, was communicated for the *American Journal of Medical Sciences* in 1874, and the autopsy, in 1876, by Dr. Thomas D. Strong of Westfield, N. Y. In this case eight months and sixteen days elapsed without an evacuation from the bowels.[1]

Intestinal Colic.

The term colic, in its etymology, relates to the colon, but it is often applied to paroxysmal, spasmodic pain in other parts. Thus, the phrase hepatic colic is used to denote the pain caused by the passage of gall-stones, and renal colic the pain caused by the passage of renal calculi along the ureter. A painful affection of the uterus is sometimes called uterine colic. In the present nomenclature of diseases there is no suffix or prefix which, joined to the name of a part, expresses a painful spasmodic affection of that part. The affection of the alimentary canal known as colic may not be seated exclusively in the colon. It is probable that the small as well as the large intestine is affected in certain cases of intestinal colic. As applied to the intestine, colic denotes pain occurring in paroxysms or in marked exacerbations, the pain being of a character supposed to indicate spasm; that is, twisting, constricting, or griping. Colic pains are symptomatic of different inflammatory affections—namely, dysentery, enteritis, peritonitis—and of the various lesions which give rise to intestinal obstruction. They also enter into the functional affection called sporadic cholera or cholera morbus. As constituting an individual malady, colic of course is independent of inflammation or structural lesions and is disconnected from any other functional affection.

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[1] For synopsis of this case, vide *Clinical Medicine*, by the author, 1879, p. 354.

An attack of colic is characterized by paroxysms or exacerbations of pain varying much in its severity in different cases. The pain is usually seated in the region of the umbilicus. It is frequently extremely severe; and the patient bends the body forward with the thighs flexed, writhes, and tries a variety of positions, groaning or crying aloud from the intensity of suffering. This severe pain continues for a few moments, and then it either ceases completely or abates, to return again with the same severity after an interval usually of brief duration. If left to continue without measures for relief, an attack may embrace a few paroxysms only or it may last for several hours. An attack lasting for a short time and ending in one or more free evacuations from the bowels is so common as to be familiar to every one.

The abdomen in attacks of colic is frequently retracted, but in some cases it is more or less tympanitic. Generally, there is absence of tenderness, and firm pressure with the hands or lying upon the belly affords relief; but in some cases there is more or less soreness and tenderness on pressure, especially over the site of the cæcum. During the paroxysms of pain the abdominal muscles are sometimes spasmodically affected, especially the rectus muscle, the sections of which are felt as hard bunches. Usually there is constipation. Nausea is sometimes present, and occasionally vomiting. The pulse is but little or not at all accelerated, and absence of fever is shown by the thermometer. The skin is cool, and during the severity of pain frequently bathed in perspiration.

In a case presenting the characters just described it is important to determine, as soon as practicable, the existence simply of a functional affection; that is, to differentiate colic from affections in which pains like those of colic occur. The diagnosis is to be made by excluding these affections. Dysentery and enteritis are to be excluded by the absence of diarrhœa and of symptoms denoting inflammation. The pain in these two affections is rarely as violent as in most cases of colic; but in cases of acute peritonitis the exacerbations of pain may equal in violence those of a severe attack of colic, and the error of confounding these two affections has been committed in not a few instances. The exclusion of peritonitis is to be based on the absence of notable tenderness over the abdomen, of increase of temperature, of frequency of the pulse, and of other general symptoms denoting a much graver affection than colic. Invagination, internal hernia, and other obstructive lesions with strangulation cannot always be at once excluded. As a rule, the paroxysmal pain occurring in connection with these lesions at the outset, and, indeed, during a considerable time, is less violent than in colic, and after a time the existence of something more than colic is shown by persisting pain, tenderness, vomiting of so-called stercoraceous matter, sometimes tenesmus, frequency of the pulse, prostration, etc. The absence of these symptoms warrants the exclusion of obstructive lesions. The possibility that some obstructive lesion may exist is not to be lost sight of in cases which at first have the appearance of simple colic. The possibility of inguinal or femoral hernia is also to be borne in mind, and a careful examination with reference to this point is not to be omitted. The resemblance of labor-pains to those of colic is to be recollected. Pregnancy is sometimes concealed, and it has happened that the physician, supposing his patient to be suffering from colic, has been taken by surprise by the cry of a newly-born infant under the bedclothes. The passage of a urinary calculus from the kidney to the bladder may give rise to pains (renal colic) resembling that of enteralgia. This affection is to be excluded by the absence of its diagnostic features.

The several varieties of colic which writers have generally recognized do not require separate consideration. Some of these varieties rest on an insufficient basis of classification. The colic has been called crapulous when it proceeds from indigestion. Every one is familiar with the attacks which sometimes follow over-indulgence at the table, ending frequently in crapulous diarrhœa. A colic is called flatulent when it is accompanied with tympanites or by the expulsion of gas with relief. This variety occurs especially in infancy. The term bilious colic has been used when the affection is supposed to depend on hepatic derangement. This dependence is merely conjectural, and there is no more reason for continuing to append the word bilious to this affection than to various others in which the coexistence of disorder of the liver is equally hypothetical. Colic has been called stercoraceous when attributable to the retention of feces, but the latter is perhaps oftener an effect than a cause of colic. In another variety, called verminous, the colic is supposed to depend on the presence of worms; but it may fairly be doubted whether colic ever proceeds from this source. The variety called saturnine or lead colic is properly a distinct affection, and will be considered as such.

The morbid condition in colic is supposed to be spasm. Its seat is therefore the muscular tunic of the intestine. That this is the pathological character of the affection is rendered probable by the kind of pain, the constipation, together with the local symptoms, and the therapeutical measures which are found to be successful. It is not improbable, as supposed by Abercrombie, that in certain cases the spasm may be preceded by paralysis of a section of the intestinal tube, and consequent obstruction to the passage of its contents. Like other spasmodic affections, it may be produced by causes which act either directly upon the part affected or indirectly through its nervous communications.

Attacks of colic are frequently, if not generally, attributable to the local action of the ingesta. They may arise from indigestion caused by either excess in quantity or the indigestible quality of food. They not infrequently follow exposure to cold or fatiguing exertions, but these causes probably act by occasioning indigestion. Certain articles of diet—for example, shellfish in some persons—give rise to attacks of colic in consequence of an idiosyncrasy which is inexplicable. The muscular tunic of the intestine is especially susceptible to spasm in infancy, and colic occurs much oftener in early than in middle and advanced life. A susceptibility to intestinal spasm belongs to some persons, constituting a predisposition to this affection. In the practice of every physician there are those who are subject to attacks of colic more or less frequently. This predisposition may continue for a certain number of years, and then disappear. Persons with this predisposition often have an uncomfortable sensation of weight, coldness, or numbness in the abdomen, with general languor and irritability, preceding the development of colic, and by means of these premonitions they are able to predict an impending attack.

Although attended with great and, if not efficiently treated, often protracted suffering, colic is not a dangerous affection. It may be doubted if alone it ever prove fatal. It is one of those violent functional affections from which recovery takes place rapidly. It has no tendency to eventuate in inflammation or in any other disease, but in proportion to its duration it is followed by fatigue or weakness, and more or less abdominal soreness may remain for some time.

The object of TREATMENT is the relief of spasm as indicated by the cessation of pain. Measures are to be directed to this object without reference to the cause of the attack or the existence of constipation. If, as is generally the case, an attack have been induced by the presence of irritating ingesta, measures directed to the spasm should take precedence of evacuants; and if, as is frequently the case, the bowels were constipated at the time of the attack, the removal of this condition is a secondary object. So long as pain continues there is a resistance to the action of cathartics, and to defer measures for the relief of the pain until the bowels have been evacuated is to needlessly prolong the sufferings of the patient.

In mild attacks warmth to the abdomen and extremities, a little spirit not largely diluted with hot water, some aromatic stimulant, such as anise, ginger, cloves, mint, etc., or a few drops of chloroform, will suffice to procure relief. Twenty drops of chloroform, repeated, if necessary, at short intervals, will sometimes afford prompt relief in severe cases; if not, opium in some form is to be given to the extent of procuring freedom from pain. Administration *per enema* is to be preferred. A drachm of laudanum, with the same quantity of the tincture of asafœtida in a small quantity of mucilage or starch, may be injected, with injunctions to the patient to resist the desire to expel it; and usually in a short time this desire passes off. If complete relief do not follow in the space of an hour, the enema may be repeated. If the suffering be extremely intense, double this quantity of laudanum in the case of an adult may be given in the first or second enema, or a solution of a salt of morphia may be injected hypodermically. If necessary, the enema or the hypodermic injection may be repeated a second or third time. A similar plan is to be pursued as regards the doses and repetitions if the opiate be given by the mouth. If hypodermic injections be given, caution is to be exercised in their repetition. Fatal narcotism has been induced by want of caution in this regard. Hot fomentations or dry heat should be applied to the abdomen. Speedy success may be counted upon by pursuing this plan of treatment.

The subsequent treatment is to be determined by circumstances relating to the abdomen. If no inconvenience be felt, measures to effect a movement of the bowels need not be employed for twenty-four or even forty-eight hours. Not infrequently, if the physician be not in haste to give a cathartic, a sufficient evacuation occurs spontaneously. If measures to effect a movement be required, a large simple enema may suffice; if not, a mild saline purgative may be given in divided doses, or castor oil if this remedy be not offensive to the patient. The diet for a day or two should be simple and restricted.

The certainty with which colic yields to an efficient opiate treatment renders it one of the affections which exemplify very palpably the resources of medicine. Some patients, however, suffer considerably from the after-effects of the opium given, and this in a measure compromises the mutual satisfaction of the physician and patient in the success of the treatment. Persons who are unfortunately affected unpleasantly by most forms of opium are sometimes able to take certain preparations with less inconvenience than others. The experience of the patient on this point should be consulted.

Enteralgia.

The term enteralgia denotes a neuralgic affection referable to the intestine. It has to the latter the same relation as gastralgia to the stomach. Gastralgia and enteralgia may be associated (gastro-enteralgia). Enteralgia and colic are not infrequently confounded; and it is not always easy to distinguish, clinically, the one from the other. Enteralgia is characterized by pain within the abdomen, with intermissions or remissions, the pain having more or less intensity, but lacking the twisting or griping character of the pain in colic. It may be so severe as to lead to contortions and oral expressions of suffering. In females enteralgia is an element of attacks of tympanites due to the formation of gas, and is often associated with hysterical phenomena.

The DIAGNOSIS involves the exclusion of the various affections from which colic is to be discriminated, and the exclusion of the latter affection. (Vide p. 520.) The so-called rheumatism of the abdominal walls (myalgia) is also to be excluded. Lumbo-abdominal neuralgia is to be discriminated by the existence of tenderness in front at the umbilical or hypogastric region, and also at the points in the lumbar and lateral portions of the body, whereas in enteralgia, tenderness on pressure is generally wanting. Moreover, this affection, as well as myalgia, is generally unilateral.

The TREATMENT involves the same principles as the treatment of gastralgia and other neuralgic affections. (Vide p. 462.)

Lead Colic—Enteralgia from Lead.

The affection now commonly known as saturnine or lead colic has been described by writers at different times and places under a variety of names. Painter's colic, plumber's colic, colic of Poitou, colica pictonum, Devonshire colic, colic of Madrid, rachialgia, dry bellyache, and other names which might be added, all relate to the same affection, which is one of the varied forms under which lead-poisoning is manifested. The affection differs from ordinary colic as regards not only its causation and clinical history, but its pathological character. It is not a spasmodic, but a neuralgic affection; that is, a variety of enteralgia.

It is developed usually in a gradual manner, and is preceded by prodromic phenomena which are the general effects of lead-poisoning—namely, pallor due to anæmia, and frequently an icterode hue of the skin, a peculiar fetor of the breath, and a metallic taste in the mouth, loss of appetite, constipation, pain in the limbs, more or less emaciation, and muscular debility. Pain in the abdomen, at first comparatively slight, progressively increases, becoming at length the chief ailment. The pain is oftenest referred to the region of the umbilicus, sometimes, however, to the epigastrium or hypogastrium, and it may be limited to other situations or it may extend over the whole abdomen, but it is usually confined within a small space. The pain frequently shoots into the back, the genital organs, and in other directions. It is sometimes dull or aching, and at other times acute and lancinating. It varies in different cases within wide limits as regards intensity. In a mild form it merely occasions annoyance, like the pain in certain cases of gastralgia, but in a severe form the suffering is extreme, causing the patient to assume a variety of unnatural and fantastic positions, and to utter loud groans or cries as in severe cases of gastralgia and ordinary colic. The abdomen may be more or less meteorized or tympanitic, but it is oftener depressed and resisting to pressure. The resistance to pressure is caused by tonic contraction of the abdominal muscles, and this in some cases is a marked feature of the affection. Pressure on the abdomen, if made gradually and over a considerable space, is generally well borne, and often affords relief. Patients are inclined to lie upon the belly with a folded pillow or some other substance, and sometimes the closed hand, placed under them over the seat of pain. Pressure, however, with the ends of the fingers or percussion of the abdomen may not be well borne. The bowels are constipated, the exceptions to this rule being infrequent, and the dejections frequently consist of small hard lumps, or scybala. More or less nausea occurs in a majority of cases, and not infrequently vomiting. Hiccough and eructations of gas are not uncommon. The appetite is lost. The urine is scanty and micturition is sometimes difficult and painful. Circumscribed projections or tumors, changing their situation, and due, evidently, to an accumulation of gas in a portion of the intestinal tube, have been observed. The pain may be exclusively in paroxysms, the patient in the intermissions being entirely free from it, but much oftener more or less constant pain is experienced, with exacerbations. The paroxysms or exacerbations vary much in different cases in duration as well as severity. They may last for a few moments only or continue for several hours. The intermissions or remissions also vary in duration from a few moments to hours and even days.

In conjunction with the foregoing local symptoms there is no fever unless there be some accidental complication. The pulse is abnormally slow and frequently irregular. It is generally fuller and harder than in health. The skin is cool. The mind is anxious and depressed. The strength fails in proportion to the severity of the pain and the duration of the affection. The affection may be complicated with other manifestations of lead-poisoning—namely, paralysis affecting certain of the voluntary muscles, especially the extensors of the forearm, amaurosis, neuralgia in different situations, occasionally epileptiform convulsions, delirium, and coma. The latter have been embraced by Tanquerel and other writers under the name of lead encephalopathy.

The affection has no definite duration, in this respect resembling neuralgic affections in general. When left to itself it may end in a few days or after the lapse of weeks, and if the patient continue to be exposed to the cause it may persist for an indefinite period. In itself it involves little or no danger to life, nor is there ground to believe that it leads to any serious abdominal disease; but other effects of lead-poisoning which may complicate or follow this affection, especially the effects embraced under the name of lead encephalopathy, are liable to end fatally. The affection frequently pursues an irregular course, varying in severity much at different periods; and relapses, after all the symptoms have ceased for days or weeks, without any fresh introduction of lead, are not uncommon.

R. Maier found in rabbits poisoned with small doses of lead degeneration and atrophy of the ganglion-cells in the intestinal wall and increase of connective tissue in the ganglia. He and Kussmaul had previously found in a case of lead-poisoning in a human being a sclerosis of the cœliac ganglia. It is probable that lead acts directly upon the sympathetic ganglia in the intestinal wall, causing sclerosis and partial necrosis of these structures.

The introduction of lead into the system in sufficient quantity to give rise to its morbid effects may take place through the lungs, the stomach, the mucous membrane in other parts, and the skin. Certain occupations involve the inhalation of lead. Persons employed in the manufacture of lead paints are most exposed, and of those who suffer from lead-poisoning the largest proportion are of this occupation. Next to this class, painters are most exposed. More or less exposure by inhalation belongs to a great number of occupations, such as paper-staining, grinding of colors, glazing cards, manufacturing earthen pottery, plumbing, shot-making, etc. Lead-poisoning has repeatedly occurred from sleeping in newly-painted rooms. Lead may be introduced through the stomach in various articles of food and drink. The colic of Poitou was due to wine, and that of Devonshire to cider adulterated with lead in order to give it sweetness. This kind of adulteration has been practised since the occurrence of the affection in the places just named, and it is perhaps still sometimes resorted to. Liquid and solid articles of diet, especially if they contain a free acid, become impregnated with lead by being in leaden vessels or earthen vessels glazed with lead. Some years ago a large number of persons within a limited district in Ohio were affected with a disease which for some time was regarded as a novel epidemic, and was called dry cholera. It was found to have the diagnostic characters of lead colic, and was traced to the common use of a cheap kind of earthenware made in that vicinity, into the glazing of which lead entered. An epidemic colic in New Orleans was traced to lead contained in soda-water; and Prof. Doremus some years ago demonstrated the presence of lead in considerable quantity in soda-water drawn from fountains in this city (New York). Water distributed in lead pipes is not infrequently the vehicle of its introduction into the system.[1] Lead sometimes enters into the substances used to color confectionery, condiments, and other articles in sufficient quantity to give rise to poisonous effects. Articles enclosed in lead-foil may become contaminated. Authentic instances of poisoning by the use of tobacco thus enclosed have been reported, and in France enclosing tobacco in lead is prohibited by law. The habit of chewing shot and pieces of the lining of tea-boxes has been known to give rise to various manifestations of lead-poisoning lasting for years before the cause of disease was discovered.[2] During the spring of 1866 two hundred and thirteen cases of lead-poisoning occurred in the Walkill Valley, Orange Co., New York. After considerable research it was ascertained that lead was contained in the flour and meal used in that part of the country. The source of the lead was ascertained to be the stones used for grinding the meal and flour. The stones were old, constantly needing repair, large cavities frequently occurring, which instead of being filled up with cement were filled with common lead. The attrition of the grinding detached minute particles of lead, and the lead thus becoming mixed with the flour and meal was transformed into the carbonate by the process of fermentation and baking.[3] Children may be poisoned by sucking leaden toys, glazed writing-cards, painted artificial nipples, etc. These are only a few of the diverse modes in which lead finds its way into the alimentary canal. The use of lead as a remedy has given rise to colic and other morbid effects. It may be introduced through the skin, although doubtless with difficulty. Lead-poisoning is sometimes attributable to the long-continued application, to the hands and face, of a cosmetic containing lead. I have known a case in which phenomena denoting lead-poisoning were attributable to the use for a long period of a dressing for the hair containing the acetate of lead. Finally, instances of lead-poisoning by means of collyria and vaginal injections have been reported.

The DIAGNOSIS of lead colic in most cases is not difficult. If the occupation of the patient involve obvious exposure to lead, a suspicion of the nature of the affection is of course at once aroused; but this and other manifestations of lead-poisoning not infrequently occur in persons who are not aware of any such exposure, and it may require no small pains to discover the mode in which the poisoning has taken place after the character of the affection has been ascertained. The diagnostic characters of lead colic as compared with those of ordinary colic offer points of difference which enable the physician to discriminate it from the latter. The gradual development, the occurrence of remissions much more frequently than intermissions, the persistency of the affection, the concurrence of pain in the back, limbs, and sometimes in the chest, the occurrence of nausea and vomiting, the obstinacy of the constipation, and frequently dysuria, characterize this affection when contrasted with ordinary colic. Coexisting paralysis in some cases, especially if limited to the extensor muscles of the forearm, confirms the diagnosis. Enteritis and peritonitis are excluded by the absence of symptoms denoting inflammation. Enteralgia, exclusive of its occurrence in connection with hysteria, is so rare, except when produced by lead, that its existence is strong presumptive evidence of this causation. A valuable sign is a blue discoloration of the gums at their junction with the teeth. The blue line on the gums may be apparent on the upper or lower jaw or on both. It is wanting in sections where teeth have been lost. It is most marked in proportion as the teeth are encrusted with tartar and in persons who neglect cleanliness in this situation. The discoloration is attributed to the formation of the sulphide of lead, sulphuretted hydrogen being evolved from the decomposition of particles of food remaining between the teeth and beneath the margin of the gums. The lead may be introduced through the mouth either with the inspired air or in food or drink, or sufficient lead may be eliminated in this situation to account for the sign. The sign is not present in all cases. Its absence, therefore, is not proof that lead colic does not exist. Clinical observation, however, shows that the sign is present in a large majority of cases. It is found frequently to precede the development of colic and other affections dependent on lead-poisoning. It is not a transient sign, but is likely to persist, not only during the continuance of the affection, but for some time afterward. In a diagnostic point of view it is highly important, and the gums should be examined with reference to it whenever lead-poisoning is suspected. A chemical analysis of the urine may show the presence of lead in this fluid. In a case in which the lead was introduced by the use of a cosmetic called "the bloom of youth," lead was obtained from the urine in sufficient quantity to form distinct particles of the metal.[1] Edes states that if the iodide of potassium be given for a few days before an examination of the urine, and the metal be not then found, it is safe to exclude plumbism from the diagnosis.[2]

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[1] Vide appendix to translation of Tanquerel's treatise, and report of Horatio Adams, M. D., in *Transactions of American Medical Association*, vol. v.

[2] Vide case in *Western Lancet*, Cincinnati, communicated by Dr. Edward Murphy of Indiana.

[3] *Philadelphia Medical News and Library*, July, 1866.

The TREATMENT of lead colic embraces palliative and curative measures. The pain is to be palliated by anodynes given by the mouth or rectum, conjoined with soothing applications to the abdomen, as in cases of ordinary colic. Chloroform given by the mouth and applied over the abdomen sometimes acts promptly and efficiently in relieving the pain. If this prove ineffectual, opium in some form is required, the doses and their repetitions to be graduated by the intensity and persistence of the pain. Relief is afforded by free purgation, and for this end saline or such other cathartics as the stomach will best tolerate may be given, and their operation aided by large purgative enemata. The obstinacy of the constipation often renders it difficult to procure free evacuations from the bowels, and active cathartics may be required. The employment of cathartics may be useful in removing lead contained in the contents of the alimentary canal. Aside from this object and the relief of inconvenience from constipation, cathartics are not indicated. Croton oil and other drastic cathartics, heretofore entering into the treatment of this affection, are contraindicated. The warm bath is useful as a palliative, soothing measure.

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[1] For the method of testing the urine for the presence of lead employed by Prof. Doremus, vide my work on *Clinical Medicine*, 1879.

[2] Vide *Therapeutic Handbook*.

Several remedies have been supposed to have a curative efficacy in this affection. Alum is one of these. Brachet, in a prize essay on the subject of lead colic, stated that he had treated 150 patients with this remedy more successfully than by opium and purgatives. From one to two drachms of alum were administered during the day in a ptisan to which 40 or 50 drops of laudanum were added, a mild aperient given on the third day, and the alum continued for two or three days after the symptoms of the affection had disappeared. Other observers have not met with a similar success with the alum treatment. Sulphuric acid has been advocated on the basis of success in a large number of cases. A drachm of dilute sulphuric acid in a quart of sweetened water forms a pleasant drink resembling lemonade, and this may be taken in the twenty-four hours. Tanquerel denied the efficacy of this remedy, giving as the basis of his denial the results of its trial in 53 cases. The prophylactic efficacy of the sulphuric-acid lemonade appears to have been satisfactorily proven in the lead-works at Birmingham, England.[1] Strychnia or nux vomica has been recommended as a curative remedy. Belladonna and nitrite of amyl have been used with some benefit in lead colic. Their use is based upon the view that the colic is due to spasm of smooth muscular fibres, either of the intestine or of blood-vessels.

The remedy now regarded as possessing the most efficacy is the iodide of potassium. Its use in the treatment of lead diseases, and also in those due to the presence of mercury in the system, dates from the publication in 1849 of a paper by Melsens.[2] Assuming that the local manifestations of lead-poisoning are due to the presence of an insoluble compound of lead in the tissues of the affected parts, Melsens claims for the iodide of potassium the power of effecting the liberation of the lead and its elimination from the system. The iodide is supposed to combine with the lead in the tissues, forming the soluble iodide of lead, which passes out of the body in the urine. Chemical examination of the intestine, the paralyzed voluntary muscles, and other organs in cases of lead-poisoning has shown the presence of lead in larger quantity than is to be accounted for on the supposition that it may exist in the body in health. The examination of the urine in cases of lead-poisoning under treatment with the iodide of potassium has shown the presence of lead, its absence in this liquid having been ascertained prior to the treatment.[3] Finally, clinical observation appears to afford proof of the efficacy of this treatment. With reference to the therapeutics of this affection, knowledge of its natural history as regards termination when left to its own course is important. Tanquerel in his valuable treatise gave the results in 31 cases in which no treatment was pursued for twelve days after admission into hospital, the affection having existed for one or more days before admission. Of 8 of these cases in which the colic was violent, in 3 a spontaneous cure took place within the twelve days—namely, on the third, eighth, and eleventh day. Of 13 cases in which the colic was of moderate severity, in 5 a spontaneous cure took place on the fourth, seventh, eighth, ninth, eleventh day, and in 1 on the thirteenth day. Of 10 cases in which the colic was light, in 6 a spontaneous cure took place within the twelve days.

In the treatment of lead-poisoning by the iodide of potassium Melsens enjoins to begin with moderate doses and to increase the doses gradually. He supposes that disturbance may arise from dissolving the lead out of the tissues rapidly. Five grains may be given three times daily at first, and the quantity increased to a drachm *per diem* if the remedy occasion no inconvenience.

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[1] Appendix to Tanquerel's *Treatise*, by Dana.

[2] *Annales de Chimie et de Physique*, 1849; also, *British and Foreign Medico-Chirurg. Review*, 1853.

[3] For facts verifying this statement, see a report of the results of treatment with the iodide of potassium in 23 cases in the New York Hospital by H. S. Swift, M. D., *New York Medical Times*, February, 1854.

The utility of the warm bath as a palliative measure has been already referred to. It may be useful in promoting the elimination of lead through the skin. It is useful by removing from the skin lead which comes into contact with, and adheres to, the surface in persons engaged in certain occupations. The sulphur bath may be employed for the latter object. This is made by adding four ounces of the sulphide of potassium to thirty gallons of water in a wooden bathing-vessel. The use of this bath renders the presence of lead on the skin apparent by the production of a dark discoloration which is readily removed by a brush. The blackness is marked in various parts of the body in painters and workmen employed in the manufacture of the preparations of lead used as paints, especially if they have neglected cleanliness; but it may be due to the presence of lead eliminated from the body and remaining adherent to the surface. The bath renders the lead which may be in contact with the skin inert, and it is useful as affording evidence that the surface of the body is entirely free from lead, whether derived from without or eliminated.

Prevention of the continued introduction of lead into the system is an essential part of the management. The occupation is to be suspended if it involve exposure; and if the affection be renewed or there be other manifestations of poisoning on resuming the occupation, it should be exchanged for some other. If the source of the poisoning be not apparent, it is to be sought for until found. This poison is one which gives no notice of its entrance into the body, and no manifestations of its presence until a certain amount of accumulation has taken place. The affection under consideration is but one, although the most frequent, of the forms of disease to which the poison gives rise. There is reason to think that obscure and indefinite ailments are not infrequently produced by it in cases in which suspicion of the cause is not awakened; and the liability to these as well as the more marked effects from the use of water conducted in lead pipes and various other sources, some of which have been enumerated, is one of the important subjects pertaining to public health.

The protection of those necessarily exposed to an atmosphere more or less charged with lead or to contact with it is also an important subject. Much may be done to prevent its introduction by attention to ventilation, clothing, baths, washing the mouth, and the occasional use of purgatives. Special means of protection, such as the application of moist sponges to the mouth and nostrils or the use of masks, under certain circumstances may be important.

After recovery from the colic an anæmic condition, with general debility, will often claim appropriate medicinal and hygienic measures of treatment.

Colic from Copper.

Writers have described a form of colic supposed to be produced by the introduction and accumulation of copper in the system, and the term *metallic colic* has been used to embrace not only the colic due to lead, but abdominal pains with other symptoms attributed to copper, mercury, and arsenic. The diagnostic features of so-called copper colic, as contrasted with lead colic, have been considered to be as follows: Limitation of pain to the abdomen, increase of pain by pressure, enlargement more frequently than retraction of the abdomen, and diarrhœa with greenish stools. These symptoms denote not merely functional disorder, but gastro-enteric inflammation. An extended and careful examination of a number of establishments in which the workmen were exposed to emanations from, and contact with, copper in various modes, conducted by Chevallier and Boys de Loury in Paris, led to the conclusion that such an affection as colic from copper does not exist.[1] This conclusion, however, is by no means inconsistent with the fact that copper within the system exerts a deleterious effect. Arsenic and preparations of mercury, taken in quantities sufficient to produce the phenomena of a local acrid or irritant poison, give rise to pains which may resemble those of colic. Accumulating within the system, these minerals occasion certain toxical effects; but they do not under these circumstances lead to abdominal symptoms which may with propriety be considered as an individual affection under the name of colic.

Passage of Gall-Stones—Hepatic Colic.

The passage of biliary calculi along the cystic and common bile-duct may give rise to symptoms resembling those of acute gastralgia. The discrimination at once is not always easy. Moreover, a paroxysm of pain from the passage of a gall-stone, or hepatic colic, is to be discriminated from affections other than gastralgia—namely, peritonitis, ordinary colic, colic with intestinal obstruction, acute gastritis, and the passage of calculi from the kidney to the bladder. Biliary calculi usually form in the gall-bladder, but they have been found in the hepatic duct and its branches within the liver. They vary in size from that of a pin's head to that of a hen's egg. They are frequently found in the gall-bladder after death, sometimes in great number. A collection of several hundred is not uncommon. Bennett relates a case in which the number amounted to two thousand, and cases have been reported in which the number was still greater. They are usually of soft consistence and of low specific gravity. They are sometimes white, but oftener either of a yellow, brown, or greenish color from the imbibition of bile. On section they are often found to consist of successive layers which differ in consistence and color. In general they are composed chiefly of cholesterin, but they frequently contain the salts of lime. Remaining within the gall-bladder, they may occasion no inconvenience; but if many or large, they may give trouble by distending the gall-bladder, and they may lead to ulceration and perforation. If the perforation be into the peritoneal sac, diffuse peritonitis follows, and will be likely to prove fatal. If the distended gall-bladder become adherent to the abdominal walls, the perforation may lead to circumscribed abscess which opens externally, and a number of calculi may be discharged through the fistulous opening.[2] They are oftener discharged by an ulcerated opening into the intestinal canal. The opening may take place into the duodenum or colon. It has been known to take place into the stomach, the portal vein, and the pelvis of the kidney. Gall-stones sometimes occasion a noise like that produced by the collision of nuts or marbles in a bag when shaken if forcible pressure or strong percussion be made over the distended gall-bladder; but in general their existence is not suspected until, having entered the cystic or common duct, they occasion, by their passage toward the intestine, paroxysms of so-called hepatic or gall-stone colic. It is evident that the time occupied in the passage of a gall-stone, as well as the suffering, will depend, other things being equal, on its size and the irregularity of its shape.

[1] Vide Nysten's *Dictionnaire de Médecine, etc.*, 1858; also Valleix, *op. cit.*, tom. v.

[2] For an account of reported cases, and also cases of cholecystotomy, vide article by Dr. G. W. H. Kemper, in *Transactions of the Indiana State Medical Society*, 1879.

The pain occasioned by the passage of a gall-stone is frequently abruptly developed without any obvious cause, but in some cases it is preceded by a sense of uneasiness referred to the right hypochondrium. In the majority of cases the attack occurs three or four hours after a meal. The suffering is often extreme, causing the patient to writhe and assume a variety of positions with the hope of obtaining relief. The pain is referred to the epigastrium or the right hypochondrium. It may radiate thence throughout the abdomen, often extending into the chest and to the right shoulder. Usually, there is more or less tenderness over the seat of the pain, but in some cases pressure affords a certain measure of relief. Vomiting speedily occurs in the majority of cases, and is repeated more or less frequently during the continuance of the pain. The bowels are constipated. The pulse, as a rule, is not accelerated, and it may be retarded. Not infrequently it becomes small and weak. The retardation of the pulse is sometimes notably great. I have known it to fall to 30 and to 20 beats per minute. Pallor of the face and coldness of the surface are marked in some cases. Chilly sensations, with rigor, are not uncommon. The duration of the paroxysm is variable. It may last for a few moments only or for many hours or days, depending of course on the period occupied by the passage of the stone either onward into the intestine or backward into the gall-bladder. At length the pain and other symptoms suddenly cease, and nothing remains but more or less soreness and the fatigue occasioned by the suffering. If the paroxysm have been of considerable duration, the urine may show the presence of bile, and jaundice is frequently produced. These symptoms disappear after the paroxysm is ended. The dejections may show deficiency or absence of bile. The absorption of bile and its absence in the stools show that the gall-stone is impacted in the common (not the cystic) duct, and that its form and size are sufficient to cause obstruction more or less complete. Under these circumstances the bile may accumulate within the gall-bladder, and distend it so that it can be perceived by the touch below the lower margin of the liver. Finally, after the paroxysm is ended one or more gall-stones may be discovered in the dejections if these be carefully examined for several successive days. In some cases, however, careful examinations of the feces fail to show the presence of a calculus or of calculi. The failure to discover them is not proof that an attack is not one of hepatic colic. The gall-stones in these cases, after remaining for a time in the cystic duct, may have passed backward into the gall-bladder, notwithstanding the valvular folds of the lining membrane. Another explanation is that the cause of the obstruction is inspissated bile, and not properly gall-stones.[1]

After an attack of hepatic colic recurrences are to be expected, but as regards the intervals cases differ greatly. Successive attacks may occur after days, weeks, months, or years. Not infrequently a greater or less number of attacks follow in quick succession—several perhaps occurring on the same day and for several days in succession. This is probably owing to a temporary dilatation of the cystic and common duct produced by the passage of calculi of considerable size, in consequence of which other stones within the gall-bladder more readily escape. The patient under these circumstances may be consoled by the reflection that the more frequent the passage of calculi the less the number that remains to give rise to subsequent attacks. The frequent repetition of attacks, if severe, may occasion considerable disturbance of the digestive system and general prostration. A single instance of death during a paroxysm has come under my observation.

The differential diagnosis of the passage of gall-stones from acute gastralgia is to be based on the following points: The occurrence of pain not excited by any obvious dietetic or other cause; the prominence of vomiting as a symptom; the presence generally of more or less tenderness; the existence of bile in the urine, and of icterus or an icterode hue of the conjunctiva and skin; retardation of the pulse; the persistence of pain notwithstanding opiates are given in full doses; the sudden relief occurring irrespective of remedies; and after the attack the discovery of biliary calculi in the evacuations.

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[1] The presence of inspissated bile in the dejections may be ascertained by chemical tests. (Vide Bartholow in *System of Medicine, by American Authors*, vol. ii. p. 1073.)

The examination of the evacuations for gall-stones not only serves to establish the diagnosis, but on the appearance of the stone or stones may be predicated an opinion that the gall-bladder either does or does not contain calculi which have not escaped. If only a single round or oval stone be discovered, it is probable that this was a solitary calculus in the gall-bladder, and other attacks need not be expected; but if several stones be discovered, polyhedral in form, with smooth or polished facets, which may be plane, convex, or concave, it is highly probable that the gall-bladder contains others, and the patient cannot count on being exempt from repetitions of the paroxysms produced by their passage. The only effectual mode of examining for the presence of stones is to dilute the feces and strain through a sieve.

Acute diffuse peritonitis may be accompanied by pain resembling in intensity that of hepatic colic; but in determining the existence of the latter, peritonitis is excluded by the absence of tenderness diffused over the abdomen, of rigidity of the abdominal walls, of tympanitic enlargement, of notable acceleration of the pulse and pyrexia, together with the general prostration which belongs to the clinical history of peritoneal inflammation. In cases of intestinal colic the pain is more paroxysmal, with intervals of complete relief. Intussusception and internal strangulation of the intestine are to be excluded by the absence of a tumor caused by the accumulation of the intestinal contents at the point of obstruction, by the absence of intestinal obstruction and of acceleration of the pulse, together with the greater gravity of the general symptoms in those affections. In acute gastritis vomiting is a more prominent symptom than in hepatic colic, and the greater gravity of the affection is shown by the pulse and by the general symptoms. In some cases of hepatic colic the pain extends downward in the direction of the right ureter, so that the passage of a calculus along this duct may be suspected. The absence of certain symptoms belonging to the history of so-called renal colic—namely, retraction of the testicles, deficiency of the urine, hæmaturia, etc.—will serve to exclude this affection. Knowledge of the occurrence of one or more prior attacks which have been ascertained to be due to the passage of gall-stones will aid in determining the diagnosis. The age of the patient is of some account in the diagnosis. Gall-stones may form at any age, but they are most likely to form in middle or advanced life, and in females oftener than in males.

In the great majority of cases gall-stones escaping from the gall-bladder find their way into the intestine, giving rise to no evil results beyond the pain, etc. which their passage occasions; but occasionally a stone becomes impacted in the biliary canal, and if situated in the common or hepatic duct retention of bile ensues, giving rise to persisting jaundice and structural changes within the liver from the distension of the bile-ducts. Charcot has described paroxysms resembling those of malarial fever, sometimes recurring regularly and presenting the quotidian, tertian, or quartan type of an intermittent, and sometimes recurring without regular periodicity. To these paroxysms this author has applied the name *hepatic intermittent fever.* They differ from intermittent paroxysms due to malaria in the time of their occurrence, this being usually in the afternoon or evening, whereas malarial paroxysms generally occur before midday. They are probably caused by septic infection incident to suppurative inflammation of the duct at the seat of the impaction. The prognosis is unfavorable, death taking place sometimes suddenly in a paroxysm. Recovery may follow the passage of the stone, either through the common duct into the duodenum or by ulcerative perforation into the intestine.[1]

Stenosis of the colon or duodenum may result from the projection of the ulcerated opening into those portions of the intestine. Intestinal hemorrhage is an occasional effect of ulcerative perforation, and death may take place from the loss of blood. Permanent obliteration of the canal of the cystic or common duct may result from the inflammation and ulceration caused by the presence of the stone. The formation of a stone within the liver is one of the causes of hepatic abscess.

The rationale of the formation of gall-stones is not well understood. Probably the deposit in a solid form of certain of the constituents of bile takes place in consequence of the presence in the biliary passages or gall-bladder of mucus, collections of which serve as the nuclei for the formation of calculi. Hence gall-stones may be the result of inflammation of the gall-bladder. It has been conjectured that cholesterin may be deposited in consequence of the bile losing the faculty of holding this principle in solution from a diminution of the taurocholic acid. It is not improbable that an excess of the salts of lime or of bile-pigment may lead to the production of calculi. Undue accumulation and retention of bile in the gall-bladder, and an unduly large proportion of solid constituents, may be considered as entering into the causation.

The TREATMENT of a paroxysm of hepatic colic consists of palliative measures. Opiates are called for in doses proportionate to the intensity of the pain, either administered by the mouth or rectum or hypodermically. If the suffering be great, the inhalation of chloroform or ether should be resorted to. Warm anodyne fomentations may be applied to the seat of pain. The hot bath may aid in procuring relief. The local application of cold by means of a bladder filled with pounded ice has been found to be useful as a palliative measure. Remedies to palliate vomiting may be indicated. Effervescing draughts and the hydrocyanic acid may be employed for this purpose. An alkaline remedy may be found useful if, as is frequently the case, the matters vomited show notable acidity of the stomach.

Emetics and cathartics have been advocated under the notion that they aid in the passage of the stone. It is irrational to suppose that they have this effect, and clinical observation does not furnish evidence of their value. The passage of the stone is due to the accumulation of bile behind it after it enters the common duct, and to contraction of the gall-bladder, which contains muscular fibres. These are wanting in the cystic and common ducts. Exciting the movements of the stomach and intestine by emetics and cathartics, if any effect be exerted, is as likely to retard as to favor the onward passage of the stone. Opium and anæsthetics probably promote its passage. If the distended gall-bladder form a tumor which is appreciable by the touch, an effort may be made, by pressure with the fingers, to propel the bile in the ducts and thus aid in the removal of the obstruction.

It is desirable, if possible, to effect the solution of gall-stones within the gall-bladder. The alkaline carbonates are given for this purpose. A treatment for this end, known in France as Durande's method, consists in the administration of sulphuric ether and the spirits of turpentine (3 parts of the former to 2 parts of the latter). Buckler[1] has proposed the use of chloroform in view of the solvency of cholesterin in this liquid. He has reported a case in which an indurated tumor felt through the walls of the abdomen, directly over the inferior margin of the liver, disappeared in five days under the use of chloroform given in teaspoonful doses after each meal. It was concluded that the tumor was the gall-bladder distended with calculi, but there is room for doubt as to the correctness of this diagnosis. It seems hardly probable that any remedies can be introduced into the system so as to enter into the composition of the bile largely enough to dissolve the cholesterin of which mainly biliary calculi are composed. Bartholow proposes to puncture the gall-bladder with a fine exploring trochar and to inject through the canula a suitable solvent.

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[1] Vide cases reported by Prof. William Osler, in *Medical Times and Gazette* (London), July 30, 1881.

The occurrence of gall-stones cannot be anticipated, but, being known, it is of course desirable to prevent the formation of others. It is to be remarked that clinical experience is not to be relied on as affording evidence of prevention, inasmuch as the success of preventive measures cannot be proven. It would seem that sedentary habits predispose to the formation of gall-stones. A change in this respect is therefore advisable. Harley and Bartholow attach much importance to restriction of diet as regards fatty, starchy, and saccharine articles of food. Buckler suggests the hydrated succinate of the peroxide of iron in cases in which gall-stones are known to have been passed, the object being to prevent their further formation, this remedy, as he supposes, controlling the fatty or cholesteric diathesis.[2] The alkaline mineral waters of Carlsbad, Vichy, and Marienbad are held in much repute as preventing the formation of gall-stones. The soda salts are considered to be most effective by Harley and Bartholow. The sulphate of soda is recommended by Harley, and the phosphate by Bartholow. The latter recommends a drachm of the phosphate of soda three times daily. He attributes the usefulness of this remedy in part to a cholagogue effect. Alonzo Clark considers the bicarbonate of soda an effective remedy, provided it be continued for a sufficiently long period. This remedy can be taken in considerable doses for a very long period without any apparent ill effects. A medical friend of the author has taken from a quarter to half an ounce daily for the last thirty years.

Gall-stones of sufficient size to occasion intestinal obstruction are sometimes lodged in the alimentary canal. These escape from the gall-bladder, not through the biliary passages, but by ulceration, the walls of the gall-bladder becoming adherent to the duodenum or to some other portion of the intestine.

Olive oil, taken largely, is a popular remedy for gall-stones. This remedy sometimes causes the discharge of small, hard bodies supposed to be biliary calculi. These are composed of a concrete form of fatty matter, like the bodies passed in some of the cases of fatty diarrhœa which have been reported. A case is quoted by Dunglison in which olive oil, taken abundantly for the relief of pains attributed to gall-stones, caused a discharge of fatty matters of a globular form, varying in size from that of a small pea to that of a grape, and of sufficient consistence to be cut with a knife, like soft wax.[3]

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[1] *American Journal of the Medical Sciences*, July, 1867, p. 51.

[2] This remedy is given by Dr. Buckler as follows: Hydrated succinate of the peroxide of iron, ℥iss; water, f℥viss: a teaspoonful after each meal, continued for several months, and resumed at intervals afterward.

[3] *Dunglison's Practice of Medicine*, 1842, vol. i. p. 135.

The reader is referred to *A Treatise on Gall-Stones*, by J. L. W. Thudicum, M. D., London, 1863, for a full consideration of their chemistry, pathology, and treatment. Vide also Murchison's *Clinical Lectures on Diseases of the Liver, etc.*

CHAPTER VIII.

SPORADIC CHOLERA.—CHOLERA INFANTUM.—ASIATIC CHOLERA.

Sporadic Cholera: Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Cholera Infantum.—Epidemic Cholera: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Prevention; Treatment.

SPORADIC CHOLERA may be considered as a functional affection of the stomach and intestine. Epidemic or Asiatic cholera belongs more properly among infectious diseases; but inasmuch as the symptoms and lesions are in great part referable to the intestine in this affection, it is placed in this chapter for convenience of reference. Moreover, the symptoms in some cases of sporadic cholera simulate closely those of epidemic cholera, and this is a reason for placing the two affections in juxtaposition. Cholera infantum, belonging among the diseases of children, does not come within the scope of this work. I shall, however, notice it briefly, referring the reader for a fuller consideration of it to works devoted to the diseases of children.

Sporadic Cholera.

The affection called cholera in its ordinary form is commonly known as *cholera morbus.* Aside from the incongruity of associating these two words, the one of Greek and the other of Latin derivation, there is no good reason for appending the word morbus, which expresses no more than is implied in the name of any affection—namely, the existence of disease. The etymology of the term cholera is somewhat doubtful, but it is supposed to signify a flow of bile. The term is of ancient date, and was introduced when various liquids were embraced under the name of bile. Not only is the term sanctioned by long usage, but it would be difficult, with our present knowledge, to substitute a better name for the affection under consideration. The term sporadic serves to distinguish this from another affection known as cholera which prevails as an epidemic. To distinguish it from the latter, which emanated from India, it is sometimes called by French writers *European*, and by British writers *English cholera.* Writers on this side of the Atlantic might with equal propriety call it American cholera, since it is sufficiently common as an indigenous affection in America. It is called by German writers *cholera nostras.*

CLINICAL HISTORY.—An attack of sporadic cholera is often abrupt, but in some cases it is preceded, for a period usually brief and rarely longer than a few hours, by a sense of weight or uneasiness referred to the epigastrium or extending more or less over the abdomen, with nausea, occasional colic pains, etc. The attack occurs much oftener in the night than in the daytime. It is ushered in by vomiting, which is speedily followed by purging. Vomiting and purging, occurring in quick succession and recurring either simultaneously or in close alternation, constitute the prominent and distinctive symptomatic features of this affection.

The evacuations from the stomach and bowels are generally abundant, and at first consist of the alimentary and fecal contents. Afterward liquid is vomited, sometimes in large quantity, often having the appearance and taste of bile, sometimes acid, and sometimes so acrid as to occasion a scalding sensation in the throat. In like manner, the dejections become liquid and occasion frequently a burning sensation at the anus. The acts of vomiting and purging occur suddenly with but little premonition. They are often violent, the contents of the stomach and bowels being ejected with force. The acts of purging are preceded and accompanied by colic pains, often extremely severe, which are temporarily relieved by a free evacuation. Severe pains, like those of spasm or cramp, often attend the acts of vomiting. In the interval between the acts of vomiting the patient usually suffers from dryness of the mouth and fauces with urgent thirst. The abdomen may be at first voluminous, but after repeated acts of vomiting and purging it becomes contracted. Tenderness over the abdomen is either wanting or slight. Spasm of the abdominal muscles sometimes occurs, causing hard bunches or knots, as in some cases of colic. Hiccough is an occasional symptom.

With the foregoing local symptoms are associated anxiety, restlessness, and a sense of exhaustion proportionate to the violence and duration of the attack. The pulse is more or less accelerated, small, and feeble. The skin is cool or even cold, and is frequently bathed in clammy sweat. The voice is feeble, sometimes altered in quality, and occasionally there is aphonia. In some severe cases cramps occur in the feet, the calves of the legs, and in other situations. These add greatly to the suffering of the patient.

The affection runs a rapid course. After continuing for several hours the acts of purging and vomiting become less severe and occur at longer intervals; the pains, etc. progressively diminish, until at length all the symptoms disappear, leaving an amount of fatigue proportionate to the violence and duration of the attack. The patient usually recovers rapidly. It is remarkable that an affection involving so much disorder should leave the affected organs in a condition to resume so speedily the exercise of their functions. This is the rule as regards the progress and termination of the affection, even if its course be not arrested by remedial interference. Exceptionally, the vomiting and purging continue, the evacuations from the bowels occur involuntarily, the pulse becomes more and more feeble, coldness of the surface is more and more marked, and contraction of the features takes place; in short, the patient falls into a state of collapse, and dies within a few hours from the onset of the attack. This unfavorable course is extremely rare. I cannot recall an example that has fallen under my observation exclusive of cases occurring in infancy.

PATHOLOGICAL CHARACTER.—Sporadic cholera appears to combine morbid conditions belonging to other functional affections of the alimentary canal. In a certain proportion of the cases considered as cases of cholera the symptoms are attributable simply to indigestion. Vomiting and purging denote in these cases the coincidence of acute dyspepsia and crapulous diarrhœa. Such cases are usually comparatively mild, and the vomiting and purging cease as soon as the offending contents of the stomach and bowels are expelled. In other cases something more than indigestion is involved in the pathology. The evacuation of liquid, often in great abundance, shows hypersecretion or morbid transudation into the stomach and intestines. The affection then combines gastrorrhœa and enterorrhœa. In some cases the secretion of bile seems to be increased, but it is difficult to estimate, from the color and the bitter taste of the vomited liquid, the relative proportion of bile. It has been common to suppose that in this, as in various other affections, disorder of the liver is in some way involved. There is no solid foundation for such a supposition; it rests on conjecture alone. It is only in a certain proportion of cases that the bile appears to be secreted in morbid quantity. The limit of our present knowledge of the pathological character of the affection is reached when the different morbid conditions into which it is resolvable are enumerated. These morbid conditions or elements of the affection are indigestion, irritation, spasm of the muscular coat of the stomach and intestines, enteralgia, morbid gastric and intestinal transudation, and perhaps in some cases hypersecretion of bile. By German authors the affection is considered as a variety of gastro-intestinal catarrh. It is impossible to distinguish, by means of post-mortem appearances, sporadic from epidemic cholera.

CAUSATION.—Sporadic cholera is not peculiar to any country, but it is of more frequent occurrence in warm than cold climates. In cold and temperate latitudes it occurs very rarely except during the summer and autumnal months. An elevated temperature is thus involved in its causation, either by contributing to the development of causes or by rendering the system more liable to be affected by them. The affection occurs oftener in childhood, youth, and middle age than in advanced life, and oftener in males than females. Frequently an attack seems to be attributable to the ingestion of particular articles of food or drink. Uncooked vegetables, fruits, ices, etc. are often supposed to stand in a causative relation to it. An attack may follow the arrest of digestion from various causes. In some cases, as already stated, the affection is apparently due to indigestion alone. It is probable that exclusive of these cases the affection involves a special cause, the nature and source of which are unknown. In several cases of sporadic cholera Finkler and Prior have found in the dejecta comma-shaped bacilli which bear some resemblance to the bacilli of Asiatic cholera, but which can be distinguished from the latter by cultivation in different media. Although Finkler and Prior consider their bacilli as the cause of sporadic cholera, other observers have failed to find comma-shaped bacteria in this disease.

DIAGNOSIS.—The diagnostic features of an attack of sporadic cholera are so strongly marked that little need be said under this head; yet there is some liability to errors of diagnosis. I have known a case of acute peritonitis, accompanied by vomiting and purging, to be considered as a case of cholera, the nature of the disease not being discovered until the autopsy was made. It is extremely rare for vomiting and purging to have sufficient prominence early in peritonitis to lead to this error. The diagnostic marks of peritonitis, however—which will be considered in another chapter—should always be sought after in cases of apparent cholera. The chief liability to error in diagnosis relates to cases of poisoning from the ingestion of acrid or corrosive substances. It is important to discriminate these cases from cholera with a view to antidotal treatment, prognosis, and medico-legal considerations. The following are the differential points: The acrid or corrosive poisons occasion vomiting, which continues for some time before diarrhœa occurs, whereas in cholera the purging is simultaneous with or follows quickly the vomiting. Moreover, the vomiting in cases of poisoning is out of proportion to the diarrhœa. The symptoms in cases of poisoning denote a condition of greater gravity; the pulse is more frequent and smaller, the expression is more haggard, etc., than in the majority of cases of cholera. Redness or charring of the mouth and throat distinguishes certain cases of poisoning. There is marked tenderness over the stomach in cases of poisoning, and more constant, excruciating pain in this situation in the intervals between the acts of vomiting; in short, the symptoms of gastritis are present. The matter vomited is frequently bloody. Finally, in cases of poisoning the symptoms are developed soon after a meal or the ingestion of something containing the poison.

During the prevalence of epidemic cholera it is not always easy to distinguish cholera nostras from Asiatic cholera. In doubtful cases the recognition of the cholera bacilli of Koch and their cultivation suffice to establish the diagnosis.

PROGNOSIS.—As already stated under the head of the Clinical History, this affection in the vast majority of cases intrinsically tends to recovery. A fatal termination is a rare exception to the rule; yet the fact that the termination is sometimes fatal is to be borne in mind, and should enforce prompt and effective measures of treatment. Occurring in persons already affected with some important disease, the prognosis is of course not so favorable as when healthy subjects are attacked.

TREATMENT.—In the treatment of sporadic cholera the first point is to ascertain whether the matters vomited have contained ingesta in more or less abundance. If not, and if there be reason to suppose that the stomach contains undigested aliment, a mild emetic may be given. Almost invariably, however, the contents of the stomach are expelled with the first acts of vomiting, and as an emetic is indicated for no other object it is very rarely called for. In my own experience I have not prescribed an emetic in this affection for the last thirty or more years. The next object is to arrest the vomiting and purging, together with the pain, etc., by the employment of some form of opiate in doses sufficient to effect this object. In the vast majority of cases this object may be speedily effected. The form of opiate should be chosen with reference to promptness of action and its retention. Laudanum, the black drop, and an aqueous solution of opium are well suited to the management of this affection, but Magendie's solution, or a salt of morphia placed dry upon the tongue, is in general the best form. A full dose—a quarter or half a grain of a salt of morphia or an equivalent dose of any other preparation to an adult—should be given directly after an act of vomiting. The first dose will perhaps be immediately rejected; if so, a second should be at once given. If a second and third dose be instantly or quickly rejected, the administration by the mouth should be abandoned, and from one to two drachms of laudanum or an equivalent dose of some other preparation may be given in a little thin starch or mucilage *per enema*. The enema should be given directly after an evacuation, and the patient should be instructed to resist as long as possible the inclination to expel it. If the first enema be quickly rejected, a second and a third may be given. If the attempt to administer the opiate by the mouth and rectum fail, the hypodermic injection may be resorted to. In whatever way the remedy be introduced, it is to be repeated after an interval of from half an hour to an hour until the vomiting and purging are arrested, the effects of each dose being carefully watched and the intervals being sufficiently long to avoid any risk of inducing narcotism.

An important part of the treatment is the withholding of liquids. The intense thirst leads the patient to drink largely between the acts of vomiting, and this tends to prolong the affection. The drink should be restricted to a tablespoonful of ice-water at short intervals, or, what is better, small pieces of ice may be taken frequently and allowed to dissolve in the mouth. Strict compliance with injunctions on the score of drink is essential to the prompt success of the treatment. If there be great prostration a little spirit and water may be taken, if retained, at short intervals.

In my experience this method of treating sporadic cholera has proved uniformly successful, and complete relief may generally be expected within an hour. No apprehension need be entertained with respect to the sudden cessation of the vomiting and purging; and the more quickly the arrest is made the better after a free evacuation of the stomach and bowels. Mercury is, to say the least, superfluous. The success of treatment without it is all that could be desired.

Care as regards diet is alone required in most cases after the affection is arrested.

I do not deem it necessary to consider other methods of treatment, since the success of the plan just stated is, as far as my experience goes, uniform and immediate. The only objection to the plan is that some persons are unpleasantly affected by opium, and after the affection is arrested the effects of the remedy may be more or less annoying for a time. This objection has not much weight, since the after-effects of opiates, however distressing, occasion far less suffering than the continuance of cholera; but they who frequently experience annoyance after the use of opium, under certain circumstances take it without inconvenience, and I have been led to observe that the after-effects are likely to be slight or wanting when opium is given in this affection to patients who usually suffer from these effects.

Cholera Infantum.

The name cholera infantum, or the more popular phrase *summer complaint*, is supposed by some to denote an affection peculiar to this country. The morbid phenomena, however, considered by American writers as belonging to this affection are described in European works under other names. Cruveilhier enumerates many of the symptoms as occurring in cases in which gelatiniform softening of the stomach due to the post-mortem action of the gastric juice is found after death. British writers on diseases of children generally describe the symptomatic phenomena under the head of diarrhœa. Weaning brash, watery gripes, and choleric fever of children are names under which these phenomena were described by Cheyne, Armstrong, and Copland. By some French writers they have been described as belonging to colo-enteritis, follicular enteritis, choleriform diarrhœa of children, and gastro-intestinal catarrh. German authors consider the affection as gastro-intestinal catarrh.

As commonly used in this country, the term cholera infantum, or summer complaint, embraces different pathological conditions—in fact, distinct affections occurring in children under two years of age. These different conditions, however, may be developed at different periods in the same case. In certain cases the symptoms closely resemble those of the sporadic cholera of adults. A child is seized with vomiting and purging, the latter usually occurring first; the acts of vomiting and purging are violent and frequently repeated; after the contents of the stomach and bowels are expelled the evacuations consist of secreted or transuded liquid in more or less abundance. The attack may cease or be arrested and recovery speedily ensue, as in cases of the sporadic cholera of adults; but this favorable course does not obtain so generally in children as in adults. If the course be unfavorable the vomiting and purging continue; the child is tormented with thirst, but everything is rejected from the stomach; great prostration ensues; collapse follows, and death takes place in one, two, or three days. The fatal result may be preceded by convulsions and coma. In some cases the violent symptoms of cholera cease, and the attack eventuates in a chronic affection accompanied by diarrhœa and occasional vomiting.

In other cases the affection is gastro-intestinal indigestion. These are characterized by diarrhœa, the dejections being lienteric and watery. Vomiting occurs either occasionally and is an accidental symptom, or it may not occur. The diarrhœa is more or less persisting, the discharges being often green in color—an appearance which Golding Bird, and more recently J. Lewis Smith, have shown not to depend on vitiated bile;[1] the appetite is impaired or lost; colic pains are often troublesome; the child progressively wastes, and death may take place from inanition. In another class of cases the affection is either enteritis or entero-colitis. The dejections in these cases contain gelatinous or stringy mucus, and are not infrequently tinged or streaked with blood. Febrile movement, with remissions, occurs in these cases. There is more or less abdominal tenderness and pain. Vomiting may be prominent as a symptom or it occurs only occasionally. Progressive emaciation and debility mark the unfavorable progress of this affection. Head symptoms are often developed in its course. The child becomes dull and somnolent and lies with the eyelids partially closed, frequently rolling the head from side to side. Convulsions and coma may occur. After death an examination reveals the evidences of inflammation of the mucous membrane of the small, and perhaps also of the large intestine, the follicles being especially involved. Enteritis or entero-colitis is liable to become developed in the cases which at first appear to be cases of diarrhœa dependent merely on indigestion. In still another class of cases the symptoms and the appearances after death denote the existence and limitation of inflammation to the large intestine.

Under the name of cholera infantum, then, are commonly embraced cases of sporadic cholera, diarrhœa from indigestion, enteritis, colo-enteritis, and dysentery. The head symptoms which are likely to be developed in unfavorable cases of each of these affections in the young child were formerly incorrectly attributed to inflammation of the meninges of the brain. They constitute the hydrencephaloid affection of Marshall Hall.

The affections embraced under the name of cholera infantum are not, as already stated, peculiar to America, but they doubtless prevail to a much greater extent in certain parts of this country than in Europe, owing, probably, in a great measure, to the greater heat in the summer months. The causation has relation to a high temperature, the affections prevailing almost exclusively during the months of June, July, August, and September. Something more than temperature, however, is concerned in their production, for they are almost limited to cities and large towns, and are more prevalent in the Northern and Middle than in the Southern States. Children of the poorer classes, in insalubrious situations and living in crowded dwellings, are more liable to be affected, but the children of those in easy circumstances and of the wealthy by no means escape. The irritation of dentition and the change of diet after weaning doubtless render the system more vulnerable. Infants brought up by hand are especially subject to these affections. The diet of the child after weaning has much to do, if not with the causation of these affections, at least with the ability of the system to resist and overcome them. Restriction to insufficient articles of nourishment, such as arrowroot and gelatin, is one source of innutrition in infancy; but there is reason to believe that infant mortality in cities is attributable in no small measure to the use of diluted, sophisticated, and artificial milk. The importance of undiluted milk from a pure source to the welfare of children is far from being generally appreciated. The quality of milk is of comparatively little consequence after childhood, because it then usually enters but little into the diet, but as the chief reliance is upon milk in infancy its purity is then of the utmost importance.

The general principles which should govern the management of the same affections after the period of early childhood are applicable to the different forms of disease embraced under the name of cholera infantum, with certain modifications which will be here referred to very briefly. The treatment, in the first place, has reference to the form of cholera infantum which is presented. In the acute form, resembling the cholera of adults, in which there is a liability to a rapidly fatal termination by exhaustion from loss of fluids, the patient falling into a state of collapse, the indications are to arrest the vomiting and purging, to restore warmth to the surface by the application of dry heat, mustard-water, and the warm bath, and to sustain the powers of the system by stimulants and nourishment. For the arrest of the vomiting and purging opiates are to be relied upon, as in the cholera of adults, but they are to be given with much circumspection in order to avoid risk of narcotism. Exclusive of this form, the affections do not occur with a sudden and violent attack, but as a rule they are developed gradually and tend to become chronic. Measures for the relief of vomiting are—a sinapism to the epigastrium; creasote, which in doses of a sixth or an eighth of a drop in mucilage, repeated after each act of vomiting, sometimes acts like a charm; the subcarbonate of bismuth in doses of from ten to thirty grains; small doses of calomel with chalk, chloroform, and hydrocyanic acid. With reference to this symptom a careful regulation of the ingesta is requisite, and milk with lime-water, given in small quantity at a time, will often be retained when other articles of nutriment are rejected.

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[1] Vide *Smith on Diseases of Children.*

With reference to the diarrhœa, occasionally a laxative is useful, and a few grains of calomel or blue mass, followed by the syrup of rhubarb, are well adapted for this purpose. There are no indications for mercury except as a remedy for vomiting and an occasional laxative remedy, the convenience of its administration being perhaps its chief recommendation in cases of young children. Opium in some form with due circumspection is to be employed. It is most efficient when given by enema, provided it be administered with proper care and retained. If given by the mouth, the Dover's powder is an eligible preparation. Hypodermic injections of morphia should not be given. If the dejections denote acidity, chalk in powder or mixture, or lime-water, is indicated. The mineral and vegetable astringents are highly important. Of the mineral astringents, the most efficient are bismuth, lead, and the persulphate or pernitrate of iron. The nitrate of silver is highly recommended by some authors. Of the vegetable astringents, krameria, tannic acid, kino, catechu, hæmatoxylon, geranium, and rubus villosus are articles from which a selection may be made or which may be tried in succession. Dr. W. Byford Ryan claims remarkably good results from belladonna conjoined with nux vomica and arsenic.[1]

Attention to diet and regimen is of prime importance. Many children, there is reason to believe, die with these affections from an insufficiency of nutriment. Pure milk is the most appropriate article of food. It may be rendered more nutritious by the addition of boiled flour, powdered biscuit, or other farinaceous preparations. Gelatin and arrowroot or other amylaceous substances alone are inadequately nutritious. The essence of meat and strong broths are often taken with avidity by very young children, and the desire for them should be indulged and encouraged. This remark is also applicable to broiled tender meats taken in a solid form. Within the past few years the use of raw meat has been recommended as a form of diet adapted to the affections under consideration, and it is frequently taken with relish. Tender beef is to be preferred, but mutton and chicken are sometimes more acceptable to the patient. The meat is to be finely hashed, and may be given in this way, or it may be made into a kind of *purée* by being reduced to a pulp in a mortar and pressed through a fine sieve, so as to separate the vessels and areolar tissue. If the child manifest an aversion to it as thus prepared, the hashed meat or pulp may be rendered palatable by the addition of salt, sugar, or some kind of preserve, or it may be added to a broth made with sago or tapioca. Cooked tender meats, however, are generally more acceptable, equally digestible, and not less nutritious. The liability to tænia as a result of eating uncooked meat renders its use of doubtful propriety.

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[1] *American Practitioner*, June, 1885.

As regards the administration of different kinds of food, the quantity given at a time is to be graduated by the ability of the stomach to retain and digest it; and this is the only rule of limitation. It is of vast importance for the physician not to deem it too trivial to enter into full and minute directions with respect to the details of nourishment. He should bear in mind that children often die from innutrition through the apprehensions and ignorance of nurses and mothers.

In conjunction with diet, alcoholic stimulants are highly useful. A few drops of brandy or some kind of spirit may be given at short intervals with advantage. The preparation known as calisaya cordial or elixir is well suited to children, combining a tonic and stimulant in a form agreeable to the taste. The need of a stimulant is shown frequently by the avidity with which children take it in these affections.

Finally, removal from the city, either to a situation on the seaboard or to a salubrious point inland, is the most efficient of all curative means. This measure, if not deferred too long, rarely fails. If other measures be not speedily successful, this, whenever practicable, should be resorted to without delay. If it be not practicable, the child should be in the open air as much as possible, and should be carried daily as far from home as convenient, in order in this way to secure a change of air.

The head symptoms referred to in the clinical history claim sustaining measures, stimulants, and nourishment, conjoined with mild revulsives.

In these affections hope of recovery need not be relinquished under an assemblage of symptoms which may seem to render the prognosis as unfavorable as possible. Patients sometimes recover after lingering for a long time on the verge of the grave. The reason is that the affections, when they prove fatal, do not necessarily involve incurable lesions of structure, but a fatal result is due to protracted irritation, exhaustion, and innutrition.

Epidemic Cholera.

The term epidemic cholera is selected as the one most frequently used and the most convenient to designate a disease which has received a great variety of names, such as Indian, Oriental, and Asiatic cholera, cholera asphyxia, spasmodic cholera, malignant, pestilential cholera, etc. etc. The etymology of the word cholera, it is true, is singularly inappropriate, accepting the derivation which signifies a flow of bile; but our present knowledge does not warrant a name expressive of the pathological character of the disease. Moreover, it is to be presumed that the disease is not essentially the same as sporadic cholera. The two are not merely different varieties of one disease, but they are distinct individual affections, having certain prominent symptoms in common, but differing in essential points relating to clinical history, pathological character, causation, and prognosis.

For an account of the prevalence of this remarkable epidemic in different countries and at different times the reader is referred to other works. Suffice it to say that the disease appears to be indigenous in India, and to have existed there, both as a sporadic and an epidemic affection, for a long period. In other parts of the world it has made brief visitations solely as an epidemic. In 1817 it began its march from Bengal, and during the following fifteen years it traversed nearly the whole of the known world. It prevailed in different parts of the American continent for the first time in 1832, and again in 1834. It began its march a second time from India in 1847, and again traversed the greater portion of the globe, prevailing in the United States in 1849, '50, '51, and '52. Beginning again its march over the globe in 1864, it reached this country in 1866, and during this and the following year it prevailed in many of the large towns in the different States of the Union. It may be proper to add that my opportunities for observing the disease in 1849 and 1852 were ample. During these years I treated, in hospitals and in private practice, about 200 cases, recording at the bedside the histories in about 90 cases. A few cases came under my observation in 1866 and 1867. The disease prevailed in different parts of this country in 1873.[1] In 1883 cholera appeared in Egypt, and since then it has prevailed in Italy, Spain, and the southern part of France.

ANATOMICAL CHARACTERS.—With the exception of the presence in the intestines of specific organisms, there is nothing in the pathological anatomy of Asiatic cholera which, so far as at present known, can be considered as pathognomonic of the disease. The same changes may be met with in severe cases of sporadic cholera. The anatomical appearances vary according as death takes place during the algid stage or during the stage of reaction. The changes of the algid or asphyctic stage will be first described. Post-mortem rigidity is marked; it comes on soon after death and continues for a long time. In some cases shortly after death visible twitchings of the muscles occur, and occasionally considerable movements of the limbs. The heat of the body is preserved for a long period after death, and there may be even a post-mortem elevation of the temperature. The surface of the body has a cyanotic hue, especially in the dependent parts and in the extremities. The serous surface of the small intestine has a rosy color. The intestine is not distended with gas, but in typical cases it contains a large (sometimes an enormous) quantity of yellowish-white fluid, holding in suspension small white flakes, and hence presents a heavy, flabby, sodden appearance. The mucous surface of the small intestine is swollen, œdematous, moderately congested, and may contain ecchymoses. The congestion and the extravasations are most marked in the lower part of the ileum, and usually are not present in either the large intestine or the stomach. The solitary follicles and Peyer's patches are, as a rule, considerably swollen, and are frequently surrounded by a zone of congestion. The mesenteric glands are moderately enlarged. The surface of the mucous membrane is coated with tenacious mucus. There are marked inflammatory changes in the intestinal coats, indicated by an increased number of lymphoid cells in the mucous and submucous coats. After death the epithelial cells lining the mucous membrane are extensively desquamated, and may be found in the intestinal contents. The view has prevailed that this desquamation occurs during life and constitutes a lesion characteristic of cholera. Competent observers have failed to find intestinal epithelium in any considerable quantity in the discharges during life. There is no ground for assuming that during life an extensive desquamation of the epithelium takes place.

In the intestinal contents and in the walls of the intestine are found comma-shaped bacilli, which were discovered by Koch, and which will be considered fully under the head of Causation. The comma bacilli are usually abundant in the intestinal contents. They are found in the glands of

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[1] For an account of the cholera epidemic in 1873, vide *Reports prepared under the Direction of the Surgeon-General of the Army*, 1875, published by the Government of the United States.

Lieberkühn, between the epithelium of these glands and the membrana propria, and to some extent in the tissue outside of the glands. They are not present in the blood or in other organs of the body.

Changes in other parts of the body are in great part explicable by the enormous transudation of fluid from the blood into the intestines. The blood is dark, and from loss of water is so thick and viscid that it circulates with difficulty during life. It is accumulated chiefly in the large veins and in the right cavities of the heart, where it may be fluid, or it may contain soft, dark, or in some cases decolorized clots. The heart, especially its left ventricle, is generally firmly contracted. By way of compensation for its loss of serum, the blood absorbs fluid from the tissues, and hence these are found abnormally dry. The muscles are firm, dry, and dark-red in color. In consequence of the concentration of the fluid in the serous cavities, the pleura, pericardium, and peritoneum are often covered with a thin layer of sticky substance which has been sometimes mistaken for an inflammatory exudation. The meningeal vessels of the brain and cord are often congested. The cerebro-spinal fluid is diminished, and the substance of the brain and cord is firm and dry. The lungs collapse to an unusual degree in consequence of the empty and dry state of the bronchi. The upper portion of the lungs is usually dry and light colored, and the lower portion is frequently engorged with blood. There are sometimes hemorrhages in the lungs. Small ecchymoses are usually present in the tissue of the pleural and of the pericardial membranes. The spleen may be small, flabby, and dark red, or it may be swollen. There is nothing noticeable in the liver. The kidneys in this stage are not usually swollen. The veins may be somewhat distended, and ecchymoses are often found. Yellowish-white patches in the cortex of the kidney, indicating fatty degeneration of the epithelium, appear in the asphyctic stage, but they are more abundant later. The mucous membrane of the pelvis and calyces is hyperæmic and ecchymosed. The bladder is contracted and nearly or quite empty.

The composition of the blood in cholera patients has been studied with great care by C. Schmidt. He found that the degree of concentration of the blood generally reached its maximum in thirty-six hours after the onset of the choleraic discharges, at which time the relative proportion of solid constituents may be nearly one and a half times greater than normal. This increase pertains mainly to the organic matters in the blood. As it is chiefly the chloride of sodium that escapes in the transudation, the relative quantity of phosphates and of potash salts in the blood is increased. The functions of the kidneys being suspended, there is an accumulation of urea and of extractive matters in the blood. The percentage of red and white corpuscles in a given quantity of blood is increased. The red corpuscles are shrunken. The white corpuscles are often found in clumps in the blood after death.

In the stage of reaction, or typhoid stage, the morbid changes are notably different. The tissues no longer are abnormally dry. The intestinal contents do not present the rice-water character, but are stained with bile and sometimes with blood. They usually have a pasty consistence. The solitary follicles and Peyer's patches may be pigmented. The congestion may have disappeared, or in its place there may have developed a more intense inflammation than was present in the first stage. Follicular ulcers are not infrequent. Changes similar to those in dysentery, with diphtheritic inflammation and necrosis, occasionally make their appearance in this stage in the large intestine. In the brain and cord there are often congestion and increase of the cerebro-spinal fluid. The lungs not infrequently are œdematous and hyperæmic. Pneumonia may develop in this stage.. The spleen may be normal in size or swollen. The most important changes relate to the kidneys. Following the anæmia of the first stage, parenchymatous and fatty degenerations of the renal epithelium ensue. Sometimes an acute diffuse nephritis develops. The kidneys are congested and swollen, especially in the cortical portion. Yellowish streaks, indicating fatty degeneration, appear in the swollen cortex, in which the normal markings are obscured. The dark-red color of the pyramids contrasts with the yellowish-white aspect of the cortex. The epithelium of the tubes in many places is desquamated and has undergone parenchymatous and fatty degeneration. Hyaline, granular, and fatty casts are found in the tubes. Parenchymatous degeneration of the liver and of other organs may be present in this stage.

CLINICAL HISTORY.—In the great majority of cases the disease is preceded by simple diarrhœa, the dejections being greater or less in number, copious, and painless. The duration of this preliminary diarrhœa varies in different cases from a few hours to several days. With this diarrhœa in some cases occasional vomiting occurs. Aside from these symptoms, nothing denotes a tendency to the disease. The system otherwise is but little or not at all disturbed, and it is often difficult to persuade the patient that the intestinal disorder is of any special importance. Of 45 cases in which the existence or otherwise of premonitory diarrhœa was noted in the histories which I have recorded, it existed in 34 cases and was wanting in 11.[1] Statistics on a larger scale show a much smaller proportion of cases in which the premonitory diarrhœa is wanting. Thus the report of the General Board of Health of London for 1848–49 contains the statement by Dr. MacLoughlin, one of the sanitary inspectors, that of 3902 cases the premonitory diarrhœa was not wanting in a single case. Of 142 cases treated in hospital in Paris by Michel Levy, the premonitory diarrhœa was wanting in only 6. Of 274 cholera cases treated in the different Parisian hospitals in 1853, the existence of premonitory diarrhœa was ascertained in 140, in the remainder of the cases it being absent or its existence not ascertained. By some the period during which the premonitory diarrhœa exists is reckoned as a stage of the disease. This is obviously improper, as there is nothing then distinctive of epidemic.cholera, and during the prevalence of the epidemic very many have the same kind of diarrhœa without the development of cholera. The simple diarrhœa which prevails extensively during the prevalence of cholera is called, in France, *cholerine*—a term adopted to a considerable extent in other countries. By German writers, however, this term is used to designate a mild form of cholera. In a practical view, the importance of taking cognizance of the premonitory diarrhœa is very great, as will presently appear.

The development of cholera is generally first denoted by characters relating to the intestinal evacuations. If diarrhœa have existed, the discharges are suddenly increased in quantity, or sudden and copious liquid discharges without previous diarrhœa mark the onset of the disease. This constitutes the attack, and in the majority of cases it occurs during the night. The characteristic stools consist of a thin liquid generally known as the rice-water or choleraic discharge. The liquid is generally yellowish-white and slightly opalescent. It contains in greater or less abundance small, white, solid particles, like grains of rice in rice-water or flakes of curd in whey. It is devoid of bile, but may be somewhat blood-stained. There is no distinctive odor. The reaction of the fluid is neutral or slightly alkaline. It contains a remarkably small quantity of solid constituents. These do not usually exceed 1 to 2 per cent. The specific gravity varies between 1006 and 1013. The chief solid ingredient is chloride of sodium. There is also present some carbonate of ammonium, which gives the alkaline reaction, and a very small quantity of albumen. The particles resembling rice-grains are composed of mucus with granular matter and some pus-cells. Occasionally a few cylindrical epithelial cells may be found, but they are not abundant. Various low vegetable organisms are present in large number. Of these the only ones of importance are the comma bacilli already mentioned, which, as will be shown under the head of Causation, are doubtless the specific cause of the disease. The quantity expelled at each act or in the aggregate is very variable. A large quantity, enough to nearly fill an ordinary chamber-pot, sometimes escapes at a single dejection, or the quantity with each act may be small. The quantity is not always in proportion to other symptoms denoting gravity. The dejections are not accompanied by pain, but by a sensation of relief. There is no smarting at the anus. The patient is suddenly impelled by a sense of distension to evacuate the bowels, and when the quantity is large it escapes very rapidly. In some cases the evacuations, after several have occurred, cease; and in other cases after a time the liquid flows away constantly, the patient being unable to prevent it. The characteristic dejections are sometimes wanting. The disease has been called *cholera sicca* when death takes place without choleraic discharges. The name, however, is inappropriate. In the so-called cholera sicca the rice-water-like liquid is present in the alimentary canal, and the quantity found after death may be unusually large. It is conjectured that in these cases the peristaltic movements of the intestine are paralyzed, so that the intestinal contents are not expelled. These cases terminate fatally with great rapidity.

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[1] In connection with the history of a cholera epidemic in Buffalo in 1849, by the author, was published a tabular abstract of the histories of 33 not fatal and of 34 fatal cases. (Vide *Buffalo Medical Journal*, 1850, vol. v.)

Vomiting usually occurs. In a diagnostic and pathological view it is less important as a symptom than the characteristic diarrhœal discharges. In some cases it is a prominent and persistent symptom, everything taken into the stomach being rejected; but in other cases it occurs occasionally, and sometimes it occurs at the onset and ceases. The matter vomited is a watery liquid, frequently consisting chiefly of water which has been drunk, but sometimes it is evidently a transuded liquid. The quantity may exceed that of the liquids ingested. The vomited liquid is usually neutral, but it is sometimes acid. The vomiting is not preceded or accompanied by much nausea. The act of vomiting occurs suddenly, and appears to arise simply from distension of the stomach.

These symptoms, conjoined with a sense of prostration, more or less feebleness, and generally frequency of the pulse, coolness of the skin, or perspiration, with, in some cases, cramps of the muscles of the limbs, may constitute the clinical history of the disease. In other words, the disease may either end spontaneously or be arrested without symptoms other than those just described. The vomiting and purging cease, improvement as regards the other symptoms follows, convalescence is at once declared, and recovery is rapid. The duration of the disease in these cases is brief—a few hours only. In a large proportion of cases, however, the disease does not pursue such a favorable course. A group of striking symptoms becomes developed, and the disease passes into what has been called the algid or cyanosed stage, or more commonly the stage of collapse. It remains to sketch the further clinical history in these cases.

Important symptoms pertain to the circulation. The pulse is usually frequent and proportionately feeble, beating 120, 130, or 140 per minute, but in some cases it becomes more and more feeble without acceleration. I have observed it to fall below the normal frequency. When the state of collapse is fully developed the pulse at the wrist is extinct. Feebleness or absence of the apex-beat of the heart and of the heart-sounds also shows greatly diminished power of this organ. The blood stagnates in the veins, giving rise to lividity at the roots of the nails and a dusky hue of the prolabia, face, and surface of the body generally. Hence the significance of *cholera cyanosis* and *cholera asphyxia* as names of this stage of the disease. If venesection be practised, the blood trickles from the aperture, not flowing in a projected or continuous stream. Leech-bites do not bleed as usual. The blood is manifestly thicker than in health.

The respiratory system presents striking symptoms. The number of respirations is usually increased, but is sometimes diminished. Dyspnœa is occasionally complained of, and sometimes exists in a distressing degree, owing to the feeble circulation, from the thickened state of the blood. The respiration is frequently suspirious or sighing, and irregular in rhythm. The expired air, when the collapse is complete, is low in temperature as compared with health (76° or 80° F.), giving to the hand a sensation of coolness. It contains more oxygen and less carbonic acid than in health, showing a notable deficiency of the changes incident to the function of respiration. The voice undergoes a marked change, becoming feeble, raised in pitch, husky, and not infrequently extinct, attributable in part to muscular debility, but chiefly to dryness and stiffness of the vocal cords. In the moribund state the tracheal râles which so often precede death from other diseases are wanting.

Very little pain is experienced except from muscular contractions or cramps. As regards the nervous system, the disease is characterized by mental indifference or apathy. The patient has no apprehensions, although prior to the attack there may have been intense dread of the disease. In some cases there is great restlessness and jactitation, and in other cases the patient lies quietly, save when disturbed by evacuations and cramps. The latter give rise to most of the suffering. Cramps are usually but not invariably present. They are seated especially in the feet, calves, and abdominal walls, the muscles of the upper extremities and even of the face being somewhat affected. The prominence of this symptom renders the name *spasmodic cholera* significant. The muscular strength in some cases is retained in an extraordinary degree. Patients if not prevented will sometimes get up and walk about even a few moments before death.

The functions of most of the secretory organs are nearly or quite suspended. No bile is contained in the matter vomited or in the dejections, and the urine is suppressed. Patients do not weep. It is a remarkable fact, however, that in nursing women the lacteal secretion may continue. The disease also does not prevent the occurrence of menstruation, but the menstrual discharge is diminished. The skin, in addition to its cyanosed appearance, is cold and often covered with a copious, viscid perspiration ; but in other cases it is cold and dry. The coldness of the surface is like that of a cadaver. Of this, however, the patient is not conscious ; on the contrary, there is generally a sense of heat and a desire to be uncovered. The heat of the body is diminished, as shown by the thermometer in the rectum or vagina. The axilla in this disease does not give a fair representation of the internal temperature. The diminution ranges generally from two to four degrees below 98°, but it may fall to 90°, and even considerably lower, and it has been known to fall to 73°. The skin is frequently shrivelled and wrinkled like the arms of washerwomen. Its elasticity is diminished, as shown by its subsiding very slowly after being pinched up. The tongue gives to the touch a sensation of coldness. Thirst is a prominent symptom. The patient craves constantly cold drinks, and iced water or ice is taken with great avidity.

The general aspect in the collapsed state is highly characteristic. The features are contracted, the eyes sunken ; the whole body is diminished in bulk ; the patient appears to have suddenly grown old, and the countenance is often so much changed that the person is scarcely recognizable by familiar friends.

Of the foregoing symptoms, most are present in all cases in which the disease is not speedily arrested. Differences relate chiefly to the degree of severity of the disease. In stating the condition of patients it is customary to say *before* or *after* collapse, and to express different degrees of the collapsed state by saying *semi-collapsed* or *completely collapsed.* Collapse is complete when the pulse at the wrist is extinct, the surface cold, etc. The collapse may ensue with more or less rapidity—from an hour to six or eight hours after choleraic dejections occur. It occurs almost invariably in fatal cases, the mode of dying being of course by asthenia; but I have known cases to end fatally without the stage of collapse, the patient becoming comatose and dying by apnœa.

The patient after emerging from the state of collapse does not, in general, pass at once into convalescence, but there follows a period of the disease commonly called the *stage of reaction.* In this stage there is usually fever, the temperature rising more or less above the maximum of health. Diarrhœa not infrequently continues, the dejections becoming green and gelatinous. Dysentery is sometimes a sequel. In some cases constipation more or less obstinate follows. Vomiting in some cases is a troublesome symptom in this stage, the vomited matter being either green or yellow. Capillary congestion of the surface, especially of the face, is frequently marked, as in cases of typhus. The renal secretion returns, and the urine is albuminous, brownish-red in color, and it contains epithelial, fatty, and hyaline casts, together with degenerated renal cells, and usually also red and white blood-corpuscles. This stage is not devoid of considerable danger. The patient may be worn out with continued vomiting or diarrhœa; somnolency and coma become developed in a certain proportion of cases, dependent probably on uræmia; and delirium of a passive kind is not uncommon, together with subsultus, sordes, and other symptoms of the typhoid state. Pneumonia is sometimes developed. A fatal termination occurs in a pretty large proportion of the cases in which the stage of reaction takes place; and in the cases which recover the powers of the system are generally impaired for a long period. Exceptionally, patients emerge from the collapsed state and pass either gradually or quickly into convalescence without febrile movement or other symptoms belonging to the stage of reaction.

Epidemic cholera has a brief duration. After the occurrence of the characteristic choleraic dejections death sometimes takes place within two or three hours, the patient dying as if from profuse hemorrhage. The duration rarely extends beyond ten or twelve hours. If life continue longer and either death or recovery ensue, the disease has run its course, and the symptoms denote its sequels or effects. Assuming the disease to be parasitic, the local conditions for the multiplication of the parasites continue only for the limited period of the duration of the disease proper.

PATHOLOGICAL CHARACTER.—Most of the striking phenomena of the disease are plainly attributable to the blood-lesions resulting from the loss of the constituents found in the choleraic dejections. The blood circulates with difficulty, and the changes incident to respiration take place imperfectly; hence the feebleness and extinction of the pulse, the cyanosis, loss of temperature, thirst, mental apathy, spasm, unchanged expired air, defective secretion, etc. etc. The retention of excrementitious principles in the blood doubtless plays an important part in the production of morbid phenomena. The coma which occurs in some cases may be due to uræmia. Cholesteræmia may occur, since the function of the liver is suspended.

The transudation, however, which is first in the catenation of appreciable events involves an underlying pathological condition. That this condition is due to the invasion and growth of the cholera bacilli to be presently described is very probable. Koch is of the opinion that these bacilli produce some poison which is absorbed into the system and causes the severe constitutional symptoms and the lesions of organs other than the intestine.

CAUSATION.—Since the preceding edition of this work an important addition to our knowledge of epidemic cholera has been made by the discovery by Robert Koch of a form of bacterium peculiar to this disease and to be regarded as its specific cause. Koch was sent by the German government as the head of a commission to investigate epidemic cholera in Egypt, and subsequently in the East Indies. He discovered the cholera bacillus in Egypt in 1883, and in the latter part of that year and in the early part of 1884 he continued his studies of this organism in India.

The cholera bacillus is shorter, but somewhat thicker, than the tubercle bacillus. It is more or less curved, and from its resemblance in shape to a comma it is called by its discoverer comma bacillus. The curve is not always that of a comma, nor is one end usually thickened, like the upper end of a comma. Sometimes, especially in cultures, the organism presents a spiral or corkscrew shape. These spirals represent either a series of comma bacilli joined together or a spiral growth of a single organism. From this appearance it is claimed that the organism should be called a spirillum or a vibrio, and not a bacillus; but there is some doubt as to the interpretation of the spiral mode of growth. The cholera bacilli multiply by transverse fission.

Although, to a trained bacteriologist, the morphological appearances of the cholera bacillus are quite characteristic, nevertheless, for certainty in diagnosis, it is necessary to resort to artificial cultures of the organism. The cholera bacillus grows readily in a great variety of media, of which the most important are bouillon, nutrient gelatin, agar-agar, blood-serum, potato, and milk. It grows best at a temperature of between 30° and 40° C. (86°–104° F.). It can be cultivated at 17° C. (62½° F.), but its growth nearly ceases below 16° C. The most useful agent for the study of the diagnostic characters of the growth of the cholera bacillus is nutrient gelatin in plate and in test-tube cultures. These characters are manifest within twenty-four hours after inoculating the gelatin and cultivating at a temperature of about 21° C. (70° F.). In the gelatin cultures on plates a typical colony of the cholera bacillus appears as a small glistening, round particle, which, when examined with a low magnifying power, presents a granular, grayish appearance with well-defined but slightly irregular borders. Each colony is surrounded by a narrow clear ring caused by liquefaction of the gelatin. In test-tube cultures the growth of the colony gradually extends from the surface down and along the line produced by sticking the platinum wire into the gelatin when inoculating the tube. Corresponding to the top of this line, the gelatin appears to be occupied by a round or conical air-bubble, from which the tapering colony extends in the line of inoculation. There is a moderate liquefaction of the gelatin around the colony. Upon sterilized steamed potato at ordinary temperatures the cholera bacillus grows very imperfectly, but between 30° and 40° C. it grows very rapidly. When cultivated and examined in a drop of bouillon the cholera bacilli are found to be endowed with the power of rapid movement.

The form and the development of the cholera bacillus, together with the characters and the conditions of its growth in various culture media, suffice to distinguish this organism from all other forms of bacteria with which we are acquainted. Many other comma-shaped bacteria are known to exist, but all of them, such as the comma bacteria of Finkler and Prior (p. 537), those found sometimes in the saliva, those discovered in cheese, in foul water, etc., can be positively distinguished from the cholera bacilli. The cholera bacilli are peculiar to epidemic cholera, and hitherto they have been found only in the intestines and the dejecta of patients affected with this disease.[1] Moreover, the cholera bacilli are invariably present in the intestinal contents and evacuations of cholera patients, except in a late stage of the disease when the patient is suffering from the sequels rather than from the immediate effects of cholera. It is certain, therefore, that the cholera bacilli and the disease cholera bear a close relation to each other, and it is difficult to conceive that this relation can be any other than that the bacilli are the cause of Asiatic cholera. Inoculation of the cholera bacilli in animals has not given clear and satisfactory results, as animals under normal conditions do not seem to be susceptible to Asiatic cholera. Still, it has been found possible, after neutralizing the acidity of the gastric juice and after paralyzing intestinal peristole by opium, to obtain in animals, by the injection of cultures of cholera bacilli into the stomach, lesions of the intestine similar to those in cholera. The intestines are congested and filled with fluid or semifluid contents, which contain cholera bacilli in enormous numbers. Satisfactory inoculation experiments on animals, welcome as they would be, are hardly necessary to prove the causative agency of the cholera bacilli, as the observations on human beings can hardly admit of any other interpretation than that which has been given. In every case where it has been possible to fulfil all the conditions of a strict proof it has been found that peculiar organisms, whenever constantly associated with the lesions of a given disease, are the specific cause of that disease; and in no instance has support been found for the hypothesis that organisms not found elsewhere and constantly associated with a disease are present simply because the disease makes a favorable soil for their growth. If, then, the cholera bacilli are the specific cause of epidemic cholera, it is important to know all the conditions which are favorable, as well as those which are hostile, to the growth and development of these organisms. Our knowledge of these conditions is as yet imperfect. It has already been mentioned that growth of the cholera bacilli is checked by a temperature much below 16° C. (61° F.). The bacilli, however, are not destroyed by a temperature far below the freezing-point. They do not form spores,[2] and are therefore much less resistant to injurious influences than the spore-forming bacilli, such as those of anthrax. Cholera bacilli in the moist condition, however, may preserve their vitality for five or six months, and after special modes of cultivation for even a year or more. When completely dried the cholera bacilli die within a short time. They are very susceptible to the action of most acids, which even in very dilute solution destroy the bacilli or check their growth. They are destroyed by carbolic acid in one-half of 1 per cent. solution. This agent is recommended by Koch as the most suitable for the disinfection of cholera stools and linen. The cholera bacilli grow rapidly upon linen soiled with cholera dejecta, so that after twenty-four hours this linen seems to present a pure culture of these organisms. After a day or two the bacteria of putrefaction predominate and displace the comma bacilli. The cholera bacilli do not develop in very foul and putrefying substances. The cholera bacilli preserve their vitality for a considerable time in water, but they do not develop in water unless this contain a certain (not too small) proportion of nutritive material.

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[1] Koch found cholera bacilli in the water of a tank in India which was used for drinking purposes, and which was contaminated with the dejecta and the linen of cholera patients.

[2] Hüppe and others describe a fragmentation of cholera bacilli into little round bodies, which they claim are more resistant than the bacilli and are capable of developing into bacilli. These round bodies they therefore regard as spores (arthrospores).

The question now arises, How far is it possible to reconcile the doctrine of a special contagium in cholera stools and the known facts concerning this contagium with the views derived from a careful study of epidemics of Asiatic cholera? The epidemiological study of cholera has led to a wide although not universal acceptance of the view that Asiatic cholera is not directly contagious. The following are the main considerations upon which is based the opposition to the doctrine of contagion:

1. Of those who are brought into contact with or close proximity to cholera patients the proportion who become affected is not larger than that of those who are not thus exposed. It is a notorious fact that physicians and nurses in attendance upon cholera patients do not contract the disease more frequently than others living in the same locality. In this respect there is a marked contrast between the development of cholera and of typhus fever in hospitals.

2. Although there is no ground for the assumption that an epidemic of cholera can ever originate in a place in which the cholera germs have not been introduced either by persons or by clothing or other substances derived from a cholera locality, nevertheless, imported cases of cholera in situations where the disease had not previously existed very frequently, and perhaps as a rule, do not lead to its diffusion.

3. The epidemic breaks out in a place almost simultaneously in different situations, without any communication of the cases with each other.

4. It is diffused too rapidly to be propagated by contagion. If contagious, it must be most highly so to account for the rapidity with which it extends; and the latter assumption is not consistent with other facts.

5. There are certain places which possess temporary or permanent immunity against the invasion of Asiatic cholera. Lyons in France is such a place. From its well-known immunity Lyons has become a refuge for persons fleeing from cholera-infected localities. Although such persons are readily admitted into the city, there has never been an epidemic of cholera in this place except in a very limited district. Many places in which there is immunity from cholera are known to exist.

These considerations lead to the conclusion that the spread of cholera is not due to its being directly communicated from those affected with the disease. To explain the epidemiological facts concerning cholera the hypothesis has been advanced, and has met with wide acceptance, that the stools of cholera patients contain a virus which is not capable of producing cholera at once, but must first undergo, outside of the body and probably in the soil, certain unknown modifications or metamorphoses. Cholera has therefore been classified among the miasmatic contagious diseases. The conditions of the soil favorable for the development of epidemic cholera have been investigated most carefully by Pettenkofer. These conditions are—first, a certain degree of porosity of the ground; second, a certain amount of moisture in the superficial layers of the ground, and this amount must be neither too great nor too small, and in most places is indicated by a sinking of the ground-water; and third, a certain amount of organic matter suitable for the growth of the cholera germs in the soil. Such matter is present particularly in soil contaminated by filth or imperfect drainage. It is apparent that these conditions of soil may in certain regions be permanently absent, and in other regions be temporarily absent. Hence is explained the permanent and the temporary immunity of certain places against the invasion of cholera.

The facts which have been mentioned in opposition to the contagion doctrine of cholera and in support of the essential influence of local conditions in the development of the disease have led Pettenkofer and his followers to reject the view that the comma bacilli found in cholera stools are the special cause of the disease. This view, they say, cannot be reconciled with epidemiological facts.

In reply it may be said that the constant presence of cholera bacilli in the intestinal contents of cholera patients is an established fact, and requires an explanation. No satisfactory explanation has been offered or seems possible other than that the bacilli are the cause of the disease. In other words, the dejecta of cholera patients do contain a special poison capable of causing cholera without undergoing any special metamorphosis in the soil. Positive facts speak for the existence of this contagium in the stools, such as the repeated instances in which washerwomen have contracted cholera by washing clothing or bed-linen which has been soiled by the dejecta of cholera patients. Macnamara relates the following well-authenticated case in his own experience in the East Indies:[1] "A small quantity of a fresh rice-water stool passed by a patient suffering from cholera was accidentally mixed with some four or five gallons of water, and the mixture exposed to the rays of the tropical sun for twelve hours. Early the following morning nineteen people each swallowed about an ounce of this contaminated water: they only partook of it once, but within thirty-six hours five of these nineteen persons were seized with cholera. In this instance the choleraic evacuation did not touch the soil; as it was passed, so was it swallowed." Cholera was not prevailing to any considerable extent in the locality at the time. While, then, these and other observations support the view that the cholera stools contain the special agent of infection of the disease, it is to be noted that this infectious agent must be received into the alimentary canal in order to produce cholera. Under ordinary circumstances it is apparent that portions of cholera stools could gain access to the digestive tract only through great carelessness. We know that bacteria are not conveyed into the air from moist surfaces, and, as has already been mentioned, the cholera bacilli are destroyed by being completely dried. The exhalations and the urine of cholera patients do not contain cholera bacilli. These are present only in the discharges from the intestinal canal, and very exceptionally in the vomit. It is explicable, therefore, that with ordinary care those who are in close proximity to cholera patients are not likely to contract the disease merely from such proximity.

We come, therefore, to the conclusion that an epidemic of Asiatic cholera is due to an infection of the soil or of the drinking-water of the locality by cholera bacilli derived directly or indirectly from the dejecta of cholera patients. That the contamination of the drinking-water with cholera germs may cause an outbreak of epidemic cholera seems certain; but it is not proven that this is a frequent mode of infection. Probably a common source of infection is direct contact with objects which contain cholera bacilli, such as might come from eating uncooked fruit, salads, etc. As to the exact mode of infection we are in most cases ignorant; but from what has been said it is apparent that the known facts concerning the origin and development of epidemics of cholera are not opposed to the acceptance of Koch's discovery of the cholera bacilli and of their causative relation to the disease.

Great importance is to be attached to the auxiliary or predisposing causes of Asiatic cholera, as it is within our power to prevent many of these. Unfavorable hygienic conditions, especially such as affect the purity of the drinking-water and of the soil, are most potent factors in aiding the development of cholera. By improvement in sanitary conditions places have been rendered nearly or quite exempt from the invasion of cholera, even in India, the home of the disease. Individual predisposition also plays an important rôle. The cholera bacilli are destroyed by acids, even by the acid gastric juice. Disorders of digestion, therefore, which reduce the acidity of the gastric juice, facilitate the entrance and development of the cholera bacilli in the intestine. Hence deprivations, intemperance, depressing emotions, over-exertion, fear of the disease, are auxiliary causes in certain cases.

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[1] Quain's *Dictionary of Medicine*, article "Cholera."

Cholera has prevailed in all climates and at all seasons. In temperate climates it has prevailed most frequently in late summer and in the autumn. Feeble persons are more apt to be attacked than the robust. As regards age, no period of life is exempt, but the largest proportion of fatal cases is among persons advanced in life. Excluding old persons, the mortality is largest after adult age, next in early infancy, and last in childhood and youth. A certain degree of immunity, at least for a few years, is afforded by having experienced an attack of cholera.

DIAGNOSIS.—During the prevalence of cholera there is no difficulty in recognizing the disease when fully developed; that is, when symptoms denoting existing or impending collapse are present. There is scarcely any other disease in which the diagnostic features are so strongly marked; but it is exceedingly important to recognize it before it has advanced sufficiently to manifest its most obvious striking characters. The diagnosis is then to be based mainly on the characteristic intestinal evacuations. As soon as the so-called rice-water or choleraic stools are apparent the patient is to be considered as attacked with cholera. In a very small proportion of cases these evacuations do not occur at all, or not until other symptoms characteristic of the disease become developed. The diagnosis in these cases must be based upon the latter.

It is important to recognize cases of cholera which appear in places where no epidemic prevails. Prophylactic measures to be effectual must be applied to the first cases of cholera which appear in a community. Hitherto it has been by no means easy to distinguish the first cases of cholera, especially if they do not terminate fatally, from sporadic cholera or cholera nostras. We possess, however, in the recognition of Koch's comma bacillus a means of certain diagnosis. In general, from twenty-four to forty-eight hours will suffice to establish the diagnosis. If it happen that the cholera bacilli are found in almost pure cultures upon the surface of the cholera dejecta or upon the linen soiled thereby, the diagnosis is established. Usually it is necessary not only to make stained microscopical preparations from the stools, but also to prepare cultures; which is done by first making a plate culture in nutrient gelatin. From the characteristic colonies which develop in the gelatin pure cultures may be made in test-tubes containing nutrient gelatin, and specimens can be examined in a drop of bouillon in order to study the properties of the living bacilli. At present, undoubtedly few practising physicians possess the knowledge requisite for the preparation of such cultures. This knowledge, however, is not difficult to acquire, and at least in every large community some one should be found who is capable of making the diagnostic tests which have been mentioned.

Cholera is to be discriminated from the simple diarrhœa which generally precedes it, and which during the continuance of an epidemic affects a large number of persons without eventuating in cholera. It is probable that this premonitory diarrhœa proceeds from the epidemic cause, but cases are not to be considered as cases of cholera when neither the choleraic discharges nor other characteristic symptoms occur. This discrimination is important with reference to determining the rate of mortality and the value of therapeutical measures. The apparent success attending the practice of some physicians or following the employment of certain methods of treatment may depend on cases of simple diarrhœa being reckoned as cases of cholera.

This disease is one of the most insidious in its approaches. Aside from a mild diarrhœa it gives no warning of the attack. So little are patients conscious of danger that they often apologize for giving the physician the trouble of visiting them, when a glance suffices to show that they are on the verge of collapse. It behooves the physician in all cases of simple diarrhœa during an epidemic of cholera to carefully watch the evacuations with reference to the diagnosis. On the other hand, during an epidemic physicians are not infrequently called to see persons who under nervous excitement imagine that they are about to be attacked, when no symptoms of the disease are present. These have been appropriately called cases of *choleraphobia*.

PROGNOSIS.—Of those attacked with cholera, a large proportion die. The average rate of mortality in hospitals varies from one-half to one-third. When developed in hospital wards among patients admitted for other diseases, it proves fatal in the great majority of cases. In private practice, especially among the better classes of society, the mortality is considerably less. In individual cases the prognosis is widely different according to the period of the disease at which the patient is first seen. If the patient be seen immediately after the attack, before serious blood-lesions have occurred, the prospect of an arrest of the disease is good; but if the disease have advanced to the stage of collapse the prognosis is always exceedingly unfavorable. After reaction from the collapsed state the danger is still great, but the cases in which recovery takes place preponderate. The statistical researches of Duchesne show the rate of mortality at different periods of life in the epidemic at Paris in 1849.[1] The proportion in 1000 in subjects less than five years of age was 148; from five to fifteen years, 50; from fifteen to thirty years, 177; from thirty to forty-five years, 254; from forty-five to sixty years, 206; and from sixty to eighty-five years, 162. The mode of dying is in general typical of asthenia. Accumulation of liquid in the air-tubes very rarely takes place.

PREVENTION OF CHOLERA.—The prophylaxis of cholera claims consideration under a distinct heading, and I give it precedence over the therapeutical management, in view of its greater relative importance. In addition to the removal, as far as possible, of all the auxiliary causes of disease which contribute to render the special cause of cholera efficient, the prevention involves prompt attention to the diarrhœa which in the great majority of cases precedes the attack. This premonitory diarrhœa is amenable to simple measures of treatment, and if effectually treated there is reason to believe the supervention of cholera is prevented. All physicians who have had much practical acquaintance with this disease will bear testimony—*first*, to the fact that an attack of cholera is generally preceded by diarrhœa; and *second*, to the fact that an attack very rarely occurs when this diarrhœa receives appropriate attention. Giving results of my own experience with respect to the facts just stated, in 1849 for the space of three months I prescribed for as many private patients with the premonitory diarrhœa as my physical endurance would permit, my practice being chiefly among the prudent classes, and I had during that epidemic but 10 cases of cholera in private practice. In only one of these 10 cases did I prescribe for any premonitions, and in the single case the prescription was for a slight nausea only. In each of the 10 cases either the premonitory diarrhœa did not exist or it was neglected. During another epidemic in 1852, I had about the same number of cases of cholera in private practice. In not a single case had I been called upon to prescribe for premonitory diarrhœa, and I prescribed for hundreds of persons with simple diarrhœa, not one of whom had an attack of cholera. The experience of others would doubtless furnish in like manner evidence of the above stated facts, and from these facts the following conclusion may be drawn: Except the very small proportion of cases in which cholera is not preceded by diarrhœa it may with almost absolute certainty be prevented. It needs but a little reflection to see the immense practical importance of this conclusion.

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[1] Valleix, *op. cit.*

The treatment of diarrhœa during a cholera epidemic is simple. An opiate conjoined with an astringent remedy, regulated diet, rest, and recumbency suffice. A few drops of laudanum with camphor, or the camphorated tincture of opium in conjunction with the tincture of kino, catechu, or krameria, a salt of morphia or opium with either tannic acid or the acetate of lead or bismuth and capsicum, may be given in doses sufficient to arrest the diarrhœa at once, and repeated often enough to prevent its recurrence. Inasmuch as the bacilli enter the body probably by way of the digestive system, the free use of the mineral acids may be useful as a prophylactic measure, these being found to be destructive to the parasite out of the body.

The great difficulty as regards the prophylaxis is in the prompt application of simple but effectual treatment in all cases of diarrhœa during the epidemic. Very many pay no attention to the premonitory diarrhœa through ignorance, and not a few of those better informed neglect it either from recklessness or because they cannot appreciate the fact that a disorder so slight and ordinary can be a precursor of a malady of such gravity as cholera. Public announcements by handbills, articles in newspapers, etc. of the importance of promptly resorting to treatment for diarrhœa fail in accomplishing the object.. The only effectual plan is to organize a sanitary police, and to provide for one or two domiciliary visits daily at every house within the limits of the epidemic, the purpose of the visits being to inquire if any one be affected with diarrhœa, to impress the importance of immediate attention to it, and when circumstances render it necessary to supply appropriate remedies at once. This plan, faithfully carried out, demonstrates that cholera is in a great measure preventable. The prevention is effected by arresting at once diarrhœa as often and as soon as it occurs. Abundant proof of the successful operation of the system of the house-to-house visitation was furnished by its practical results in London and other towns in Great Britain during the epidemic of 1849.

Another mode of escaping the disease is to remove without the range of its prevalence. Persons not compelled to remain by necessity or by a sense of duty should go beyond the limits of the epidemic; and the removal of persons in districts where, owing to the activity of auxiliary causes, the disease is especially rife, should be enforced as a sanitary measure by municipal authority.

The foregoing remarks relate to the prevention of cases of cholera during an epidemic. To prevent the epidemic is an object of still greater importance. This is to be done by thorough sanitary measures in regard to the removal of filth, attention to sewers, cesspools, and privies, also to wells or other sources of water-supply, together with proper protection against causes of disease from waste-pipes in houses, by providing against overcrowding of tenements, etc., and by efficient quarantine regulations. Ships, merchandise, luggage, etc. coming by sea or land from a cholera region should be effectually disinfected. The clothing of persons from an infected place should be disinfected, but only those need be detained in quarantine who are suffering from cholera or diarrhœa. It is of the utmost importance to disinfect thoroughly the stools, the linen, and the bed-clothing of cholera patients—in fact, everything with which by any chance the discharges of the patient can come in contact. The most useful agent for disinfecting the stools is carbolic acid, which may be employed in a 5 per cent. solution. Sulphate of iron is not an effective disinfectant for the stools. By these means the introduction, or at least the spread, of cholera germs may be prevented. If these means do not prove protective, it is because they are not, and perhaps cannot be, employed with sufficient completeness; for with our present views of the causation of the disease it may be affirmed that if the cholera bacilli can be completely excluded cases of cholera cannot occur.

When the introduction of the disease is not prevented, the prevention of its diffusion is practicable by the prompt and effective disinfection of every house in which it occurs. In this way an epidemic is "stamped out." That this is practicable was demonstrated in the city of New York in 1866. A thousand cases occurred in that year in different situations widely separated from each other. Cases occurred in 362 houses, all of which were instantly disinfected and all local appreciable causes of disease were removed. In no instance did the disease extend beyond the house in which a case occurred. These facts are of momentous importance.[1]

TREATMENT.—To consider the host of remedies and of therapeutical measures which have been advocated as more or less efficacious in the treatment of this disease would require not a little space. There are but few articles in the materia medica which have not been tried, even including antimony and drastic purgatives. Much injury has doubtless been done by over-medication under the idea that the treatment, as regards activity, must be proportionate to the amount of danger from a disease. On the other hand, many of the remedies which have been employed exert little or no effect either for good or harm. It would be unprofitable to devote space to the consideration of the great variety of practice which the literature of cholera affords. Accepting the parasitic doctrine, a specific remedy must be a parasiticide, capable of securing by its toxical effect upon the parasite its destruction within the intestinal canal. As yet no such remedy is known; but there is reason to believe that the disease is frequently controlled by efficient treatment, and when not arrested the recovery may depend on the judicious employment of measures for that end. In the present state of our knowledge the objects of treatment are the prevention of the immediate serious consequences of the action of the parasite, and to obviate the tendency to death. I shall limit myself to a brief statement of the principles of treatment which my own experience and reflections have led me to regard as most consistent with our present knowledge.

The treatment is to be considered as applicable to the different stages—namely, before collapse, during the collapsed stage, and after reaction. Prior to collapse the paramount object is the arrest of the intestinal effusion. This effusion is the first appreciable link in the chain of morbid sequences, and, if it be promptly arrested before it has proceeded so far as to affect seriously the blood and circulation, the patient is usually safe. The remedy on which most dependence is to be placed in effecting this object is opium. Some form of opiate is to be given promptly in doses sufficient to effect the object. The form of opiate is to be chosen with reference to promptness of action and the probability of its being retained. Opium in substance is unsuitable, from the comparative slowness of its action. Laudanum or the acetated tincture or an aqueous preparation is to be preferred; but the article which I have been led to regard as the most eligible is a salt of morphia, administered by placing it dry upon the tongue. In the endeavor to effect the object of treatment in this stage moments are precious, for there is always danger that if the object be not promptly effected the patient will fall into the collapsed state. The opiate should therefore be given at once in a full dose. A grain of a salt of morphia is rarely if ever too large a dose for an adult. A physician should if possible remain with the patient. If the first dose be quickly rejected, a second should be instantly given. The remedy is to be repeated at intervals of from one-half to three-fourths of an hour until the dejections cease. If, owing to the occurrence of vomiting, the administration by the mouth be ineffectual, it should be given by the rectum, and in cases in which the symptoms are urgent both modes of administration should be resorted to. The system, even in this stage of the disease, is not readily affected by opiates thus given. In view of the importance of the object, if it be necessary in order to effect it some risk of inducing narcotism is justifiable; but if the administration be in the hands of the physician, and the effects of the doses watched with care, danger from this source may be avoided. The practical point is to employ the remedy promptly so as to effect the object, bearing in mind that the delay of half an hour or an hour is often fatal. Relying upon the opiate, it is best not to add other remedies, lest by increasing the bulk of the doses they be rejected. A full dose is preferable to small doses frequently repeated, because the effect within a short space of time is greater and the remedy is more likely to be retained. Aside from the rejection of the remedy, vomiting is, if possible, to be prevented in view of its perturbatory effects. The patient in this stage should be restricted to a very small quantity of water or spirit and water given at short intervals, or to small pieces of ice. Perfect quiet is important. He should not be permitted to get up to go to stool, and he should be urged to resist as much as possible the desire to evacuate the bowels. Frictions, the warm bath, sinapisms, etc. in this stage are useless and hurtful.

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[1] For further details and for the processes of disinfection, vide *Report of the Metropolitan Board of Health*, New York, 1867.

I have repeatedly succeeded in arresting the disease by this plan of treatment, and when it has been arrested before the stage of collapse the recovery is usually speedy. Regulated diet, rest, with perhaps a tonic remedy, suffice for the cure. The bowels should be allowed to remain constipated for several days, and then if movements do not spontaneously occur simple enemas will probably be sufficient. If not, a little rhubarb or some other mild laxative may be given. I believe no other plan of treatment promises more than this, but it is not to be expected that it will always prove successful. It will fail—or rather it is not available—when, owing to the persistent vomiting and frequent purging, the remedy is not retained sufficiently long to exert its effect; and it is not available when, owing to the great rapidity of the transudation, the state of collapse occurs so quickly that there is not time enough to obtain a remedial effect. These difficulties are equally in the way of success from any remedies.

The foregoing remarks on the treatment prior to the phenomena of collapse were written before the hypodermic method of administering opiates and other remedies had come into vogue, and the inquiry has arisen whether by means of this method the opiate plan of treatment of cholera in the first stage may not be carried out more efficiently than by giving opiates by the mouth or rectum. Considering the great importance of the promptness of the effect of opiates, and the uncertainty attending their administration by the mouth or rectum, owing to their rejection by vomiting or purging, and the difficulty of absorption if they be retained in the alimentary canal, the hypodermic method seems to offer a great advantage as regards speediness and reliability of effect. The hypodermic injection of Magendie's solution of morphia was employed in a large proportion of the cases which came under my observation in 1866. The vomiting, purging, and cramps were generally arrested promptly by this measure; recovery, however, followed in a very small proportion of cases. So far as my experience goes, it affords evidence of the efficiency of the measure with regard to the immediate objects—namely, the arrest of the vomiting, purging, and cramps—but it does not afford proof of its curative efficacy. With regard to the latter, the character of the patients is to be borne in mind, nearly all who were seen in the first stage being inmates of the hospitals, and many being affected, when attacked with cholera, with some serious chronic disease, such as phthisis, Bright's disease, etc. The hypodermic injection of a solution of morphia in this disease is to be employed with circumspection. Narcotism is sometimes induced by an amount much below that which might be given by the mouth without risk. I have known a little less than a grain, given in two injections with an interval of two hours, to produce deep narcotism. The tolerance of opiates in large doses by the stomach or rectum is not a criterion of the quantity to be injected beneath the skin in epidemic cholera.

Rectal injections of brandy and a strong tea-infusion were used considerably, with apparent efficacy, in arresting the evacuations and preventing collapse. The proportions used were half an ounce of brandy and two ounces of the tea-infusion, the injections being repeated every half hour, every hour, or after longer intervals according to circumstances.

In the stage of collapse the plan of treatment indicated prior to this stage may prove not only ineffectual, but hurtful. It is still an object to arrest intestinal transudation if it continue, but to employ opiates largely for this object is not judicious with reference to the recuperative efforts of the system. The symptoms in this stage are due mainly to the damage which the blood has sustained in the loss of its constituents from the transudation which has already taken place. Opiates may be given, and, owing to the remarkable degree of tolerance under these circumstances, they may be given in considerable doses; but much care should be observed not to induce narcotism. They should be given by either the mouth or the rectum; but never, in this stage, by the hypodermic method. Astringent remedies, if the stomach will retain them, may be added, such as tannic acid, the acetate of lead, bismuth, etc. If, however, these or other remedies provoke vomiting, they will do more harm than good. Remedies to allay vomiting may be tried—namely, the hydrocyanic acid, creasote, chloroform, etc.

In a large proportion of cases after collapse has taken place little can be done with much hope of success. Even if the vomiting and purging cease recovery may not follow. The blood may have been damaged irremediably. Under these circumstances it is plain that active treatment can effect nothing. Recovery, however, in a certain proportion of cases takes place and under a great variety of treatment. It may take place when no treatment is pursued. My first case of cholera in 1849 illustrated the fact just stated. The patient was brought into hospital completely collapsed. I remained with him several hours and resorted to various therapeutical measures. At length all measures were discontinued. He was allowed to drink abundantly of cold water, under the impression that the case was utterly hopeless, and therefore the indulgence could do no harm. Much to my surprise, after an absence of several hours I found the vomiting and purging had ceased and reaction was coming on. He recovered rapidly. I have been led to doubt whether any active treatment is of use in the collapsed stage, and I cannot doubt that it is often prejudicial. The object of treatment in this stage, aside from the arrest of vomiting and purging, is to excite and aid the efforts of nature in restoring the circulation, together with the functions dependent thereon. The measures to be employed for this object are external warmth, stimulating applications to the surface, diffusible and other stimulants, and alimentation.

The application of heat may be made by means of warm blankets or bottles of hot water placed near the body. The more active modes of applying heat are of doubtful propriety. I have never seen benefit from the warm bath or the application of steam or hot air. It is not desirable to excite perspiration, and if perspiration occur it should be wiped away with warm, dry cloths. Violent friction does more harm than good. The surface may be gently stimulated with sinapisms or the tincture of capsicum. Diffusible stimulants in the form of spirits and water should be given as freely as the condition of the stomach will permit, always recollecting the risk and the evils of inducing vomiting. They will be most likely to be retained if given in small quantities at a time and often repeated. If vomiting be provoked by either drinks, remedies, or aliment, more or less injury is done. The ethers, stimulants such as capsicum, the essential oils, cardamom, ginger, etc., are appropriate if they be grateful to the stomach and retained. Concentrated nourishment—essence of meat, chicken-broth, and milk—is to be given in small quantities at a time, provided the stomach will retain it. It is doubtless desirable to introduce liquid into the system as far as possible. The only objection to the free ingestion of water is the risk of provoking vomiting. Small lumps of ice should be freely allowed.

If the patient emerge from the collapsed state, the indications are to support the system by the moderate use of stimulants and by alimentation; to restore the function of the kidneys by diuretic remedies and mucilaginous drinks, bearing in mind that uræmia is one of the dangers of this stage; to restrain diarrhœa, if it occur, by anodynes and astringents; to strengthen by tonics; and to palliate by appropriate remedies the various symptoms which may arise.

The disinfection of the intestinal excreta in cases of cholera, as has already been mentioned, is important in view of the fact that they contain cholera germs; and during the prevalence of an epidemic this precautionary measure should be resorted to in all cases of diarrhœa.

CHAPTER IX.

INTESTINAL WORMS.

Ascaris lumbricoidès.—Oxyuris vermicularis.—Tricocephalus dispar.—Ascaris mystax.—Tæniæ.—Trichina spiralis.—Anchylostoma duodenale.

OF the various *Entozoa* which dwell within the human body, the largest number is found in the intestines. They give rise to more or less disturbance, and are properly enough considered in connection with the functional disorders of the alimentary canal. I shall treat of intestinal worms only in so far as concerns the practical duties of the physician. The study of these and of other parasites, as belonging to comparative anatomy and physiology and as a branch of zoology, is highly interesting and has led to valuable practical results, but other points than those which relate directly to them as involving morbid conditions would be here out of place.

The intestinal worms of frequent occurrence are the *Ascaris lumbricoides*, lumbricoid or round worm, and the *Oxyuris vermicularis*, thread or seat-worm. The worm known as *Tricocephalus dispar* is sufficiently common, but is of little or no practical importance. The different species of *Tænia*, or tape-worm, are of rare occurrence in this country, but their presence constitutes an important affection. These different worms will be noticed under distinct heads. The worm called *Anchylostoma duodenale* will be noticed in connection with the cylindrical worms. As appropriately classed among the intestinal worms, because received and developed within the alimentary canal, the *Trichina spiralis* will be considered in this connection.

Ascaris lumbricoides.

With this worm every young physician soon becomes familiar. It resembles the common earth-worm. It is the most common of all the intestinal worms. It is of a whitish or yellowish color; and the body is round, fusiform, and marked by a number of fine transverse rings. The mouth is surrounded by three tubercles. The sexes are distinct. The males usually measure from four to six inches in length and the females from ten to fourteen inches.

The common lumbricoid worm inhabits the small intestine. It rarely exists in early infancy, and is most common between the ages of three and ten. It is not very uncommon in adults, but is extremely rare in old age. This species of worm is not usually solitary. In different cases the number of lumbrici varies greatly. Sometimes the number is very great, and in autopsical examinations they are occasionally coiled together so as to form balls or masses of considerable size. Cruveilhier cites a case in which more than a thousand were found after death. They are not infrequently brought into the stomach by acts of vomiting, and are ejected from the mouth. They may find their way into the nares, and they have been known to pass into the Eustachian tube, frontal sinuses, and larynx. They may migrate from the intestine into the biliary ducts and gall-bladder. In a subject dead with lumbar abscess connected with caries of the vertebræ I found a great number within the hepatic ducts, some extending along the ducts and others coiled up. They may possibly give rise to hepatic abscess. They may penetrate into the pancreatic duct. They have been found in the peritoneal cavity and in abscesses within the abdominal walls. In these cases they escape through intestinal perforations caused by ulceration or sloughing incident to other morbid conditions. It is not probable that the worms ever perforate the intestines, as was formerly supposed. They have been observed to escape from the body through fistulæ communicating with the intestinal canal.

The SYMPTOMS denoting the presence of these or other worms within the intestinal canal are obscure. A host of symptoms having little or no significance have been enumerated by writers. Nothing is more common than the passage of lumbrici from the stomach or bowels, when there have been no symptoms of disorder. The morbid effects have heretofore been greatly exaggerated. These worms, it is probable, very rarely give rise to serious results except when they migrate into the biliary or pancreatic ducts, the Eustachian tube, or the larynx; and these migrations are exceedingly infrequent. There is no foundation for the belief that they give rise to a form of fever, as implied by the term *worm fever*. They are not infrequently, in cases of fever, passed from the bowels and found in the intestine after death, but their presence is merely a coincidence. That they may give rise to convulsions, epileptic paroxysms, chorea, and other affections of the nervous system, as is generally supposed, must be considered as by no means established.

Symptoms generally regarded as denoting these worms are—colic pains, tumefaction of the abdomen, impaired appetite, excessive flow of saliva, diarrhœa, itching of the nose, swelling of the face, offensive odor of the breath, disturbed, dreamy sleep, and grinding of the teeth during sleep. It is never prudent for a physician to declare positively that a child between three and ten years of age has not worms, for he is liable to be convicted of error by the operation of a vermifuge given without his sanction; but the only readily available diagnostic proof is the presence of one or more in the stools. When more or fewer of the above-named symptoms are present a brisk purgative may be given, the stools being examined with reference to diagnosis. If one or more worms be passed, it is proper to act as if there were others remaining. According to Davaine,[1] a microscopical examination of the feces will show the characteristic ova if lumbrici be present. He states that their ova are readily recognized, and hence the diagnosis may be made positive in this way. Practitioners in general, however, will be satisfied to base the diagnosis upon other facts.

The lumbricoid worm is propagated by ova. The body of a female lumbricus contains a vast number of eggs, estimated by Eschricht to be sixty-four millions. These ova are discharged with the feces. The observations of Davaine and others show that their vitality is retained for many months. Their ingress into the alimentary canal is probably by means of water containing them, and, finding favorable conditions in the small intestines, the production of the worms takes place. That special conditions are required may be inferred from the fact that these worms are so common in certain periods of life and so infrequent at other periods. As regards the nature of the conditions, we have no positive knowledge; but mucus in abundance is supposed to be a favorable nidus for their production.

The expulsion of lumbricoid worms is generally effected without difficulty. Beginning with a brisk purgative, which alone is sometimes sufficient, an anthelmintic remedy is to be given for several successive days. Of the various anthelmintics, those best suited for the destruction of these worms are chenopodium or wormseed, santonin, and spigelia or pinkroot. Santonin is most easily administered, and is perhaps as effective as the other articles. The dose is from 3 to 6 grains, repeated three or four times daily, given either mixed with sugar or made into *dragées* or troches. Wormseed is taken most conveniently in the form of the oil. From 5 to 10 drops may be given three times daily mixed with sugar or in an emulsion. The pinkroot has long been relied upon as an efficacious remedy. The infusion may be used, but the fluid extract of spigelia and senna is the most eligible form, the dose being from 1 to 4 drachms three times daily. Tanacetum or tansy and absinthium or wormwood are doubtless efficacious remedies. All these remedies act toxically; that is, by poisoning the worms. After two or three days a brisk purgative should be given. If with this treatment no worms be discharged, their non-existence may be inferred. If worms be brought away by this treatment, it need not be continued or repeated unless, from the continuance or recurrence of symptoms denoting disorder, it be inferred that their expulsion is not complete. The absence of the ova in the stools, according to Davaine, may be relied upon as proof positive of the absence of the worms.

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[1] *Traité des Entozoaires et des Maladies vermineuses de l'Homme et des Animaux domestiques*, 1860. The reader is referred to this work as treating of entozoa minutely and comprehensively. See, also, Heller, in *Ziemssen's Cyclopædia*, vol. vii., Am. ed.; Leuckart, *Die menschlichen Parasiten*, Leipzic, 1863–76; and Cobbold's *Treatise on Parasites*, London, 1879.

With a view to prevent the ingestion of the ova of lumbrici attention should be directed to the purity of the water used as drink. Water from shallow wells in the neighborhood of dwellings and from small rivulets should be avoided. Filtration would probably prove effectual in the way of prophylaxis. Davaine states that it is not common in Paris for children and others to be affected with this worm—a fact which he attributes to the custom of using filtered water.

Oxyuris vermicularis.

This worm, called also *Ascaris vermicularis*, and commonly known as the thread-, pin-, seat-, or maw-worm, inhabits the large intestine, especially the rectum. The size is small, as indicated by certain of the names in common use, and the length varies between two and five lines. The vermicular oxyurides frequently exist in immense numbers, and they are sometimes voided in large masses or balls agglomerated by mucus. They occur especially in young children, but they are not extremely uncommon at any period of life. I have met with several examples of their occurrence in aged persons.

These worms occasion pain in the rectum, tenesmus, and more especially pruritus at the anus. The latter symptom, which is often due to an herpetic eruption in this situation, should always excite suspicion of the presence of these parasites. The itching often occurs in paroxysms, and is particularly troublesome at night. Excitation of the sexual organs frequently occurs, with seminal emissions, and it is stated that the habit of masturbation in either sex may originate from this source. In girls the worms sometimes migrate into the vagina and occasion pruritus with leucorrhœa. The diagnosis is made without difficulty. On examination of the stools the worms are seen, in greater or less number, adhering to the feces, and on an ocular inspection of the anus they may generally be discovered. It is doubtful if they ever lead to any serious disorders, but owing to the irritation which they occasion and the loss of sleep, the general health may become more or less impaired.

The TREATMENT is simple and efficacious, but it may require a certain amount of perseverance. The same anthelmintics may be employed as for the destruction of the lumbricoid worms, given in the same way. Purgative remedies are to be given from time to time. In addition, enemas of either cold water, salt and water, or soap and water are to be administered once or twice daily. These are to be continued for a fortnight or longer, until all the local symptoms disappear. The treatment by enemas alone will frequently prove effectual. Various remedies in enemas have been advised, such as wormwood, vinegar, empyreumatic oil, asafœtida, camphor, sulphide of potassium, etc. Injections containing a small percentage of carbolic acid are efficacious. Infusion of quassia and decoction of aloes are also useful in enemas. The irritation or slight inflammation about the anus which is sometimes caused by the presence of these worms may be relieved by the application of a soothing unguent or the daily injection of a little almond or olive oil. Extreme cleanliness is essential in the treatment. Zenker and Heller have found the eggs of this parasite beneath the nails. The danger of auto-infection from this source should be guarded against.

Tricocephalus dispar.

This worm, as the name tricocephalus denotes, is characterized by the hair-like appearance of the cephalic extremity. It is from an inch and a half to two inches in length. The filiform cephalic portion forms about two-thirds of its length. As in the ascaris lumbricoides and the oxyuris vermicularis, the two sexes are distinct. The posterior or caudal extremity of the male is curved; that of the female is straight and bluntly pointed. It inhabits the cæcum especially, but is sometimes found in the colon, and very rarely in the small intestine. The propagation is by ova, which, according to Davaine, are readily recognized by means of the microscope in the dejections. The parasite is found frequently, when sought after, in the bodies of subjects of all ages dead with different diseases. Some years ago, when engaged in examining bodies dead with typhoid fever, I was led to examine the cæcum for it, and I rarely failed to find one or more. It is not positively known that its presence gives rise to disorder. Barth of Paris reported a case in which death took place with the symptoms of meningitis, and after death no lesions were found within the head, but the intestines contained an enormous quantity of worms of this species.

The foregoing species of intestinal worms belong in the class styled by helminthologists *Nematoda*, or cylindrical worms. (See Part I. p. 94.) Another worm belonging to this class was described by Bellingham under the name of *Ascaris alata*. It is the same parasite as the ascaris mystax, which infests the dog and cat. According to Cobbold, about seven instances of its occurrence in man have been noted. This parasite, as regards its practical importance, is in the same category with the tricocephalus dispar.

Tæniæ, or Tapeworms.

On the natural history of tæniæ, constituting the class *Cestoda* in helminthology, and commonly known as tapeworms, much light has been shed by the researches of Küchenmeister, Von Siebold, Leuckart, and others. Referring the reader to other works for a full account of these researches, suffice it to say that different varieties of tænia have been shown to be the cystic entozoa known as *cysticerci* and *echinococci* in a state of maturity or complete development. A cysticercus is an immature or undeveloped tapeworm. Different varieties of cysticerci are found in the solid parts of different animals, and each variety, received into the alimentary canal of another animal, and, there finding favorable conditions, becomes a perfect worm or tænia, each kind of cysticercus becoming a particular kind of tænia. A cysticercus, on the other hand, is the product of the ovum of a tænia. The eggs of the tænia, passing out of the body, finding their way into the bodies of men and other animals, and probably entering into the circulation, are transported to and fixed in solid parts, where they become cysticerci. The later, as long as they remain in solid parts, reach only a certain stage of development; if, however, the parts containing the cysticerci be eaten by another animal, they attain their complete development in the alimentary canal of the latter. Thus a particular cysticercus found within the liver of the rat, if eaten by the cat, is developed into the kind of tapeworm frequently found in the intestine of the latter; and the ova from this tapeworm received into the body of the rat give rise to the kind of cysticercus found in the liver of this animal. The ovum from the tænia of the dog, received into the body of the sheep, becomes the cystic entozoon called *Cœnurus cerebralis*, found in the brain of the latter, which, if eaten by the dog, becomes the tænia found in the intestine of this animal. These highly interesting, and in a practical view important, facts have been substantiated by observation and experiment.

The tænia or tapeworm is distinguished, as these names indicate, by its ribbon-like form. It is composed of a great number of segments or joints called proglottides, each one of which, after it attains to its full maturity, is provided with male and female generative organs. Several varieties have been found in man, but as regards medical practice the discrimination is not of much importance, the same measures of treatment being alike applicable to all. In the vast majority of cases the tænia developed in the human intestine is one of three kinds; and it will suffice to notice these.

In this country, of the three kinds of tænia, the one which has been supposed to be most common is the *Tænia solium*, or solitary tænia, so called because it is supposed always to exist singly. This is true as a rule, with some exceptions. Two or more tæniæ have been observed together, but such instances are rare. The tænia solium varies in length between four or five and thirty-five feet, the number of segments or joints numbering from 800 to 1000. Toward the head it becomes attenuated, forming a thread-like neck of considerable length, and in this portion there are transverse markings or rugæ, but no visible joints. The joints succeeding the neck are about as long as they are broad, but lower the length is twice or thrice the breadth. From the resemblance of these joints to the seeds of a gourd the worm has been called *Vermis cucurbitinus*, or gourd-worm. The generative foramina are placed near the middle of one of the margins of each joint, and are generally alternate. The head is about the size of a pin-head, hemispherical or triangular in form, and under the microscope with a low power it is found to present four projections or suckers and a coronet of twenty-six alternating short and long hooklets surmounting a projection called the rostellum. The joints of the inferior part are constantly thrown off, and are passed almost daily by stool. These exfoliated joints contain an immense number of ova. Bennett quotes an estimate of the number of ova in the tapeworm of a cat to be 12,500,000. It is fortunate that worms are not developed from the ova within the alimentary canal!

The second variety, called *Tænia mediocanellata* (*Tænia saginata*), has not until late years been generally recognized as distinct from the tænia solium, and the relative frequency of its occurrence in this country has not as yet been ascertained. It is probable that here, as in England and Germany, it is more common than the tænia solium. The generative foramina are situated alike in both varieties, but the joints are longer and thicker, and the head has four suckers, without hooklets or rostellum.

The third variety is known as the *Tænia lata*, or broad tapeworm, reckoned by some helminthologists as a species distinct from the true tænia, and named *Bothriocephalus latus*. This worm is distinguished from the tænia solium by the breadth of the joints, which are broader than they are long, by the situation of the genital orifices, which are in the middle of the joints, and by the different conformation of the head. The head is unprovided with hooklets, and hence this and the second variety are distinguished as the unarmed tæniæ, while the tænia solium is said to be armed. The form of the head is elongated, and instead of suckers it has two lateral longitudinal depressions or fossæ; hence the name *bothriocephalus*, from βοθρίον, a small pit. This worm may attain to even a greater length than the tænia solium. Like the latter, it is usually solitary. A tænia called *Bothriocephalus cordatus*, distinguished by the form of the head, is only known to infest the inhabitants of North Greenland.

Of these varieties of tapeworm, tænia mediocanellata and tænia solium are those commonly met with not only in this country, but in most countries. As already remarked, tænia mediocanellata seems to be the most common. Bothriocephalus latus has been observed chiefly in Russia, Switzerland, Ireland, and Japan. It is observed that this variety is likely to affect inhabitants living on the coasts of seas, lakes, and rivers, whence it has been inferred that it proceeds from a cystic entozoon existing in fish.[1] The opinion, however, is also entertained that the embryos of this worm may be taken into the system in drinking-water, and may be developed in the intestine without the intermediate stage of cystic development in the solid parts. The tænia solium is attributable to the ingestion of the *Cysticercus cellulosæ*, which is common in the flesh of swine. It is the presence of this entozoon in large numbers which renders pork "measly." Pork, eaten raw or partially cooked, is the source of this form of tapeworm. Thorough cooking destroys the cysticercus, and the development into the tapeworm does not then take place. It is a matter of observation that butchers and cooks are often affected with tapeworm, and it is stated that in Abyssinia, where the custom of eating uncooked meat prevails, nearly all persons of different ages become affected with tænia. The production of tænia in children by the use of raw meat as food in cases of so-called cholera infantum has been already referred to. The tænia mediocanellata is derived from a cysticercus found in beef. Tænia solium is the pork-tapeworm, and tænia mediocanellata is the beef-tapeworm. The frequency of one or of the other of these species in a given district bears a strict relation to the amount of underdone measly pork or beef consumed by the inhabitants.

The habitat of a tapeworm is the small intestine. When it attains a great length it extends into the large intestine, and it has been found to reach nearly to the anus. No age is exempt from the liability to its occurrence, but in the majority of cases the ages are between twenty and forty years. It occurs oftener in females than in males. Professor Armor has reported a case in which an infant five days old passed segments of tænia. Two months after the birth of the child the mother was treated for tænia.[2] The frequency of its occurrence varies much in different countries and in different sections of the same country. The report of the military surgeons of the French army from 1840 to 1848 showed only 7 cases among 250,000 soldiers. During a practice of forty-three years I have not met with more than thirty or forty cases.

The existence of a tapeworm is not indicated by well-marked diagnostic symptoms. The symptoms attributed to it are vertigo, tinnitus aurium, disturbance of vision sometimes amounting to temporary amaurosis, pruritus at the nose and anus, salivation, disordered appetite and digestion, colic or neuralgic pains in the abdomen, and emaciation. These are symptoms which may be due to other morbid conditions, nor are these symptoms uniformly, if generally, marked in cases of tapeworm. The existence of the worm is often discovered accidentally, and in persons who prior to the discovery considered themselves perfectly well. In the case of a young female affected with sciatic neuralgia I was led to prescribe a full dose of turpentine, and the result was the expulsion of a large quantity of tapeworm, the existence of which had not been suspected. After the existence of the worm is known disorders of all kinds are likely to be referred to it, and often the imagination creates various morbid sensations. Convulsions, epilepsy, and chorea are supposed to be occasional effects, but clinical observation supplies but little data for this supposition. The pathological effects of the presence of this worm have doubtless been greatly exaggerated. Tapeworms are almost invariably found in the intestines of dogs destroyed for experimental purposes, apparently in perfect health.

The DIAGNOSIS must rest on the discovery of detached joints or segments of the worm in the evacuations. This mode of diagnosis is always available. Segments are generally passed at short intervals, and hence it is usually not

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[1] Küchenmeister surmises that infection occurs from eating uncooked salmon.

[2] *Detroit Review of Medicine*, Jan., 1872.

long before its existence is ascertained. If there be reason to suspect its existence before segments have been seen, a brisk purgative may be given, and this will be likely to be followed by the expulsion of a greater or less quantity. If an examination of the stools for some time, and especially after the administration of purgatives, fail to discover any portions of the worm, the physician may be satisfied of its non-existence. Persons not infrequently fancy they have a tapeworm. This is one of the notions likely to be seized upon by hypochondriacs, and in some cases it becomes a fixed belief of which the mind cannot be dispossessed, becoming, in fact, an insane delusion. Several instances of this kind have fallen under my observation. A few instances are on record in which portions of the worm have been vomited.

With reference to TREATMENT a point of importance is the natural duration of the life of this parasite. It is undoubtedly long-lived. It is not uncommon for fragments to be passed at short intervals for many years. Cases have been reported in which this was observed for twelve, twenty-five, and even thirty-five years. If an interval of several months elapse without any segments having been discharged, and if subsequently they make their appearance, it is probable that a second worm has become developed. Sooner or later, the worm dies a natural death and a cure takes place spontaneously. If successive remedies have been used in such a case, the last remedy of course gets the credit of having effected a cure. For a cure to be effected with certainty the head must be expelled. The frequent exfoliation of segments is natural, and if, as an effect of a remedy or from any cause, a large portion or even all the body be expelled, reproduction by growth may take place as long as the head remains. It is, however, a matter of observation that the parasite is likely to die when the separation takes place near the head. As positive evidence of a cure the head is to be sought for; but it is not found in the larger proportion of the cases in which a cure is effected.

Of the remedies to be employed for the destruction of the tapeworm, one of the oldest, and certainly in many cases an effective remedy, is the oil of turpentine. It is most likely to be effective if given in large doses. A half ounce may be given in emulsion, with the addition of some aromatic tincture to prevent nausea, this dose being repeated every half hour until two or three ounces have been taken, or sometimes the whole amount taken at once is well borne. Strangury is an occasional result, but this is less likely to occur than when the remedy is given in small doses. After a large dose the patient feels as if slightly inebriated. This remedy, thus given, may be repeated if necessary after intervals of several days. Given in the quantity just stated, it generally acts as a purgative. Chabert's empyreumatic oil, which has been much in vogue as a remedy for tænia, is probably efficacious chiefly from the turpentine which enters into its composition, and is much more disagreeable than the turpentine alone. It is stated by Bellingham and others that turpentine, given in moderate doses and repeated for several successive days, is frequently successful.

The male fern (*filix mas*) is a tæniafuge, the efficacy of which rests upon the testimony of many observers of large experience. In my own experience it has proved promptly efficacious. It may be administered in powder, from half a drachm to a drachm being given in divided doses in the course of a few hours, but the oil and ethereal extract are more eligible preparations. The oil may be given in doses of a drachm either in mucilage or in gelatin capsules. The dose of the extract is from 20 to 30 grains. Dr. Alexander Fleming advises a drachm of the oil, with mucilage, to be given in an ounce and a half of sweet milk at bedtime, the dinner and evening meal of the day having been omitted. This mode of administration is advised on the supposition that milk being a favorite food of the worm, it is more effectually poisoned by imbibing the remedy in this vehicle.

Another effective tæniafuge is the kousso (*Brayera anthelmintica*), the dried flowers of a tree in Abyssinia, where tapeworm is exceedingly common. This remedy is said to be relied upon in that country. Within the past few years it has been used with much success in Europe and in this country. Half an ounce of the powdered flowers mixed with water may be given in a dose, both the liquid and sediment being taken.

Another remedy is an emulsion of pumpkin-seeds (*Cucurbita pepo*). The emulsion is prepared by pounding two ounces of the seeds in a mortar with half a pint of water and straining through cloth. This quantity will constitute a dose which may be repeated for several days. This remedy has proved successful in the hands of a number of physicians in this country. It has the advantage of producing no unpleasant effects, being, aside from its action as a vermifuge, innocuous.

The bark of the pomegranate-root (*Punica granatum*) is another anthelmintic remedy which has been found destructive to the tapeworm. A decoction made by boiling two and a half ounces of fresh bark in a pint and a half of water until this quantity is reduced one-half is the form in which this remedy is to be administered. The whole of the quantity just named may be given in the course of a few hours. It generally produces purging, and not infrequently vomiting. Kameela, the powder and hairs from the capsules of *Rottlera tinctoria*, is a powerful anthelmintic which has been found promptly efficacious in cases of tænia. The dose is from one to two drachms given in honey or thick gruel. It acts efficiently as a purgative. The remedy has been given with great success in combination with the ethereal extract of the male fern. Carbolic acid in doses of half a grain or a grain given hourly, if well tolerated, throughout the day, followed by a cathartic, has been found efficacious.

These are the most valuable of the anthelmintics which experience has shown to be capable of destroying the tapeworm toxically. The powder of tin (*pulvis stanni*), given in doses of a scruple or more, mixed with honey, has in some cases proved efficacious, probably destroying the worm by its mechanical action. It is far less reliable than the toxical remedies. Active cathartics, such as calomel and jalap, scammony, gamboge, and croton oil, occasionally succeed, their action probably being mechanical. They will generally bring away greater or less portions of the worm, but alone they are not to be relied upon for effecting a cure; that is, for destroying the worm. In conjunction with the toxical anthelmintics they are generally useful.

The success of the treatment will depend much upon accessory measures. The worm is nourished, not by nutriment sucked from the intestinal walls, but from the alimentary contents of the intestine, which enter the body of the parasite by endosmosis. An important part of the treatment, therefore, is to weaken the parasite by starvation. The patient, by way of preparation for the administration of the tæniafuge, should abstain from those articles of food which are digested in the small intestine for at least a day or two. Beef-tea or chicken-soup may be allowed freely. The treatment should be begun with a mild purgative, and the administration of the anthelmintic remedy should be followed after a few hours by a pretty active purgative. If the treatment prove unsuccessful, the strength of the patient should be restored by a return to a full diet and tonic remedies, and the same plan again repeated, employing the same remedy or selecting another. The treatment is to be repeated, allowing sufficient intervals for recruiting the strength, until a cure is effected. It is to be borne in mind that if the greater part of the worm be expelled, although the head may not be found, the death of the worm is likely to follow, and the treatment therefore need not be repeated until the discharge of fragments is again observed.

The prophylaxis involves care never to eat meat not thoroughly cooked. In meat much underdone the cysticerci which may be contained in it are perhaps not destroyed. Purity of the water used as a drink may be an important measure of prevention. The use of filtered water is to be recommended. The meat contained in the Bologna sausages is liable to contain living cysticerci. In view of the danger of acquiring a tapeworm it is doubtful if it be ever judicious to advise raw beef or pork as an article of diet.

Trichina spiralis.

From the discovery of the *Trichina spiralis* by Paget, and its description by Owen in 1835, to a recent date, this parasite had been observed occasionally in the muscles of subjects in the dissecting-room, but in a pathological view it was not considered to have any importance. Facts, however, accumulated within the past few years have shown that the introduction and multiplication of trichinæ within the body of man may give rise to morbid effects of great gravity which often prove fatal. Much information has been obtained respecting the natural history of the parasite, the circumstances under which the human body becomes infested by it, and the morbid phenomena which characterize the disease. This information has added to the nosological catalogue a new and highly important affection known as the *trichinal* or *trichinatous* disease, *trichinosis* or *trichiniasis*.

The trichina spiralis belongs in the class *Nematoda*. As found in the muscles it is coiled up in a cyst containing a granular substance at first, and afterward calcareous matter. When expelled from the cyst the parasites are seen to be round worms $\frac{1}{28}$ of an inch long and $\frac{1}{620}$ of an inch in thickness. So long as they remain in the muscular tissue the worms are quiescent; when, however, they are taken into the stomach and set free by the action of the gastric liquids upon the muscular tissue and the cyst, they pass from the stomach into the intestinal canal, and immediately begin to grow rapidly, attaining to three or four times their former size; they acquire fully-developed generative organs, copulate, and, at the end of a week the female parasites contain living young in great abundance. According to Virchow, each trichina gives birth to two hundred young; Gerlach says four hundred, and Leuckart, one thousand. The young trichinæ shortly after birth penetrate the mucous membrane, and find their way rapidly to the different muscles throughout the body. They appear to select the voluntary muscles for their permanent dwelling-place. It is stated that they have not been found in the muscular walls of the heart. In other than muscular organs they are rarely found.

Trichinæ are found especially in the muscles of swine, but they have been found also in the muscles of various other animals—namely, eels, cats, rats and mice, dogs, badgers, hedgehogs, and moles. They find their way into the bodies of swine from these animals feeding upon the flesh and excrement of other animals infested with the parasites, especially rats and mice. Hence, to prevent trichiniasis in swine and in other animals which serve as food for man, it is highly important to cut off all the sources of the disease in the diet of these animals. They find their way into the alimentary canal of man chiefly from the eating of trichinous pork not subjected to processes of cooking sufficient to destroy the parasites. They are not destroyed by salting or smoking, and they may retain their vitality in roasted or broiled meat much underdone. To effectually secure their destruction every portion of meat which is eaten should have been subjected to thorough cooking. The custom which prevails among the Germans of eating sausages and smoked hams uncooked involves a liability to the disease. It has been traced to eating pork chops which were underdone in the centre, although sufficiently cooked on the outside. Meat abounding in trichinæ may present no evidence of the fact as regards the gross appearance, especially if a calcareous deposit have not taken place in the cysts which contain them; hence, trichinous meat may be sold and bought without any suspicion of its being unwholesome. The only reliable evidence of the presence of trichinæ in the meat procured for food is afforded by microscopical examination. The trouble, however, of such an examination precludes its being generally resorted to as a means of protection. Moreover, a microscopical examination of a few specimens is not adequate to determine that trichinæ are not present. They may not be found in the few specimens examined, although present in other portions. Hence, this test is reliable only in a positive not in a negative point of view.

After becoming encysted in the muscles the trichinæ retain their vitality for an indefinite time. In one case trichinæ were found encysted and alive in portions of muscle attached to a cancerous tumor removed from a patient. The previous history of this case showed conclusively that the patient was affected with trichiniasis twenty-four years before the operation.[1] The flesh of animals, therefore, which have been affected with trichiniasis will ever afterward be likely to contain living trichinæ. Moreover, the flesh of a single trichinous animal may produce trichiniasis in a very large number of persons, so that the disease in its prevalence may present the character of an epidemic. Muscular trichinæ, however, may after a time become completely calcified, in which case they are innocuous.

The clinical history of the disease caused by trichinæ is highly important with reference to an early diagnosis. The primary symptoms relate to the alimentary canal, and are due chiefly to the perforation of the mucous membrane by the newly-produced worms. Abdominal pains, vomiting, and diarrhœa characterize the first stage of the disease. These symptoms occur within ten days after the ingestion of trichinous meat; that is, as soon as the young worms have been produced and become developed so as to begin to migrate toward the muscles. It is not difficult to understand that the aggregated punctures of the mucous membrane by these parasites should occasion notable disturbance when it is considered that the trichinæ which have been found to be contained in a half pound of meat may be sufficient to give birth in a few days to a brood numbering 30,000,000. It is stated that peritonitis may be produced by the passage of worms into the peritoneal cavity.

The secondary symptoms relate to the muscles. Pains resembling those of myalgia are occasioned by the entrance of the trichinæ into the muscles. Certain of the muscles become contracted in some cases, and their extension occasions great suffering. Constitutional disturbance more or less marked accompanies both the primary and the secondary symptoms. The general symptoms are not unlike those of typhoid fever, for which the disease is likely to be mistaken. The temperature in the axilla varies between 101° and 106°. Œdema of the face or lower extremities often occurs, and sometimes there is anasarca. Sweating is generally prominent as a symptom. Death takes place in a certain proportion of cases after a protracted period of suffering and exhaustion, being often preceded by coma. It is stated that life in some cases is destroyed by the impairment of the respiratory muscles from the presence of the parasites. Aphonia is a symptom which exists in some cases, attributable to the presence of trichinæ in the muscles of phonation. Deafness may be caused by trichinæ in the stapedius muscle. The danger, *cæteris paribus*, is proportionate to the abundance of trichinæ generated within the alimentary canal. If the number be not sufficient to cause death from the amount of local and constitutional disturbance which they occasion, recovery takes place very slowly, the illness lasting for several weeks or even months. The trichinæ become encysted in the muscles, thereafter remaining quiescent.

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[1] *Medical News and Library*, Philadelphia, May, 1866.

Diarrhœa and abdominal pains, followed by muscular pains, together with more or less constitutional disturbance, should excite suspicion of this disease. Inquiries respecting food should be instituted, and if any portion of the suspected meat remain, it should be examined microscopically. The diagnosis may be made still more complete by harpooning some one or more of the painful muscles and obtaining portions sufficient for examination with the microscope. Davaine suggests that the presence of trichinæ in the stools may be ascertained, and the diagnosis in this way rendered positive. If clinical observation should establish the availability of the latter method of arriving at a diagnosis, it would have this advantage—namely, the existence of the disease may be positively ascertained before the trichinæ migrate from the alimentary canal. The practical importance of making the diagnosis thus early is sufficiently obvious.

It is evident that the treatment of this disease to be effective must be employed while the worms are in the alimentary canal. Hence, the importance of an early diagnosis. It remains to be ascertained whether treatment can be effectively employed in this stage, and to determine the measures most likely to prove effective. The object is either the destruction of the worms or their expulsion from the intestines. Cathartics are indicated for the latter object, but clinical experience has yet to show what toxical anthelmintics are best suited to destroy this parasite. Benzine has been found by Mosler to destroy the trichinæ in the intestinal canal, when given in doses which are well borne by the patient. Mosler's method of treatment with this remedy is to have a mixture consisting of two drachms of benzine, an ounce of licorice-juice and mucilage of gum-arabic, and four ounces of peppermint-water. Of this mixture a tablespoonful is to be given every one or two hours. This remedy, however, has not proved efficacious; but it is to be considered that the diagnosis is rarely made prior to the emigration of the parasites from the alimentary canal. The carbolic acid is a remedy which should be tried with a view to the destruction of the worms in the intestinal canal. After the trichinæ have left the intestines the opportunity for destroying or expelling them has passed. Recovery now depends on the ability of the system to endure them until they become encysted. Measures to palliate pain, to restore disordered functions, and to support the powers of life are indicated, and the success of the treatment will depend on the judicious employment of measures for these ends.

The prevention of this disease is the great practical benefit to be derived from its discovery. The mode of prevention is sufficiently simple—namely, abstaining from meat, especially the flesh of swine, not thoroughly cooked. The process of smoking, pickling, or salting cannot be relied upon for the destruction of this worm. Uncooked sausages cannot be eaten without danger. Heller states that the parasite is destroyed by a temperature of 155° F.

Anchylostoma duodenale.

This worm was discovered by Dubini in Milan in 1838. Its relation to a severe form of anæmia which prevails in Egypt was first made known by

Griesinger in 1851. The disease to which this parasite bears a causative relation has received various names, of which the following are the most important: Egyptian chlorosis, anchylostoma disease, anchylostomiasis, tropical chlorosis, the anæmia of the workers in tunnels, etc. The anchylostoma disease is common in Egypt, in Brazil, and in some other tropical countries. It has also been observed in Italy, and was not uncommon among the workmen engaged in building the St. Gothard tunnel. It has recently been observed among workmen in coal-mines and in brickyards in Belgium, also in the neighborhood of Cologne, and occasionally in other parts of Germany. It has been thought that the parasites are brought to these places by Italian workmen. This parasite has been found also in certain cases of geophagia. No cases of the disease have been described as occurring in this country.

The anchylostoma duodenale belongs to the family Strongylidæ of the order of nematoid worms. It is also called strongylus or dochmius duodenalis. It is found in the upper part of the small intestine in man. The body of the worm is round and thread-like. The length of the female is from 6 to 18 mm., that of the male from 6 to 10 mm. There is an average of about three females to one male present. The conical pointed head is bent backward, and presents a mouth surrounded by a horny capsule, and possessing on the ventral side four hook-like teeth and on the dorsal side two vertical teeth. The eggs of anchylostoma are oval in shape, from 44 to 67 μ long and from 23 to 40 μ broad. They undergo the first segmentation within the intestine. Outside of the body, in muddy water and under artificial cultivation at a temperature of from 25° to 30° C., embryos develop and escape from the eggs, and then become encysted as larvæ in a clear chitinous layer. These larvæ or embryos when taken into the human intestine develop into the mature worms. The anchylostoma worms do not multiply within the intestine, so that the number there present corresponds to the number of embryos which have been swallowed.

The anchylostoma duodenale bites with its claw-like hooks or teeth the intestinal mucous membrane, and may penetrate to the submucous coat. Here it sucks the blood from the intestinal vessels. The female worms are those found most gorged with blood. Sometimes the living worm is found rolled up in the substance of the mucous or the submucous coat in a little cavity filled with blood. The parasite leaves behind an ecchymosis of the size of a pea with a white spot in the centre, presenting a little hole through which blood can ooze.

The anchylostoma duodenale proves dangerous both by causing digestive disturbances and by withdrawing blood from the body. The leading symptom of the anchylostoma disease is anæmia, which may become as profound as pernicious anæmia, and in fact has often been mistaken for pernicious anæmia. When a large number of anchylostoma embryos are introduced at once or rapidly into the intestine, the anæmia develops acutely; and when they are introduced more slowly the anæmia is chronic in its development. Other symptoms are colicky pains in the abdomen, nausea, vomiting, constipation alternating with diarrhœa, and abnormalities of the appetite. The stools may or may not contain blood.

The DIAGNOSIS is based upon finding the eggs or the mature worms in the feces. These are present after the administration of a brisk cathartic. The number of eggs found gives a clue as to the number of worms present. The degree of anæmia is not always proportionate to the number of worms present.

The DURATION of the anchylostoma disease varies between a few months and several years.

The PROGNOSIS depends upon the degree of the anæmia and the obstinacy of the diarrhœa and other digestive disturbances. A fatal termination is not uncommon when the disease is left to pursue its own course. Nevertheless, the disease may cease spontaneously by the passage or death of the parasites. The anchylostoma eggs are sometimes found in the feces of those who present no symptoms of disease.

The TREATMENT consists in the administration of anthelmintics and of iron. An anthelmintic of great repute in Brazil against this parasite is doliarina, a preparation made from the juice of the tree *Ficus doliaria*, and mixed with aromatics and some form of iron. About a drachm of this is given three times daily. The ethereal extract of male fern in large doses has also been found useful in expelling the parasites. Thymol in form of powder has likewise been recommended.

Animals not entozoic, such as snakes, slugs, and lizards, are not infrequently exhibited to physicians as having passed from the alimentary canal. It is a popular notion that they may live and grow indefinitely within the stomach and intestine and give rise to a variety of morbid symptoms. In the great majority of cases the statements with respect to the passage of these animals are simply falsehoods, proceeding generally from a morbid exaggeration of that craving to become objects of curiosity and interest which enters largely into the mental constitution of many persons. In some cases, however, the statements are honestly made, persons being deceived. Prof. Dalton performed a series of experiments to ascertain the duration of life of the common garden slug (*Limax agrestis*) and the water-lizard (*Triton millepunctatus*) after having been introduced within the stomach of a dog. The former were found completely dead at the end of nine and a half minutes, and no traces of them were discoverable when the dog was killed an hour after their introduction. The lizards were found perfectly dead and about to undergo the digestive process at the end of fifteen minutes.[1] It is safe to set down most of the cases referred to as cases of either self-deception or imposition. Maggots, however, have been known to be discharged from the nasal cavity, the mouth, and the rectum. They may be developed in the nasal cavity from ova inhaled.

CHAPTER X.

DISEASES OF THE PERITONEUM.

Acute Diffuse Peritonitis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Circumscribed Peritonitis.—Chronic Peritonitis.—Hydro-peritoneum.

THE peritoneal cavity, like other serous cavities, has been generally regarded as a large lymph-sac lined with a single layer of flat, polygonal cells called endothelium. (For a different conception of the nature of this cavity the reader is referred to p. 115.) It communicates by means of minute openings, called stomata, between the endothelial cells with the lymph-spaces in the subserous connective tissue. Liquids and small particles are readily absorbed from the peritoneal sac by the adjoining lymphatic canals. By this free communication with the lymphatic system the peritoneum readily receives infectious and otherwise deleterious substances which may be present in adjoining organs. This disposition favors the propagation of inflammation from these organs to the peritoneum. Peritonitis may be acute or chronic, diffuse or circumscribed. It will be convenient to consider separately acute diffuse or general peritonitis, circumscribed or partial peritonitis, and chronic peritonitis. Some writers divide the disease into several varieties according to its different causes or to the circumstances under which it is developed. It will suffice to notice these diversities under the head of Causation. Puerperal peritonitis is an important variety presenting certain peculiarities, but it does not fall within the scope of this work to consider this form fully or separately. It is treated of at length in works on obstetrics and on the diseases of women.

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[1] *American Journal of Medical Sciences*, April, 1865.

Acute Diffuse Peritonitis.

ANATOMICAL CHARACTERS.—The pathological changes are essentially the same as in inflammation of other serous membranes. The intestine, as a rule, is distended with gas. The inflammation usually begins at one spot, and extends with more or less rapidity over the entire membrane. The inflammatory changes are usually most marked at the starting-point, and are elsewhere more intense in certain places than in others. At first there is redness due to congestion and ecchymoses. The serous surface loses its normal lustre, and soon it is coated with a thin, grayish layer of fibrillated fibrin, which at first contains few cells, but afterward becomes richly infiltrated with pus-cells, and then assumes a greenish-yellow color. The exuded fibrin causes the coils of intestine to become agglutinated to each other, to neighboring viscera, and to the abdominal walls. Together with the exudation of fibrin there is an effusion into the peritoneal cavity of more or less serum containing flocculi of fibrin and some pus-cells. The subserous tisssue is swollen and œdematous, and contains pus-cells, usually in swarms about the veins. The other coats of the intestine may be somewhat altered. The muscularis is usually œdematous, and, especially in cases of puerperal peritonitis, there may be inflammation of the mucous membrane. While the chief source of pus-cells is from the blood, there is sufficient evidence that the endothelial cells lining the peritoneum likewise take an active part in the inflammatory process. They become swollen, granular, multinucleated, and by their proliferation produce young cells.

Different cases of peritonitis vary greatly as to the quantity and character of the inflammatory products. These products are fibrin, serum, and pus-cells, mixed with a certain number of red blood-corpuscles. According to differences in causation and in individuals, sometimes one and sometimes another of these products preponderates. In some cases the exudation is chiefly fibrinous, in others chiefly serous, and in others purulent. Again, the exudation may be described as sero-fibrinous, sero-purulent, fibrino-purulent, etc. Peritonitis with fibrinous exudation and with but little fluid is sometimes called dry or adhesive. In some cases the peritoneal cavity is distended by a large quantity of serum. Such cases are generally secondary to ascites. In the majority of cases the exudation may be described as sero-fibrinous; that is, the peritoneal surfaces are coated with fibrin, and the cavity contains a moderate amount of serum rendered somewhat turbid by admixture with fibrin and pus-cells. Purulent effusions are also frequently met with. All grades of transition occur between the moderately turbid serum just described and thick creamy pus. Peritonitis due to perforation, to traumatism, and to puerperal fever and other infectious processes is generally purulent. In septic peritonitis the exudation may be foul-smelling and ichorous. In very rare cases it has a colloid consistence. Not infrequently it has a reddish or brownish tint from admixture with blood, but a truly hemorrhagic exudation belongs to certain forms of chronic rather than to acute peritonitis. It is not rare to find collections of serum or of pus encysted in different parts of the peritoneal cavity by means of fibrinous or of old organized adhesions. In connection with these collections of pus there have been observed suppuration and ulceration of portions of intestine with which the pus is in contact, and even perforation from without inward (ulcerative peritonitis).

If peritonitis terminate in recovery the serum is absorbed, and the fibrin, having undergone molecular disintegration, is likewise absorbed. Large collections of pus cannot be absorbed, but the pus may lose its watery constituents, become inspissated, and be converted into a kind of cheesy mass in which lime-salts may be deposited. New connective tissue in the form of adhesions and of thickening of the serous membrane is a result of peritonitis. The method of formation of this new tissue resembles that already described for inflammation of the pleura (p. 117). There may be adhesions of the coils of intestine to each other, to the solid viscera, and of the opposing parietal and visceral layers of the membrane. These adhesions may be innocuous, but they may also be the cause of serious and even fatal consequences. In the spaces or loops formed by bridles of newly-formed tissue portions of intestine are liable to become strangulated. The contraction of the new fibrous tissue may lead to constriction of the intestine. Coils of intestine may be so bound together by adhesions that their peristole is greatly impaired. In cases in which the cause of the peritonitis is not evident it is always necessary to search carefully for some lesion to which the inflammation can be referred. The presumption is in any given case against a primary peritonitis. A causative lesion will be found in most cases in some organ or part within the abdominal cavity, such as perforation of a hollow viscus, hernia, typhlitis, perityphlitis, and inflammation of other parts adjoining the peritoneum, especially the female generative organs.

CLINICAL HISTORY.—Acute peritonitis is sometimes developed gradually. Abdominal pain and soreness, progressively increasing, may be felt one, two, or three days before the local and general symptoms are sufficient to show the existence of the disease; but in most cases the attack is abrupt and the disease is quickly declared. The disease is often in these cases ushered in by a chill. Pain is usually a marked symptom. Beginning at a particular point, it extends over the whole abdomen. It is burning or lancinating in character. It is increased at times so as to constitute marked exacerbations, and not infrequently in these exacerbations the character of the pain is that of spasm or colic. Acute pain is produced by a deep inspiration. The respirations are usually shortened, and by way of compensation increased in frequency. The movements of the diaphragm are restrained, and those of the ribs proportionately increased; the breathing, in other words, is costal. Acts of coughing and sneezing occasion intense pain. Movements of the body are painful. The degree of suffering from pain varies in different cases. It is generally great, often extreme, occasionally moderate or slight; and this symptom may be wanting. Tenderness on pressure over the abdomen is usually marked. This symptom is very rarely wanting. The tenderness is often such that the slightest pressure is painful, but the degree of tenderness varies considerably in different cases. The pain and tenderness are frequently most marked in a particular circumscribed portion of the abdomen. These symptoms continue during the course of the disease. Owing to these symptoms the patient often lies upon the back, with the knees and thighs raised, in order to lessen the tension of the abdominal muscles and to relieve the abdomen of the weight of the bedclothes. This decubitus, however, is by no means constant. The patient may lie on the side with the knees and thighs flexed, or even on the back with the lower limbs extended. More or less tympanites is generally present, due to paralysis of the muscular coat of the intestine. The abdomen is often greatly distended and tense; and this condition persists during the course of the disease. The abdominal distension offers a mechanical obstacle to the descent of the diaphragm, and thus increases the dyspnœa, which if great is accompanied by cyanosis. The distension may be sufficient to displace the heart and to push the liver upward. If the tympanites be not great, the abdominal muscles, especially the rectus muscle on either side, are usually notably rigid and resisting. The bowels, as a rule, are constipated, but the exceptions to this rule are not very infrequent. Constipation sometimes exists at first, diarrhœa subsequently occurring, and occasionally diarrhœa exists at the outset. The latter symptom is most frequent in cases of puerperal peritonitis, in consequence of the coexistence of inflammation of the intestinal mucous membrane. Vomiting is a frequent, and in some cases a prominent symptom. The vomited matter consists of mucus and the contents of the stomach, and afterward of bile, which often has a notably green color. This grass-green or spinach color is, however, not characteristic of peritonitis. The acts of vomiting occasion great pain. In the latter part of the course of the disease the contents of the stomach are ejected by regurgitation rather than by vomiting. Thirst is usually urgent. Obstinate and painful singultus is a not infrequent symptom.

The temperature of the body is more or less raised, but there is much variation in different cases as regards the degree of pyrexia. In some cases there is but a moderate increase of temperature; and in other cases there is hyperpyrexia. There may be considerable variation in the same case during the course of the disease. The temperature sometimes falls to nearly the normal shortly before death. The pulse is more or less accelerated. It often becomes quite frequent, numbering 120 or 130 per minute; but in some cases the acceleration is moderate. It is generally small, and sometimes hard or wiry. The sense of prostration is considerable or great. Perspiration is liable to occur. The countenance denotes the gravity of the disease, and the expression is haggard, pinched, and anxious. In some cases the upper lip is elevated and drawn tightly over the teeth. This appearance is characteristic, and when present it points to the disease, but it is by no means uniformly present.

Difficulty in micturition is a frequent symptom, due in part to paralysis of the muscular tunic of the bladder in consequence of inflammation of its investing membrane, and partly to loss of power over the abdominal muscles in consequence of the abdominal distension. Moreover, the pain occasioned by the effort to micturate leads the patient to postpone it as long as possible. Retention of urine, requiring the use of the catheter, is not uncommon. The urine is high-colored, and throws down a lateritious deposit of urates. Pain in the penis is a symptom in certain cases of peritonitis. This pain is sometimes extremely intense. In some cases there is priapism with intense venereal desire.

The intellect in cases of acute peritonitis is generally not disordered. Slight delirium sometimes occurs, especially in fatal cases, toward the close of the disease.

PATHOLOGICAL CHARACTER.—Acute peritonitis, as regards pathological character, does not differ essentially from other acute inflammations of serous membranes.

CAUSATION.—Acute diffuse inflammation of the peritoneum, exclusive of puerperal peritonitis, is not a frequent disease. The liability of the peritoneum to become inflamed is very much less than that of the pleura or the pericardium—a fact not easily explained. In the great majority of cases peritonitis is incidental to some other affection of the abdominal viscera. The most frequent cause, exclusive of its occurrence in childbed and of the cases in which it is produced by traumatism, is perforation of the alimentary canal. Perforation of the ileum is incident to the intestinal lesions of typhoid fever, and peritonitis thus produced is one of the occasional events pertaining to the clinical history of that form of fever. The intestinal ulcerations which occur in certain cases of phthisis sometimes, although very rarely, lead to perforation, and also other ulcers situated in either the small or large intestine. Perforation from ulceration or sphacelation of the extremity of the appendix vermiformis of the cæcum is not of very rare occurrence. (Vide p. 485.) Perforation of the stomach and of the duodenum is an occasional event in cases of gastric and duodenal ulcer. Whatever be the seat of the perforation, the gaseous and other contents of the alimentary canal escaping into the peritoneal sac, peritonitis becomes at once developed. The escape of bile from rupture of the gall-bladder or of the cystic, hepatic, or common duct gives rise to peritonitis. Other causes are the discharge of pus into the peritoneal cavity from hepatic, ovarian, or other abscesses, rupture of the urinary bladder or ureter, and the evacuation of hydatid cysts. Inflammation, at first local, may become general, as in certain cases of intussusception and strangulation of intestine, of typhlitis, and perityphlitis without perforation. Injections into the cavity of the uterus have been known to give rise to the disease, the liquid injected passing into the peritoneal cavity through the Fallopian tubes. In certain cases of puerperal peritonitis the inflammation begins in the uterus and is propagated to the peritoneum. The disease may be produced traumatically by accidental wounds of the abdominal walls and violent contusions. It may be caused by surgical operations which involve opening of the peritoneal cavity in cases of ovariotomy, of laparotomy in cases of obstruction, etc. It sometimes follows paracentesis, very rarely, however, except in cases in which the kidneys are diseased. It occurs in intra-uterine life—a fact which Sir James Simpson was the first to observe. The extension of inflammation from the umbilical vessels to the peritoneum explains the occurrence in some instances of infantile peritonitis.

Acute peritonitis is one of the local affections incident to the morbid conditions of the kidneys collectively called Bright's disease. In two cases under my observation it was developed in connection with acute articular rheumatism, the patient recovering in each case after a short duration of the disease. Exposure to cold may give rise to it. I have known its occurrence to be attributable to this cause. It has been observed to occur not infrequently in the Northern States among laborers employed in the winter and spring to repair water-wheels damaged by ice, this occupation requiring them to work standing in water.

Occurring as an epidemic affection in childbed, it doubtless proceeds from a special cause. During the prevalence of puerperal peritonitis, epidemic erysipelas has been observed to prevail, showing some pathological relationship between the two affections.

DIAGNOSIS.—The diagnosis of acute diffuse peritonitis is not difficult in cases in which its characteristic features are well marked, as they are in the majority of cases. These features are sometimes obscure or wanting, and it is then liable to be overlooked or to be confounded with other affections.

From acute enteritis it is distinguished generally by a greater degree of pain, greater frequency of the pulse, more tenderness on pressure over the abdomen, more tympanites, rigidity of the abdominal muscles, and the evidence on all sides of a graver malady.

It is sometimes mistaken for functional colic. The pains in some cases of peritonitis are like those of colic; but colic lacks the continuous pain, the abdominal tenderness, the muscular rigidity, the tympanites, the pyrexia, the frequency of the pulse, prostration, etc. of acute peritonitis. These symptoms, however, may not be immediately developed in peritonitis, and a little delay may therefore sometimes be necessary in arriving at a positive diagnosis.

Myalgia (muscular rheumatism) affecting the abdominal muscles may give rise to certain of the local symptoms of acute peritonitis. Instances, however, of myalgia limited to these muscles are rare. The diagnostic points are as follows: In peritonitis movements of the body occasion pain over the whole abdomen, whereas in myalgia the pain is limited to certain muscles, and perhaps confined to one side of the abdomen. The same is true of tenderness on pressure. Moreover, in peritonitis the pain from pressure is proportionate to the amount of pressure, but in myalgia deep, firm pressure may be made without augmenting the pain. These are diagnostic points pertaining to the local symptoms, but the general symptoms present in acute peritonitis are wanting in the so-called muscular rheumatism.

Lumbo-abdominal neuralgia with hyperæsthesia of the abdominal walls or integument sometimes simulates closely peritonitis as regards certain local symptoms. The pain may be severe, the tenderness great, the decubitus on the back with the thighs and knees flexed, and not infrequently there is more or less tympanites. Cases presenting these symptoms are not uncommon. The patients are generally women, and they manifest more or less of hysterical phenomena. The general symptoms of acute peritonitis, frequency of pulse, prostration, etc., are wanting in these cases; but attention to certain points connected with the local symptoms will suffice for the differential diagnosis. The tenderness in the neuralgic affection is more superficial; the patient shrinks from the slightest touch, but firm, prolonged pressure with the open palm is often well borne, and may even afford relief, whereas in peritonitis the pain is always proportionate to the amount of pressure. The muscular rigidity of peritonitis is wanting.

I have known acute peritonitis to be mistaken for sporadic cholera in a case in which vomiting and purging occurred early and were prominent symptoms. In the exceptional cases in which these symptoms are sufficiently prominent to suggest that affection, the error of diagnosis is to be avoided by finding, associated with the vomiting and purging, the local symptoms of peritonitis—namely, pain, tenderness, tympanites, and muscular rigidity, together with general symptoms denoting an inflammatory affection—symptoms not belonging to the history of sporadic cholera.

Acute peritonitis is sometimes remarkably latent, the local symptoms, usually so strongly marked, being obscure or wanting. Pain and tenderness may be slight or even absent. Muscular rigidity in such cases may be a very valuable diagnostic symptom. As regards occasional latency, this disease resembles other inflammations of serous membranes—namely, pleuritis and pericarditis. Pain and tenderness may be obscure in consequence of the coexistence of other affections which occasion delirium or blunt the perceptions of the patient, and from the absence of these local symptoms peritonitis may be overlooked. In some cases of peritonitis produced by perforation—in the course of typhoid fever, for example—the manifestations of pain and tenderness may not be sufficient to direct attention to the abdomen. In such cases other local in conjunction with general symptoms must be relied upon in the diagnosis. Certain physical signs may enter into the diagnosis of peritonitis. There is not infrequently liquid effusion sufficient to be recognized by percussion and palpation. (Vide Diagnosis of Hydro-peritoneum.) A peritoneal friction sound may be heard by auscultation over the liver.

It is desirable in cases of peritonitis to determine whether the disease be idiopathic or dependent on some one of the various local circumstances which in the majority of cases stand in a causative relation to it. In consequence of the relative infrequency of idiopathic peritonitis the probabilities in any case are in favor of the existence of some one of these causative circumstances. Developed in the course of typhoid fever, it generally, but not invariably, proceeds from perforation of the intestine. If developed in a case of pulmonary tuberculosis accompanied by diarrhœa, chronic peritonitis not preceding its development, perforation connected with intestinal ulceration is the probable cause. Perforation of the stomach is probable if the peritonitis occur suddenly in a case presenting the symptoms of gastric ulcer. Perforation of the intestine, connected with latent ulceration, is to be very strongly suspected whenever the affection is developed suddenly, without any appreciable cause, in a person apparently in health; and if the pain and tenderness first occur in the neighborhood of the cæcum, and continue most marked in this region, the seat of the perforation is probably the appendix vermiformis. In cases of perforation gas usually escapes into the peritoneal cavity. Its presence here gives rise to intense tympanitic resonance on percussion, extending over the liver and taking the place of hepatic flatness. Tympanitic resonance over the liver, however, cannot be considered as proof of the presence of gas in the peritoneal cavity, inasmuch as this resonance is found not infrequently when the transverse colon is much distended with gas or when this portion of the intestine is pushed upward above the lower margin of the liver; but persistent hepatic flatness is proof almost absolute against perforation. Clinical observations, in my experience, substantiate this statement. In a cadaver in which the intestines are nearly free from gas, and even when the muscular walls of the abdomen are notably stiff from cadaveric rigidity, the injection of a small quantity of air into the peritoneal cavity suffices to render the resonance over the liver tympanitic. This experiment I have repeatedly performed.[1] Perforation of the ureter may be strongly suspected if the sudden development of the disease have been preceded by symptoms denoting obstruction from calculus or some other cause. In like manner, perforation of the ductus communis choledochus is the probable cause if symptoms denoting the impaction of gall-stone in this duct have preceded. The disease is attributable to intussusception or internal strangulation of the intestine when preceded and accompanied by symptoms denoting these causes of obstruction of the bowels. Its connection with renal disease is to be determined by an examination of the urine for albumen and tubular casts. Finally, if evidence of the existence of any of the causative circumstances referred to be wanting, and the disease be fairly attributable to exposure to cold, it is to be considered as idiopathic.

PROGNOSIS.—Acute general peritonitis is always a grave disease, involving danger to life. In the majority of cases the danger is greatly enhanced either by the circumstances on which the development of the disease depends, as, for example, intestinal perforation, or by coexisting affections, such as renal disease. We have no data for determining the amount of intrinsic tendency to death belonging to simple idiopathic peritonitis. There is reason to believe that the rate of fatality in all cases depends much on the mode of management. Judiciously treated in uncomplicated cases, the prospect of recovery is good. In complicated cases—including under this head cases in which the disease is connected with gastric or intestinal ulceration, Bright's disease, etc.—the prognosis is always very unfavorable. Under certain circumstances the disease runs rapidly to a fatal issue. It may destroy life within a few hours if developed as a result of perforation. Recovery, however, is not impossible in cases of perforation, provided the opening be not large and if closure be quickly effected by adhesions around it. The average duration in fatal cases is from five to six days.

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[1] Vide paper by the author in *Transactions of the Medical Society of the State of New York*, entitled "On the Physical Diagnosis of Peritonitis with and without Perforation of the Alimentary Canal," 1882; also article by the author, entitled "On the Persistence of Flatness on Percussion over the Liver, as Proof that Acute Diffuse Peritonitis is not associated with Perforation of the Stomach or Intestine," in the *Medical News*, Jan. 17, 1885.

The mode of dying is by either rapid or slow asthenia. An unfavorable progress of the disease is denoted by a progressive increase of the feebleness and frequency of the pulse, progressively increasing prostration, coldness of the extremities, cyanosis, hiccough, regurgitation, etc. On the other hand, a favorable progress may be inferred from improvement as regards the general symptoms just referred to, together with diminution of the tympanites and muscular rigidity of the abdomen. Convalescence is generally slow, more or less tenderness and colic pains persisting during the progress toward recovery.

TREATMENT.—The treatment of this disease, as inculcated and generally adopted of late years in this country, is in striking contrast with that formerly pursued. The change consists—*first*, in the disuse of bloodletting and cathartics; and *second*, in relying mainly on the use of opium. Governed by the general principles which should regulate the employment of bloodletting in acute inflammations, the indications for this measure are not present in peritonitis, a disease which tends to destroy life, sometimes very quickly, by asthenia. Bloodletting has been much employed in the treatment of this disease, and clinical observation has abundantly shown that it is not, to say the least, a curative measure. It is a measure which must do either good or harm in a disease like this, involving always not a little danger to life; and if it do harm instead of good, the amount of harm, even if not large, may bear heavily on the result. Cathartics also have been much employed in the treatment of this disease. Not only are they devoid of curative efficacy, but they are pernicious. An important point in the treatment of an acute inflammation of any part is to secure for the part as complete rest as possible. The friction incident to the peristaltic movements tends to prolong and increase the inflammation in peritonitis. An object of treatment is the arrest of these movements, thus securing rest for the inflamed membrane. Cathartics not only conflict with the means for effecting this object, but they aggravate the inflammation by increasing the peristaltic movements. Moreover, they are objectionable on the same score as bloodletting and other depletory and depressing measures. Clinical observation furnishes no evidence of the usefulness of cathartics in the treatment of acute peritonitis. On the other hand, it has occurred to me, as to others, to see the intensity of the inflammation reproduced by the too early administration of a cathartic when the disease appeared to be progressing favorably, the patient's life apparently being lost in consequence. Cathartics, then, should not enter into the treatment of this disease. The bowels should be allowed to remain constipated, if evacuations do not spontaneously occur, during the career of the disease, even if this period be a week or more. In pursuing this course the physician must expect in some cases to encounter the importunities of the patient or friends for medicine to open the bowels. If the patient suffer from an accumulation of feces within the rectum, its contents may be cautiously removed by repeated simple injections. Much relief, if tympanites be great, is sometimes obtained by the introduction into the rectum of a flexible tube, which allows the escape of gas.

An important object effected by the use of opiates is the arrest of the peristaltic movements; and this is a special object in this disease, but opiates are also useful, as in other inflammations, by rendering the system more tolerant of the local affection. The latter is also an object in the treatment. With reference to both these objects opiates are to be given in doses sufficient to produce certain effects. Relief of abdominal pain is evidence that the first object is effected. The doses, therefore, should be sufficient to produce this effect. The pain is to be relieved as quickly as possible, and the return of pain prevented by repeating the required doses at proper intervals. The success of the treatment will depend much on its being begun early in the disease. Time should not be wasted by delay for any preliminary measures. The treatment should begin with a full dose, the amount being regulated by the intensity of the pain. For an adult a quarter or a half of a grain of a salt of morphia or an equivalent dose of some other form of opiate may be given at once. In two or three hours the effect of the first dose can be appreciated. If the patient be not then free from pain, another dose is to be given, the dose being increased or diminished according to the degree of pain. And during the course of the disease the opiate is to be repeated, if required, at intervals, in doses sufficient to prevent return of pain. Narcotism is to be avoided. In order to avoid this the physician is to observe the effects of the doses as regard somnolency, contraction of the pupil, and diminished number of respirations. Judgment and careful watching are important, lest narcotism be produced. The somnolency from the opiates should never become so great that the patient cannot be easily aroused. The respirations should not be reduced below twelve per minute. If, as not infrequently happens, they are found below this number or irregular, the intervals between the doses are to be lengthened or the doses diminished. In carrying out this method of treatment it is desirable that the physician should see the patient frequently. If this be impracticable, as it often is in country practice, the administration of the doses must be entrusted with minute instructions to an intelligent and faithful assistant or nurse. The doses are to be gradually diminished in proportion as the local and general symptoms denote improvement.

The extent to which opiates are to be given in order to secure the objects of the treatment will vary much in different cases. Other things being equal, the doses required will depend on the degree of pain; but the susceptibility of different persons to opium varies greatly, and in peritonitis, as in some other diseases, there is sometimes an extraordinary tolerance of this remedy. Thus, in one of the cases treated successfully by Prof. Alonzo Clark the patient took within the first twenty-six hours, of opium and the sulphate of morphia, a quantity equivalent to 106 grains of opium; in the second twenty-four hours she took 472 grains; on the third day, 236 grains; on the fourth day, 120 grains; on the fifth day, 54 grains; on the sixth day, 22 grains; and on the seventh day, 8 grains, after which the treatment was suspended. This patient, Prof. Clark had reason to believe, was not accustomed to the use of opium in health, and was not intemperate. As regards the amount of opium required, this case is exceptional, the patient having an enormously increased tolerance of this drug. Rarely is more than half a grain of a salt of morphia every two, three, or four hours needed. Often the requisite effects are produced by smaller doses. If there be irritability of the stomach, the opiate should be given by hypodermic injections. Vomiting conflicts with the great object for which opiates are given—namely, rest of the inflamed part, and therefore everything is to be avoided which will be likely to excite it.

In the TREATMENT of this disease the main reliance is upon opiates; but other measures are important. Quietude of the body is to be enjoined, and the bladder is to be relieved by the timely use of the catheter if there be retention of urine. Rubefacients, sinapisms, or turpentine stupes may be applied over the abdomen. Blisters are contraindicated. All the good to be done in the way of revulsion will be effected by rubefacients, and counter-irritation beyond this does harm by increasing constitutional disturbance and contributing to depress the powers of life. Warm fomentations to the abdomen, if grateful to the patient, are useful; poultices are uncomfortable from their weight, and the water-dressing or the spongio-piline is to be preferred. I cannot speak of the utility of cold applications from much observation, but it is safe to trust to the feelings of the patient in deciding between cold and warm applications. Cold may be applied by means of compresses dipped in ice-water and renewed at intervals of a few minutes, or the ice-bag. Sustaining measures are indicated in proportion as the symptoms denote failure of the vital powers. Alcoholic stimulants and concentrated nourishment constitute the supporting measures which are not less appropriate to this disease than to other diseases tending to destroy life by asthenia. I have seen cases in which life apparently was saved by the free and persevering use of alcoholic stimulants in conjunction with the use of opiates. The employment of cathartics in convalescence calls for great circumspection; and they may not be required, the bowels acting spontaneously. The liberal use of opiates continued throughout the disease does not appear to conduce to constipation, and it does not prevent the occurrence of diarrhœa in a certain proportion of cases. Quinia in full doses as an antipyretic remedy is indicated if there be hyperpyrexia.

As an addendum to the treatment of this disease may be offered a suggestion relating to measures which doubtless are too bold to commend themselves without having been carefully considered. These measures are, making an opening through the abdominal wall, washing out the peritoneal cavity by injecting a tepid disinfecting liquid, and maintaining the opening so as to secure effectual drainage. These measures of course are applicable only to cases in which the presence of septic, infective morbid products may be inferred. In many instances these products probably constitute the chief source of danger. Modern experience in ovariotomy and in the operation of laparotomy in cases of intestinal obstruction has shown the admirable results which follow cleansing of the peritoneal surfaces, and the absence of the great danger formerly attributed to laying open the abdominal cavity. The time may come when paracentesis abdominalis for the purposes just stated will have been proved to be as applicable to certain cases of peritonitis as are thoracentesis, injections into the pleural cavity, and a permanent opening in the chest to cases of suppurative pleurisy.

The foregoing paragraph was contained in the last edition of this work, published in 1881. Since that date several cases have been reported in which the abdomen was opened, the purulent contents washed out, and antiseptic injections employed. Dr. Z. B. Adams reported a case for the *Boston Medical and Surgical Journal*, Nov. 20, 1884, in which this practice was successfully pursued. A successful case has been reported to me in a letter by Dr. Eug. A. Crouse of Iowa. Leyden and others in Germany advocate the treatment of suppurative peritonitis after the manner of empyema, and illustrative cases are cited.[1]

Circumscribed Peritonitis.

Inflammation limited to a circumscribed portion of the peritoneum is almost invariably a complication of a prior affection of the parts covered by the inflamed portion of the membrane. Circumscribed peritonitis may be acute or chronic, but these varieties cannot be sharply separated from each other. When inflammation reaches the surface of an organ covered by peritoneum the latter always becomes inflamed. Hence, a local peritonitis is frequent in various lesions of the stomach, intestine, liver, gall-bladder, spleen, urinary bladder, and the uterus and its appendages. Ulceration of the stomach leads to a peritonitis limited to the site of the ulcer. Cirrhosis of the liver in the great majority of cases is accompanied by inflammation of the serous investment of this organ. This inflammation is called perihepatitis. It leads to thickening of the hepatic capsule and to the formation of adhesions with the diaphragm and other adjoining parts. A similar perihepatitis often accompanies abscess, cancer, and some other affections of the liver. Perisplenitis signifies a local peritonitis about the spleen and is not infrequently found at post-mortem examinations. Among the most frequent and important forms of circumscribed peritonitis is inflammation of the pelvic peritoneum in females—pelvic peritonitis, perimetritis, peri-oöphoritis. Intestinal ulcers and typhlitis often lead to circumscribed peritonitis. In consequence of adhesions a perforation of the intestinal canal may give rise to only partial peritonitis. Encysted collections of pus may be present in the peritoneal cavity, without diffuse peritonitis. Diaphragmatic pleuritis may be accompanied by inflammation of the adjacent peritoneum.

The inflammatory products in circumscribed peritonitis are the same as in the diffuse form. The exudation in many instances is chiefly fibrinous. In some chronic cases resulting in formation of new connective tissue it is probable that from the onset there is simply a new growth of connective tissue without any free exudation. The perihepatitis accompanying cirrhosis of the liver is usually of this simple proliferative form. The peritoneal thickening and fibrous adhesions which result from circumscribed peritonitis may cause displacements and deformities of the viscera. Displacements of the uterus and of the ovaries thus induced are frequently met with in gynæcological practice. Circumscribed hemorrhagic peritonitis will be considered in the following article.

The SYMPTOMS of circumscribed peritonitis are pain and tenderness within a space corresponding to the extent of the inflammation, accompanied perhaps by more or less febrile movement. These symptoms are marked in proportion to the acuteness of the inflammation, but circumscribed peritonitis is oftener subacute or chronic than acute. Physical exploration in some cases furnishes diagnostic evidence. The presence of fibrin may occasion a friction sound or a tactile sensation of rubbing due to the attrition of opposed surfaces by the diaphragmatic respiratory movements. This sign is most likely to be obtained when the peritonitis is situated over the liver and spleen. It is obtained in cases of circumscribed oftener than of diffuse peritonitis, and is of comparatively less importance in the latter, because the symptoms generally suffice for the diagnosis if the inflammation be diffuse.

It must be difficult, if not impossible, to determine the existence of circumscribed peritonitis not connected with an affection of the parts covered by the inflamed portion of peritoneum. Cases, however, are so rare that, the existence of circumscribed peritonitis being ascertained, an affection of the parts covered by the inflamed membrane is to be inferred.

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[1] Vide *Verhandlungen des Vereins für innere Medicin zu Berlin*, 1884; also, *Am. Journ. of Med. Sciences*, April, 1885, p. 573.

The PROGNOSIS in cases of circumscribed peritonitis will depend on the nature and extent of the affection of which the peritonitis is a complication. The adhesions resulting from the inflammation may be either conservative or deleterious.

So far as the peritonitis is concerned, palliative treatment is alone called for.

Chronic Peritonitis.

Chronic peritonitis may be of the following varieties: 1, with exudation of serum, fibrin, and pus, as in acute peritonitis; 2, chronic serous peritonitis; 3, peritonitis with predominant production of connective tissue; 4, hemorrhagic; 5, tuberculous; 6, cancerous.

Chronic peritonitis with exudation of serum, fibrin, and pus may follow an acute peritonitis or may develop slowly and insidiously. It is more frequent as the sequel of an acute peritonitis than in a latent form, but it is in either case a rare variety of the disease. There is sometimes, but not always, coincident disease of some of the abdominal viscera. In consequence of the formation of membranous adhesions the fluid part of the exudation often becomes sacculated. Sometimes the exudation consists almost wholly of fibrin, by which the intestines and other viscera may be agglutinated to each other and to the parietal peritoneum. The intestinal walls are softened and easily torn. Chronic peritonitis with an exudation almost wholly purulent sometimes develops insidiously. When acute peritonitis passes into a chronic state the pain, tenderness, and tympanites diminish, but they do not wholly disappear. There are usually recurring febrile paroxysms with intervals of apyrexia. Gastric and intestinal disorders are present. The patient is troubled more or less by vomiting. The bowels are irregular, constipation alternating with diarrhœa. The patient emaciates and becomes anæmic. Sometimes irregularities due to encysted collections of fluid in the peritoneal cavity may be made out by inspection, palpation, and percussion. When the disease begins insidiously it is usually not susceptible of diagnosis. The prognosis is unfavorable, but recovery may take place by absorption and by the formation of adhesions. Collections of pus may ulcerate and escape into the bowels or externally.

Chronic serous peritonitis is usually secondary to ascites. Together with the accumulation of a large amount of serous fluid in the peritoneal cavity there are evidences of inflammation. The serous fluid may be rendered somewhat turbid by the admixture of pus-cells. In some cases there is fibrinous exudation upon the peritoneal surface; and in others the chronic inflammation is manifested by fibrous thickening of the peritoneum, either diffuse or in the form of nodules. The nodules may resemble tubercles in their gross appearance. These various appearances may be combined. Peritonitis is not a frequent complication of ascites. It is more likely to develop after repeated tappings than when paracentesis has not been performed. In very rare instances chronic serous peritonitis is an idiopathic affection. Cases of this nature were formerly described as idiopathic dropsy of the peritoneum, but it is probably more correct to regard them as inflammatory in origin. A serous fluid accumulates, as in ascites; but none of the recognized causes of ascites are present, such as obstruction of the circulation in the liver, in the lungs, or in the heart, or as Bright's disease, hydræmia, tuberculosis, or cancer. In most cases there is either a free inflammatory exudation or a formation of new connective tissue in the peritoneum; but there is sometimes no more sign of inflammation than in an ordinary case of ascites.[1] Peritonitis complicating ascites may be suspected when the symptoms of the former disease, which have been described, develop; but it rarely happens that these symptoms are manifested with sufficient intensity to be diagnostic. It is very difficult to distinguish idiopathic chronic serous peritonitis, on the one hand, from ascites, and, on the other hand, from tuberculous peritonitis. This difficulty is increased by the rarity of the disease, which in a given case raises a strong presumption against its existence. A probable diagnosis can be reached only by excluding the symptoms and causes of the two diseases in question. In ascites due to that large group of causes occasioning obstruction to the circulation the spleen is almost invariably enlarged, while in the disease under consideration this organ is not affected. Serous peritonitis may occur in chronic alcoholism, as pointed out by Leudet.[2]

Peritonitis with predominant production of connective tissue may be called *proliferative peritonitis.* The new connective tissue is in the form either of adhesions or of thickening of the peritoneum. The two alterations may be combined. This form of chronic peritonitis may develop without any production of fibrin or of pus, or a moderate amount of these products may be present. Proliferative peritonitis with adhesions as the chief lesion is generally circumscribed. This has been mentioned in the preceding article. Peritonitis with fibrous thickening of the peritoneum is sometimes observed in connection with cirrhosis of the stomach. The serous covering of the stomach and intestine, the omentum, the mesentery, and the mesocolon are sometimes enormously thickened by the production of fibrous connective tissue. The peritoneum in other situations may also be thickened. The omentum, mesentery, and mesocolon are usually retracted. The coils of intestine may be inextricably united to each other by the new growth of tissue. Not only is the cavity of the stomach encroached upon, but the lumen of the intestine may also be diminished. There is usually serous or sero-purulent liquid in the peritoneal cavity, and there may be fibrin upon the thickened peritoneum. Chronic inflammation of the gastro-intestinal mucous membrane is generally present. This form of peritonitis is comparable to the chronic interstitial inflammation of parenchymatous organs. The symptoms point to some severe abdominal trouble. The symptoms of chronic gastro-enteric inflammation are often present. Vomiting, diarrhœa, dull pain, and emaciation may be mentioned as frequent symptoms. The distinction often cannot be made from tuberculous peritonitis or from cancer of some of the abdominal organs. The same induration of the peritoneum and of the abdominal organs which may be felt through the abdominal walls is often present in tuberculous peritonitis.

Hemorrhagic peritonitis may be either diffuse or circumscribed. Reference is not had in this connection to a simple staining of the inflammatory products with blood, such as often occurs in cancerous and tuberculous peritonitis, but to a special form of inflammation characterized by the development, upon the peritoneum, of successive thin layers of connective tissue, rich in wide capillaries with thin walls, which readily rupture and give rise to large and small hemorrhages. The new membrane is reddish or brown in color, from infiltration with blood. Extensive extravasations of blood form hæmatomata. This peculiar form of peritonitis is analogous to chronic hemorrhagic pachymeningitis, which will be described in the next section of this work. Friedreich has observed two cases of diffuse hemorrhagic peritonitis in patients with ascites who had been repeatedly tapped. The circumscribed form was first described by Virchow. It is met with especially in the recto-uterine fossa, where it causes bloody tumors known as retro-uterine or pelvic hæmatoceles. A similar hæmatoma may occur in the recto-vesical fossa in males. The prognosis in pelvic hæmatocele is good, the blood being often absorbed with astonishing rapidity. The consideration of this form of disease belongs to works on gynæcology. There are no symptoms diagnostic of diffuse hemorrhagic peritonitis. Cornil and Ranvier mention the occurrence both of diffuse and of circumscribed hemorrhagic peritonitis in hypertrophic cirrhosis, in articular rheumatism, in tuberculosis, and in Bright's disease. It is a rare form of inflammation.

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[1] In ascites the peritoneum is often thickened, and even when no changes are visible to the naked eye it is not perfectly normal. The endothelial cells are changed in size and in shape, and present evidences of proliferation (Delafield). Similar changes exist in the pleura in hydrothorax and in the tunica vaginalis testis in hydrocele.

[2] "Des Lésions du Péritoine chez les Alcoolisés," *Gaz. hébdom.*, 1879, No. 49.

Tuberculous peritonitis, of all the forms of chronic diffuse peritonitis, is the most common and of the greatest clinical importance. In acute miliary tuberculosis the peritoneum, especially the omentum and the serous covering of the liver and of the spleen, is studded with small, gray miliary tubercles. This acute tuberculosis of the peritoneum is accompanied usually by little or no serous effusion. It is not attended by recognizable symptoms, and is not included under the heading of tuberculous peritonitis. So, too, the eruption of miliary tubercles, combined often with local inflammation, on the peritoneum, covering the site of tuberculous ulcers of the intestine, is of pathological rather than of clinical interest.

The most frequent form of tuberculous peritonitis is characterized by abundant production of firm connective tissue. This new tissue causes thickening of the serous membrane and intimate adhesion of coils of intestine and other abdominal organs to each other and to the abdominal walls. The intestinal coils are inextricably matted together. The omentum is drawn up into a thick, hard mass which can sometimes be felt during life as a transverse band above the umbilicus. The mesentery and mesocolon are likewise thickened and retracted. Tubercles are present everywhere in the thickened peritoneum and in the adhesions. They appear as translucent miliary granulations, as yellow caseous deposits, and as fibroid nodules. Some fibrinous exudation is present. Cases differ much as to the quantity and the character of the effused liquid. The quantity may be small, but more frequently it is abundant, so that during life hydro-peritoneum is simulated. In the majority of cases the liquid consists of turbid, blood-stained serum. A hemorrhagic effusion is common in tuberculous inflammation of all serous membranes. Fresh ecchymoses and old pigment-stains are often present in the fibrous tissue, especially near the tubercles. The fluid part of the exudation is frequently shut into separate cavities by means of the extensive adhesions. Encapsulated collections of pus are found not very infrequently. These, as well as softened tubercles, may lead to ulcerations into the intestinal canal from without. The adherent coils of intestine may also ulcerate into each other. Even fecal fistulæ, opening through the abdominal parietes, may be formed.

In another form of tuberculous peritonitis there are few or no adhesions; there is an abundant collection of somewhat turbid, and often bloody, serum; and the peritoneum, which may be thickened, is studded with a large number of tubercles which may coalesce into the form of nodules and plaques as much as an inch in diameter. This form has been called tuberculous ascites.

Still less frequent is a third form described by Wilks and Moxon under the name of latent peritoneal tubercle. The peritoneum is studded with tubercles in the midst of a soft, gelatinous, fibrinous exudation by which its surfaces adhere to each other.

Tuberculous peritonitis in extremely rare instances may be a local affection. It is usually accompanied by tubercles in other parts of the body. As a rule, there is pulmonary tuberculosis, but cases are not very infrequent in which the pulmonary lesions are comparatively trifling, and there may be no pulmonary tuberculosis whatever. Tuberculous pleurisy is a very frequent accompaniment of tuberculous peritonitis. Primary tuberculosis of the genito-urinary organs—in females of the uterus and Fallopian tubes, in males of the epididymis and testicle, also of the bladder, ureters, and kidneys—has been repeatedly followed by tuberculous peritonitis. The mesenteric glands, the spleen, and the liver often contain tubercles in all forms of this disease. There may be intestinal ulcers and waxy degeneration of various organs. Cirrhosis of the liver has been repeatedly observed associated with chronic tuberculous peritonitis. Tuberculous peritonitis may occur at any age. It is rarer before the fourth and after the fortieth year of age than at other periods.

As regards clinical history and diagnosis, tuberculous peritonitis is an obscure disease. It may begin acutely or may be attended by acute exacerbations, but as a rule it is insidious in its onset and slowly progressive in its course. There is usually moderate fever, with more or less acceleration of the pulse. Vomiting and other digestive disturbances are more or less prominent. Diarrhœa alternates with constipation. Other general symptoms are—night-sweats, hectic paroxysms, progressive emaciation, anæmia, and general debility. As regards local symptoms, abdominal pain and tenderness may be wanting, and they rarely are prominent symptoms. Increasing distension of the abdomen by fluid and by meteorism may be the first symptom to attract attention. It may be possible to make out encysted collections of fluid in the abdomen and tumors formed by agglutinated coils of intestine. The detection of the thickened and retracted omentum is a significant sign.

The DIAGNOSIS of tuberculous peritonitis is not easy. When there is a large accumulation of fluid the differentiation from ascites is difficult. In the diagnosis the following points are in favor of tuberculous peritonitis: the presence of tubercles in the lungs and in other organs; the simultaneous occurrence of pleurisy; no notable decrease in the size of the liver; no enlargement of the spleen; the presence of some fever and abdominal tenderness; no history of intemperance; no marked collateral dilatation of the abdominal veins; a large quantity of albumen in the fluid; and the recognition of some of the local signs mentioned, such as the contracted omentum. The distinction of tuberculous from simple chronic peritonitis is all the more difficult from the fact that both may occur in connection with chronic pulmonary tuberculosis. In suspected cases the fluid withdrawn by tapping may be examined for tubercle bacilli, but failure to find these would not exclude tuberculous peritonitis.

The termination of tuberculous peritonitis is always fatal, either from the disease itself or from tuberculosis of the lungs or other organs. The duration is from one or two months to a year or more.

Of the tumors of the peritoneum, *carcinoma* is the only one which demands clinical consideration. The occasional occurrence of echinococcus cysts in the peritoneum may be simply mentioned. Carcinoma of the peritoneum is almost always secondary. Colloid cancer is probably the only form which develops primarily in this situation. It occurs as a diffuse growth, involving the omentum and other parts of the peritoneum. Colloid cancer has its seat by preference in the peritoneum, and it attains sometimes an enormous size, a growth weighing one hundred and fourteen pounds having been reported. Peritoneal cancer may develop as secondary to cancer of the ovary, uterus, testicle, retro-peritoneal glands, rectum and other parts of the intestine, stomach, liver, pancreas, etc. It may appear in the form of one or more large tumors in the serous membrane, or there may be countless little cancerous nodules studding the peritoneum. The latter distribution is called miliary carcinosis of the peritoneum. By the aid of the microscope it is readily distinguished from tuberculosis, which it resembles in gross appearance. Peritoneal carcinoma is not necessarily accompanied by inflammation. Usually, however, there is considerable accumulation of brownish serum which may contain fibrin. The brown color comes from admixture with blood. A new growth of connective tissue, with agglutination of intestinal coils, may accompany cancer as well as tubercle of the peritoneum. In cancerous peritonitis the local symptoms do not differ materially from those in tuberculous peritonitis. Febrile movement is less constant and marked in the former, and perspiration is not so liable to occur. The age of the patient is greater than in the majority of cases of the latter. When the primary cancer is recognized, the diagnosis of cancerous peritonitis does not usually involve much difficulty. When, however, the primary growth is latent the diagnosis is not easy. The recognition of tumors seated in the peritoneum is of assistance in diagnosis. The examination of the fluid withdrawn by tapping may also be of assistance. It has been pointed out by Foulis and by Thornton[1] that in cancer of the peritoneum or in cancer of the ovary, with ascites, the fluid in the peritoneal cavity often contains large cells in groups (budding cells, group-cells). The prognosis is fatal.

As regards the TREATMENT of the different varieties of chronic peritonitis, the objects are, to palliate the local symptoms, and by tonic remedies, nutritious diet, and hygienic influences to enable the system to tolerate the disease as long as possible.

Hydro-peritoneum—Ascites.

The term hydro-peritoneum or ascites denotes peritoneal dropsy. The fluid accumulated in the peritoneal cavity consists of clear serum with the properties described in Part I. (p. 33) as belonging to transudations. Like other dropsies, hydro-peritoneum is a symptom or an effect of disease, not strictly a disease in itself. It is, however, convenient and proper to give separate consideration to ascites, as the accumulation of liquid in the peritoneal cavity may be the first and chief manifestation of disease, and as it occasions inconvenience and danger, to which therapeutical measures are specially directed. Hydro-peritoneum may occur either as a local dropsy or as part of a general dropsy; that is, in conjunction with anasarca and with dropsical accumulations in other serous sacs.

With very few exceptions, ascites, occurring as a local dropsy, is the result either of disease of the peritoneum or of obstruction in the main trunk of the portal vein or in the branches of this vessel within the liver. In most if not all cases in which the ascites is attributable to disease of the peritoneum the fluid accumulation is to be regarded as an inflammatory effusion rather than as a transudation. This is proven by the presence of a certain degree of inflammation in these cases, and by the fact that the fluid, in the amount of albumen which it contains, is, as a rule, more closely allied to exudations than to transudations. The inflammation is often no more than a chronic thickening of the peritoneum, either diffuse or in the form of nodules, and it may not be attended by the exudation of fibrin. This form of peritonitis has been described in the preceding article as chronic serous peritonitis. Tubercles and cancer of the peritoneum may also be causes of ascites, even when marked inflammatory changes are absent; but here, too, the serous accumulation is to be attributed to inflammatory alterations, which careful examination will usually reveal. It is a rule that slight inflammatory changes in the vascular walls are attended by an effusion chiefly serous. Obstruction of the main trunk of the portal vein, as a result of thrombosis or pylephlebitis, in most cases is followed by ascites. Cirrhosis is the most common morbid condition, seated in the liver, which causes ascites. Syphilitic hepatitis is also a cause of ascites. Echinococcus, abscess, cancer, and other tumors of the liver may cause ascites by compressing the portal vein or its principal branches. Atrophy of the liver induced by external pressure or by growths in its substance may likewise be a cause. Advanced amyloid degeneration of the liver is accompanied by ascites as a rule. These various diseases of the liver will be considered in the following chapters. With the possible exception of amyloid liver, they all cause ascites by interfering with the portal circulation through this organ.

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[1] *British Med. Journ.*, 1878.

The causes of hydro-peritoneum in conjunction with general dropsy are obstruction to the circulation in the lungs and through the heart and a hydræmic state of the blood. The most important of the pulmonary causes are emphysema and fibroid induration and contraction of the lungs, such as may follow chronic pleurisy. Uncompensated valvular lesions and other organic diseases of the heart are frequent causes of ascites with general dropsy. Bright's disease is by far the most important cause of ascites referable to hydræmia; but other cachexiæ, as the tuberculous, the cancerous, the syphilitic, and the malarial, may also cause hydræmic dropsy. The connection between hydræmia and dropsy has been discussed in Part I. (p. 63 *et seq.*). From the foregoing it is evident that dropsy of the peritoneum, as of other parts, may be divided into mechanical dropsy and cachectic dropsy (p. 33). Ascites due to mechanical causes (obstructions to the circulation in the liver, lungs, or heart) reaches a higher degree than that due to hydræmia.

Ascitic fluid consists, as a rule, of yellowish serum. In cachectic peritoneal dropsy it is clear and watery in appearance. In cirrhosis of the liver it is darker in color. It may be stained with blood, especially in tuberculosis and in cancer of the peritoneum. The specific gravity is rarely below 1004 and seldom exceeds 1020. The percentage of albumen is less than in exudations. In dropsical accumulations in the peritoneal cavity the quantity of albumen is usually less than 2½ per cent., whereas in inflammatory exudations in the same situations the quantity generally exceeds 4 per cent. In cachectic ascites the amount of albumen rarely exceeds 1 per cent., whereas in ascites due to tuberculosis of the peritoneum the quantity is, as a rule, greater than 2½ per cent. Chemical examination of fluid removed by tapping may thus be of assistance in diagnosis. Ascitic fluid contains fibrinogen, and (as explained on p. 34) it often coagulates spontaneously when withdrawn from the body. At autopsies ascitic fluid is by no means always found devoid of coagulated fibrin. Since Schmidt's investigations as to the coagulation of fibrin it has not been found possible to draw as sharp a distinction between fluids free from fibrin and those containing this substance as was formerly done.

The transudation may present a milky appearance from an admixture of chyle in consequence of compression or of rupture of the thoracic duct or large chyle-vessels. This condition is called chylous ascites (hydrops chylosus.) A similar appearance may be due to the presence of a large number of fat-granules and of cells containing molecules of fat. This constitutes hydrops adiposus. It has been observed in connection with cancer and with tubercle of the peritoneum. In one case of chylous ascites which followed residence in a tropical climate Winckel found in the fluid countless parasites resembling the Filaria sanguinis detected by Lewis in the blood of those suffering from chyluria.[1]

In the account of the clinical history of ascites reference is not had to slighter transudations, such as occur in cachectic states, and which, as a rule, neither give rise to symptoms nor call for therapeutical interference.

The effusion of liquid and its accumulation take place without pain, tenderness, or any local subjective symptoms. The enlargement first directs attention to the abdomen. In the majority of cases it goes on with rapidity, and the abdomen soon becomes considerably or greatly distended. The symptoms referable directly to the dropsy proceed from the mechanical pressure of the liquid and are in proportion to the quantity of the effusion. The distension of the abdominal walls by the liquid and its weight occasion more or less inconvenience. The functions of the abdominal organs (stomach, liver, kidneys) are impaired by compression. Pressure on the veins within the abdomen gives rise to œdema in the lower extremities. The movements of the diaphragm are restrained or arrested, breathing being carried on by the costal muscles, and if the accumulation be very large the capacity of the chest is diminished by the upward pressure. The appetite is usually impaired. A sense of fulness is frequently felt after taking food in moderate quantity. Vomiting and diarrhœa are occasional symptoms.[2]

Enlargement of the superficial veins on the anterior aspect of the trunk is a consequence of the passage of a portion of the blood from the portal vein into the systemic venous system through anastomosing branches. The superficial veins of the abdomen in some cases, of the chest in other cases, and sometimes in both situations, become more or less dilated, presenting a varicose appearance. Hernial protrusions at the umbilicus and in other situations are occasionally produced.

When the fluid accumulation is considerable, œdema of the lower extremities is a pretty constant symptom, the amount of œdema varying much in different cases. The limbs, scrotum, and penis sometimes become enormously swollen. Some œdema of the lower limbs preceded an enlargement of the abdomen sufficient to attract the attention of the patient in one-half of the cases (21) among those analyzed by me, the histories of which contained information on this point. Œdema of the face and upper extremities does not belong to the clinical history of local hydro-peritoneum. If present, it usually denotes coexisting renal or cardiac disease. In the progress of the affection emaciation and pallor become marked. The attenuation of the upper part of the body, the distended abdomen, and the œdematous lower extremities combine to render the appearance highly characteristic of the affection. Cholæmia or icterus is occasionally present. It occurred in 7 of the 46 cases which I have analyzed.

Fever does not attend the progress of this affection. The pulse may be more or less increased in frequency and proportionately feeble, or it is more or less enfeebled without acceleration. The urine is frequently scanty. The mind is usually intact, but in a certain proportion of cases, toward the close of life, delirium, convulsions, and coma occur. These symptoms may be due to uræmia or to retention in the blood of the excrementitious principles of the bile (cholesteræmia).

The mode of dying in the great majority of cases is by slow asthenia. A rapid and large accumulation of liquid may destroy life by interference with respiration; and apnœa and asthenia are combined when coma precedes death.

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[1] Winckel, *Deutsches Archiv für klin. Med.*, 1876, Bd. 17, p. 303.

[2] Vide paper by the author, entitled "Clinical Report on Hydro-peritoneum, based on an Analysis of Forty-six Cases," *American Journal of Medical Sciences*, April, 1863.

The PROGNOSIS in most cases is unfavorable. The cases in which a permanent recovery takes place are few. The morbid conditions on which the affection is dependent are generally incurable, and lead to its return, sooner or later, in most of the cases in which the dropsy disappears or is removed. The duration of the affection is variable. In the fatal cases of those which I have analyzed, the duration varied between six weeks and seventeen months, the average duration being more than five months. Cases in which the affection is attributable to obstruction of the portal vein by non-infecting emboli or by thrombi may end in recovery, changes in the obstructing thrombus or embolus taking place which permit return of circulation through the obstructed vessel. Instances apparently exemplifying this fact have come under my observation.

The DIAGNOSIS of hydro-peritoneum is generally made without difficulty, yet there is a liability to errors which may lead to serious results. A distended bladder has been punctured, the case being supposed to be one of peritoneal dropsy. There is a tradition that John Hunter made this mistake. On careful examination the tumor formed by the distended bladder may generally be felt through the abdominal walls, but the introduction of the catheter is the diagnostic test. The sacculated bladder may cause a notable anterior projection of the abdomen. The contents of the saccular appendage may generally be removed by the catheter combined with suction by means of an india-rubber bag. Pregnancy, the liquor amnii being unusually abundant, has been mistaken for dropsy and the uterus has been punctured. Careful examination through the abdominal walls and *per vaginam*, together with auscultation of the abdomen, should prevent this error. Great corpulence has led to error and to the operation of paracentesis, as in the famous case of "dry tapping" related in the lectures of Sir Astley Cooper. Finally, tympanites, large abdominal tumors, and ovarian cysts are to be discriminated from peritoneal dropsy.

The enlargement of the abdomen from dropsy begins at the lower part; and if the abdomen be not largely distended it is more marked below than above when the patient stands or sits. The enlargement on both sides is equal, and the abdomen is symmetrical. If the patient lie upon either side, the weight of the liquid causes the depending side to sag. Percussion on one side, the open palm being placed on the opposite side, frequently causes a characteristic shock, called the sense of fluctuation. Sometimes this sensation is more appreciable when the palm is placed near the point at which the percussion is made. The former method of percussion gives rise to what is called diametrical, and the latter to peripheral fluctuation; but a more satisfactory application of percussion is to compare the results when the patient is placed in different positions. Percussing first in the sitting or standing posture, tympanitic resonance, from intestinal gas contained in the intestine floating upon the liquid, is usually found at the upper part of the abdomen, extending below for a greater or less distance, and flatness from this point to the pubis. Placing the patient on the back, the change in the relative situation of the liquid and the intestine is shown by the greater extension of the tympanitic resonance toward the pubis. Similar proof of the presence of liquid is obtained by percussing upon either side of the abdomen successively, the body being inclined first to one side, and then to the other. The exceptional cases in which this test afforded by percussion is not available are those in which the intestinal coils are fixed by morbid adhesions. This is rare in cases of a purely dropsical affection.

The several affections simulating dropsy which have been named may generally be excluded by the application of percussion as just described.

Ovarian cysts of sufficient size to distend the whole abdomen are the most likely to be confounded with hydro-peritoneum. These are generally, however, distinguishable by the appreciation of the form of the cyst through the abdominal walls, the existence of a tumor on one side before it extended to the whole of the abdomen, the absence of symmetry in the enlargement of the two sides, together with the want of evidence of the presence of liquid in the peritoneal sac, afforded by percussion when the body is placed in different positions. The chemical and the microscopical examination of the fluid removed by puncture often assists in this differential diagnosis. When the fluid is thick and viscid it is ovarian. The thin ovarian fluids present more difficulty in diagnosis. The presence of a large quantity of a modified form of albumen, called paralbumen, is diagnostic of ovarian cysts. So, too, the recognition of a large number of granular corpuscles devoid of nuclei, in which the granules are insoluble in acetic acid and in ether, is diagnostic. These bodies are known as the ovarian corpuscles of Drysdale. The detection of a few of these corpuscles and the presence of a small quantity of paralbumen are not sufficient for the diagnosis. Ovarian fluids very rarely coagulate spontaneously. If cylindrical epithelial cells be found, the fluid comes from an ovarian cyst, but these cells are rarely discovered. The specific gravity of ovarian fluids is generally higher than that of ascitic.

The TREATMENT of hydro-peritoneum relates—*first*, to the dropsical effusion; and *second*, to the morbid conditions on which the effusion depends. As regards the dropsical effusion, the object is to effect its removal or diminution. For this object, medicinal and surgical measures may be employed. The medicinal means consist of remedies to eliminate water from the blood and thereby induce absorption of the effused liquid. The elimination of water from the blood is to be accomplished by diuretics and hydragogue cathartics. Diuretics in most cases effect but little. It is generally not easy to obtain much diuretic effect from any of the remedies of this class, the difficulty arising from the slowness with which the remedies enter the general circulation, owing to the fact that the morbid conditions giving rise to the dropsy usually involve obstruction to the portal circulation. Of 13 cases treated with diuretic remedies, in 8 no effect upon the dropsy was produced; in 5 more or less diminution of the dropsy followed; but in only 2 cases was the improvement marked and progressive. Little dependence, therefore, is to be placed on diuretics, but inasmuch as, if properly prescribed, they do not cause much disorder or prostration, they should be fairly tried. The saline and vegetable diuretics may be given in succession, and a diuretic effect is more likely to be produced if several be employed in combination.

Hydragogue cathartics act more efficiently than diuretics, but clinical observation does not furnish much evidence of success from their use. Of the different hydragogues, elaterium is the most reliable. I am accustomed to prescribe this remedy in doses of a quarter of a grain repeated, at short intervals until abundant liquid evacuations are produced. Afterward the doses may be repeated according to circumstances. Owing to the disturbance and prostration caused by the prolonged use of hydragogues, they are liable to do harm and should not enter largely into the treatment.

The apocynum cannabinum, or the Indian hemp indigenous in this country, is considered by some as an efficient hydragogue in this form of dropsy. From some trial of this remedy in Bellevue Hospital I have been led to think it is less reliable than the elaterium. The decoction of the root or the extract may be employed.

It is important to bear in mind that in order to secure the desired effect of diuretics or hydragogue cathartics upon the dropsy the ingestion of liquids is to be restricted, the patient drinking no more than the wants of the body require.

The surgical method of effecting the removal of the liquid is tapping. With respect to this operation the views heretofore held are that it is to be resorted to only when the abdominal distension occasions great distress or danger, and not until the indirect means have been thoroughly tried; that the dropsy generally increases more rapidly after the operation; and that the operation involves danger if the system be much prostrated. These views are erroneous. Tapping effects promptly, without perturbation and without impairing the vital powers, the object for which diuretics and cathartics are employed, the latter being generally ineffectual, disturbing the digestive functions, and enfeebling the powers of life. The operation is a trivial one, involving little risk of accidents or of peritoneal inflammation. A patient under my observation had repeatedly tapped himself, using as a trocar a blade of a pair of scissors, and a piece of an ordinary clay pipe-stem as a canula. There is no danger from increased rapidity of effusion directly after the operation. The patient is spared not only the inconvenience and distress, but the permanent injury caused by the prolonged pressure of the liquid upon the abdominal and thoracic viscera, and he is in a condition more favorable for the action of remedies other than those which have special reference to the removal or diminution of the dropsy. Clinical experience shows that in some cases, even when the dropsy is dependent on cirrhosis, the liquid does not accumulate for weeks, months, and years afterward. In another work I have cited several instances in which, after a great number of tappings, the dropsy ceased to return and recovery seemed to be complete.[1] It is judicious to resort to tapping as soon as the accumulation of liquid is sufficient to occasion much inconvenience, adopting this direct method in lieu of the indirect means, provided the latter do not prove immediately efficacious, and repeating the operation whenever the abdomen becomes again distended. These views were enunciated by me in a paper published in 1863, and were based on the results of tapping in 20 cases under my observation, the operation in these cases being performed but once in 11 cases, and in the remaining 9 cases being repeated from three to thirty times. The conclusions drawn from the facts contained in the histories of these cases are the following: 1. Tapping may be resorted to as a palliative measure when the condition of the patient is such that only temporary relief is to be expected. 2. In a certain proportion of cases the dropsy returns more or less quickly, and it may be necessary to repeat the operation many times. The repetitions, however, are innocuous. This was illustrated in one case in which it was performed thirty times within eighteen months. 3. In some cases the dropsy does not return for a considerable or even a long period, and in some cases it never returns. That it will prove a curative measure is not to be expected in the majority of cases. Since the publication of that paper the correctness of these conclusions has been substantiated by a large number of cases under my observation. If it be deemed advisable to avoid a rapid withdrawal of the liquid, it may be removed by aspiration. An aspirating needle, however, should not be used. My adaptation of Davidson's syringe is the most convenient aspirator.[2]

The dropsical collection is sometimes discharged spontaneously, the walls of the abdomen becoming attenuated by distension and rupture taking place. The perforation in these cases is most likely to occur at the umbilicus or at some point at the median line. In a case at Bellevue Hospital a protrusion of a cyst-like tumor took place at the umbilicus, which, after attaining a large size, burst, and the contents of the peritoneal sac were discharged through the opening. Four months after the spontaneous discharge took place the dropsical effusion had not returned.

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[1] Vide *Clinical Medicine.*

[2] Vide paper entitled "Early Tapping in Cases of Ascites," by the author, read at a meeting of the British Medical Association, in August, 1883, and published in the *British Med. Journal*, and in the *Journal of the Am. Med. Association*, Sept. 15, 1883.

The treatment relating to the morbid conditions on which the dropsy depends embraces, in the first place, measures to prevent an increase of the incurable lesion which exists in the majority of cases—namely, cirrhosis of the liver. It will be seen, when we come to consider this lesion, that it is caused by spirit-drinking. Change of habits as regards the use of spirits is therefore the most important of the measures coming under this head. In the second place, certain accessory or co-operative causative conditions may be removed—causes which act by impoverishing the blood and impairing the forces which carry on the circulation. This part of the treatment embraces tonic medication with nutritious diet and other measures to strengthen and invigorate the system. Well-directed treatment for this end, although not curative, will often do much to prolong life and to secure as much improvement of health as is compatible with existing structural lesions.

Mechanical compression of the abdomen after tapping, by means of a swathe or a laced supporter, is a measure of importance, as not only affording comfort, but tending to prevent a renewal of the dropsical effusion.

CHAPTER XI.

DISEASES OF THE LIVER.

Suppurative Hepatitis.—Chronic Interstitial Hepatitis (Cirrhosis).—Syphilitic Hepatitis.—Acute Yellow Atrophy of the Liver.

OF diseases affecting the solid abdominal viscera, the greater number and the more important are seated in the liver. These will be first considered, and afterward diseases of the spleen and pancreas. Acute diffuse hepatitis, considered as a disease distinct from acute yellow atrophy of the liver, has so little practical interest, except in certain tropical climates, that it is not considered in the present edition of this work. Suppurative hepatitis or hepatic abscess, chronic interstitial hepatitis, syphilitic hepatitis, and acute yellow atrophy of the liver will be treated of in this chapter, and the next chapter will embrace the different structural together with functional affections.

Suppurative Hepatitis—Abscess of the Liver.

Anatomical Characters.—Suppurative hepatitis is always circumscribed and leads to the formation of abscess. There may be only one abscess, which may attain a very large size, so as to occupy the greater part of the liver. Such large solitary abscesses are found especially when the disease is of traumatic origin or when it occurs in tropical climates. They are sometimes called tropical abscesses. There may be multiple abscesses, varying in number between three and forty or more. When very many the individual abscesses are usually of small size. In a case under the author's observation five and a half quarts of pus were at once evacuated by an opening made through the thoracic walls. The patient recovered. An abscess has been found to contain after death eighteen pints of pus, the entire organ except the left lobe being transformed into a mere sac. The abscess may be situated at any part of the gland. It is oftener seated in the right than in the left lobe, and oftenest at the posterior and superior portion of the right lobe.

We may assume that abscesses in the liver, as well as abscesses elsewhere, are caused by the invasion of micro-organisms, such as have been considered as the cause of suppuration in Part I. p. 81. In most cases, and probably by proper methods in all, it is possible to demonstrate the presence of bacteria in hepatic abscess. In the majority of cases, at least in temperate climates, the bacteria enter the liver by the blood-current, either through the portal vein or the hepatic artery. They lodge within the capillaries of the liver, where they usually form zooglœa filling up a certain number of capillaries. The hepatic cells adjacent to these capillaries become cloudy, lose their nuclei, and disintegrate. In this way necrotic foci are produced which appear to the naked eye as grayish or yellowish-gray spots embracing one or more hepatic lobules. Around these areas of necrosis there is an emigration of white blood-corpuscles, which appear first in the interlobular connective tissue, rarely in the centre of the lobules. This accumulation of pus-cells is accompanied by the breaking down and liquefaction of the necrotic tissue. The result is the formation of an abscess-cavity. Small abscess-cavities so produced may coalesce and make larger cavities. As a rule, the longer the duration of the disease the smaller the number and the larger the size of the abscesses.

The purulent matter from an hepatic abscess often does not differ from ordinary pus, but it may contain liver-cells and shreds of necrotic tissue. It is not infrequently reddish or brownish in color from extravasated blood, or it may be stained with bile when the abscess communicates with one of the bile-ducts. Sometimes the abscess contains few or no recognizable pus-cells, but instead *débris* composed of fatty and albuminous granules, pigment-particles, bacteria, and perhaps shreds of dead tissue. The walls of the abscess at first consist of exposed hepatic tissue more or less discolored, ragged, and infiltrated with pus-cells; but after a time a wall of connective tissue is formed which increases in thickness and firmness with age. Sometimes the abscess-wall becomes gangrenous, especially when air enters the abscess or when the abscess is secondary to gangrene in some part of the body. An instance of this is reported in the author's *Clinical Medicine.*

If the contents of an abscess be discharged, its walls may cease to secrete pus and cicatrization may ensue. The pus in small abscesses may become thick and dry and remain innocuous, or an abscess may become enveloped with extremely firm fibrous tissue and cease to grow; but as a rule, if life continue, the pus makes its way, as in other abscesses, in the direction in which the pressure is least, and is at length discharged in some cavity, canal, or outlet.

When the abscess reaches the surface of the liver a circumscribed inflammation of its serous covering takes place. This, as a rule, leads to adhesion between the surface of the liver and the diaphragm, the abdominal wall, or some neighboring viscus.

In the larger proportion of cases the evacuation is through the thoracic or the abdominal parietes. When it takes this direction a fluctuating tumor after a time makes its appearance, caused by the accumulation of the pus beneath the integument. The tumor, if left to itself, increases, becomes pointed, the skin becomes reddened, and finally the discharge is effected by ulceration. The most frequent situation of the tumor is below the ensiform cartilage, but it may appear at any point over the site of the liver. Sometimes the pus burrows beneath the integument, and the tumor is situated at a greater or less distance from the liver. It has been observed as high as the axilla, and below on the inner side of the thigh. In the latter case it is liable to be mistaken for lumbar abscess. Of course partial peritonitis and adhesion over the circumscribed space occupied by the abscess are essential in order that the pus may be discharged externally. This conservative provision does not always precede the opening of the abscess, and then the contents are discharged into the peritoneal cavity; peritonitis is at once developed, and under these circumstances death speedily follows. The direction next in frequency which the pus takes is through the diaphragm into the chest. Partial peritonitis precedes the perforation of the diaphragm, and partial pleuritis ensues, frequently limited to a small space, preventing evacuation into the pleural cavity. Occasionally, however, the latter conservative provision does not take place, and the pus, discharged into the pleural cavity, at once causes general pleuritis. If this be prevented by adhesion around the opening through the diaphragm, the lung becomes perforated, and the evacuation is into the bronchial tubes, the pus being discharged by expectoration. The abscess is sometimes evacuated into the pleural cavity, and afterward perforation of the lung takes place and the pus is expectorated. Other less frequent directions in which the pus is discharged are the following: Into the stomach or intestine, the pus being then discharged by vomiting or stool; into the pericardium, causing pyo-pericarditis, or if the quantity of pus be large speedy death; into the pelvis of the right kidney, the pus being discharged with the urine; into an hepatic vein or the vena cava, giving rise to purulent infection of the blood; into the gall-bladder or biliary ducts, and thus, if there be no obstruction, the pus finds its way into the duodenum. In a case which came under my observation an abscess pointed externally and was opened; subsequently perforation of the stomach took place and liquids ingested escaped through the external opening, the patient dying from inanition.

Clinical History.—The clinical history of hepatic abscess is obscure. The symptoms rarely point distinctly to this affection. Pain exists if the hepatitis be near the surface of the liver and whenever partial peritonitis ensues; but even then pain is not always a prominent symptom, and if the inflammation be deep-seated there may be no pain. Pain is felt in the right shoulder in a small number of cases. Rigidity of the abdominal walls over the liver or of the right rectus muscle is a sign to which much importance is attached by several observers. Febrile movement is rarely marked, and may be wanting. Chills usually occur during the suppurative process. These sometimes occur regularly, simulating intermittent fever, but generally they are irregular. Jaundice is rare. The appetite and digestion may be more or less disturbed or they may be unaffected. The nutrition may be but little or not at all affected. Reference is now had to symptoms prior to the discharge of the pus or its appearance beneath the integument. The affection is not infrequently completely latent. It is not uncommon in tropical climates to find an abscess in the liver in bodies dead with various diseases when hepatic disease had not been suspected during life; and the first intimation of the existence of this affection during life in certain cases may be the discharge of pus from the stomach, bowels, or air-passages.

The clinical history after the discharge takes place will of course depend upon the direction in which the abscess is evacuated. In certain cases the phenomena of the affections caused by the presence of pus are superadded—namely, of peritonitis, pleuritis, pyæmia, etc. If the evacuation take place either externally or into the alimentary canal or through the bronchial tubes, symptoms are produced analogous to those connected with purulent discharges in other situations—namely, anæmia, debility, emaciation, and, in cases which pursue an unfavorable course, hectic fever, colliquative diarrhœa, and exhaustion, death taking place by asthenia.

CAUSATION.—Abscess of the liver may be produced by wounds of this organ. It sometimes, though rarely, follows injury over the region of the liver without wound of the latter or even of the integuments. Suppurative inflammation may invade the liver by extension of suppuration from adjoining parts. An abscess may be caused by perforation of a gastric ulcer.

Usually, however, the special agent which causes suppuration, and which probably consists of bacteria, is brought to the liver by the blood-current in the form of septic emboli, or it enters the organ from the intestine through the bile-ducts. In one group of cases the septic material is derived from some affection involving the rootlets of the portal vein in the stomach, the intestine, or the spleen. Ulcerative and necrotic processes in these parts, particularly dysenteric ulcers, are likely to give rise to secondary abscesses of the liver. As will be described in a subsequent article, suppurative pylephlebitis is usually accompanied with multiple abscesses of the liver. In another group of cases the primary focus of infection is in parts of the body other than those from which the portal vein derives its blood. Injuries and suppurations of the head, of the extremities, and of other parts may cause the entrance of the bacteria of suppuration into the blood, and these wherever they lodge and develop give rise to pyæmic abscesses. It is an interesting fact that although these bacteria must pass the pulmonary circulation before they are carried to the liver, nevertheless they not infrequently leave the lungs intact, although giving rise to hepatic abscesses. An explanation of the relatively frequent involvement of the liver by pyæmic abscesses is probably to be found in the slowness of the circulation in this organ in consequence of the diminished *vis a tergo*, so that bacteria more readily lodge in the hepatic than in the pulmonary capillaries.

Abscesses starting from the bile-ducts are rare, except in connection with the formation of biliary calculi within the hepatic ducts.

The causation of the large, single abscesses not uncommon in tropical countries is not understood. Such abscesses without evident causation are not uncommon in those countries. Cases apparently identical with tropical hepatic abscess sometimes occur in New York in those who have never visited a tropical country. There has been much discussion as to the relation between these abscesses and dysentery. In some cases tropical abscess appears to be secondary to dysentery; in other cases the dysentery follows the abscess, or both may develop simultaneously, as if dependent upon a common cause. Again, tropical hepatic abscess may develop without the appearance of dysentery at any time during the history of the case. A plausible supposition is that the bacteria of suppuration in these cases enter the liver from the intestine through the bile-ducts.

Abscess of the liver is called primary or idiopathic when not referable to some antecedent affection. The so-called tropical abscesses of the liver, at least many of them, in the present state of our knowledge are to be classified as primary. Hepatic abscesses resulting from pyæmia, pylephlebitis, and affections involving the rootlets of the portal vein are called secondary, pyæmic, or metastatic abscesses. Pyæmic abscesses are generally small and multiple; primary abscesses are usually of large size, and are either single or few in number. It is probable that the large abscesses are due to coalescence o small abscesses.

Pyæmic abscesses of the liver are not uncommon in infants as a result of inflammation of the umbilical vein.

Primary abscess of the liver is more common in males than in females, and is rare in children.

DIAGNOSIS.—When abscess of the liver is considered clinically as an individual affection, reference is had not to the small, multiple pyæmic abscesses, which are not susceptible of diagnosis, but to cases in which the patient's life is sufficiently prolonged for the abscess to attain a certain size.

The diagnosis by means of symptoms cannot be made with any degree of positiveness prior to the discovery of the abscess by manual exploration or the discharge of pus into the alimentary canal or the bronchial tubes. The symptoms which have been named as belonging to its clinical history should suggest its existence as possible or perhaps probable, especially in a warm climate, but they are not sufficiently distinctive to render the diagnosis positive. Not infrequently grounds of even a suspicion of its existence are wanting. When a fluctuating enlargement or tumor is perceived over the site or in the neighborhood of the liver, it is to be discriminated from a subcutaneous, phlegmonous inflammation, carcinoma of the liver, a hyatid formation, and a distended gall-bladder. The first of these is distinguished by pain, hardness, and redness of the integument, preceding the sense of fluctuation. Carcinoma offers a resemblance only when the medullary variety exists. The duration of the tumor and the nodulated condition of the liver as felt through the abdominal walls will serve for the recognition of this affection. A hydatid cyst is of slower development, and is not attended with the pain, tenderness, chills, and febrile movement which usually accompany an abscess. A hydatid cyst, however, may be the seat of suppuration, becoming converted into an abscess, and then the discrimination is impossible. A distended gall-bladder, which is the tumor most likely to be confounded with an abscess, is to be distinguished by its situation, the pear-shaped form, and by the fact that in consequence of peritoneal adhesions rarely taking place it has a mobility which an hepatic abscess has not. The antecedent history is of course always to be taken into account. Enlargement of the liver frequently, but not invariably, exists in cases of hepatic abscess, and the enlargement may be found to be limited to a particular direction, not affecting the whole organ—a point of some importance in the diagnosis. Exploration by means of a fine hollow needle attached to a hypodermic syringe is admissible whenever there is reason to suspect the existence of abscess, and by this method the differentiation of abscess from the other affections just named can be made demonstratively. There is no risk of injury in resorting to this procedure, and it should therefore not be omitted.

If the abscess discharge through the bronchial tubes the diagnosis is generally made without difficulty. The sudden expectoration of pus in large quantity without evidence of antecedent and coexisting empyema or pulmonary abscess, and the expectoration coming from the bronchial tubes of the right lung, render the diagnosis altogether probable. The presence of bile or disintegrated hepatic tissue affords demonstrative proof of the source of the purulent expectoration, but these are rarely present. A reddish or brown color of the expectorated pus is of diagnostic import. If the abscess discharge into the pleural cavity, the diagnosis is established by symptoms of hepatitis and suppuration having preceded the sudden development of pyothorax.

The discharge of the abscess into the stomach or intestine is followed by the vomiting of pus or its appearance in the stools. The diagnosis is to be based on these events, together with antecedent symptoms pointing to the existence of suppurative hepatitis. The discharge into the peritoneal cavity, pericardium, and vena cava gives rise to no phenomena distinctive of hepatic abscess. Its existence may have been foreshadowed by the symptoms preceding the discharge, but the proof can only be afforded by an autopsical examination. The discharge into the pelvis of the kidney gives rise to pyuria, but it may be difficult or impossible to determine that the pus comes from the liver and not from the kidney or some other source.

Prognosis.—The prognosis, even in the cases in which the abscess is evacuated in the most favorable directions, is unfavorable. When it discharges into the peritoneum, pericardium, or vena cava death speedily follows. If it discharge into the pleural cavity the termination is usually fatal. The cases which offer a fair prospect of recovery are those in which the discharge takes place through the integument, the bronchial tubes, or into the alimentary canal.

The prospect of recovery is of course less when two or three abscesses exist than when there is a single abscess.

Statistics show the duration in fatal cases to be longest when the discharge takes place through the bronchial tubes. The average duration in fatal cases is between three and four months. In cases which recover statistics show the longest duration to be when the discharge is through the integument. The average duration in cases which recover is between four and five months.

Treatment.—From what has been said respecting the clinical history and diagnosis, it follows that there is seldom an opportunity to employ therapeutic measures with a view to prevent suppuration. Even if the existence of circumscribed hepatitis were early ascertained, it is doubtful if the formation of an abscess could be prevented. Cases only in which the abscess is appreciable by palpation or an exploratory puncture, and those in which the contents are discharged into the bronchial tubes and alimentary canal, admit of treatment with a positive knowledge of the existence of the affection. In a case in which the abscess is ascertained to be making its way through the abdominal or thoracic parietes an early opening should be made and the pus evacuated. If a free opening be made before the pus has made its way beneath the integument exterior to the abdominal or thoracic walls, there is danger of the pus escaping into the peritoneal cavity in consequence of the absence of peritoneal adhesions over the abscess. Under these circumstances, in order to avoid this danger, an incision may be made through the skin and muscles down to the peritoneum without opening the abscess, and the wound filled with lint. This procedure induces firm adhesions around the wound, and in three days the abscess may be opened with safety if a spontaneous opening do not in the mean time occur. If, however, the walls of the abdomen or chest have been perforated by ulceration, a free opening may be made at once without hesitation. Prof. Janeway directs attention to the following methods of determining whether or not adhesions have taken place between the liver and abdominal wall: Hepatic friction murmur shows the absence of adhesions, but renders probable their speedy formation. Adhesions exist if the liver do not descend with inspiration. If a long needle be introduced into the liver, the part projecting will move upward as the liver descends, and downward as the liver ascends, if no adhesions exist. He advises the application of a bandage to promote adhesions by preventing movements of the liver.[1]

It is advisable to resort to aspiration as soon as the existence of an abscess is demonstrated by an exploratory puncture. The withdrawal of the pus by means of a hollow needle or trocar of small size, attached to Davidson's syringe or some one of the different instruments for aspiration now in use, need not be delayed on account of any doubt respecting peritoneal adhesions. If these have not taken place, there is no risk of the escape of pus into the peritoneal cavity. By promptly aspirating, the danger of rupture of the abscess from delay is avoided, and also whatever harm may result from the entrance of air. The pus should be removed as soon as it is discovered. The aspiration should be repeated if there be a reaccumulation of pus. By pursuing this plan a free opening may perhaps not be required. If, however, there be reaccumulations of pus, an opening should be made and the abscess treated here as in other situations. The details of the treatment belong to surgery. Aside from the local treatment, tonic and supporting measures are indicated. Good diet and out-of-door life, in so far as the patient's strength will permit, are far more important than medication.

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[1] Vide paper in *Transactions of the New York State Med. Association*, vol. i.

When the abscess evacuates through the bronchial tubes or the alimentary canal, palliative remedies are called for to relieve undue irritation of the bowels and air-passages. In addition to these, measures hygienic and medicinal, which tend to support, strengthen, and invigorate the system, are indicated, as when the discharge is through the integument.

Chronic Interstitial Hepatitis—Cirrhosis of the Liver.

An attempt has been made to distinguish various forms of cirrhosis of the liver, but concerning some of these forms there is much controversy. It seems proper to recognize at least three forms of hepatic cirrhosis—namely, the cirrhosis of Laennec, biliary or hypertrophic cirrhosis, and syphilitic cirrhosis; and these will receive separate consideration. The present article is devoted to a description of the cirrhosis of Laennec, which is the form longest recognized and the one generally understood when the term cirrhosis of the liver is used.

ANATOMICAL CHARACTERS.—The name *cirrhosis*, signifying a tawny or orange color, was applied by Laennec to the affection under consideration on account of the yellowish color of the nodules which the liver usually presents in this disease. These nodules, now known to be parts of the pre-existing liver-substance, were supposed by Laennec to be new growths replacing the original parenchyma. As the essential change, however, was found to consist in the development of fibrous tissue, the term cirrhosis came to be applied not only to the liver, but also to the fibroid induration of other organs, especially of the lungs and kidneys, although this application is etymologically incorrect. The term sclerosis, signifying induration, is more appropriate. The customary division of the pathological changes of cirrhosis of the liver into two stages, in one of which the liver is enlarged and smooth or slightly granular, and in the other small, firm, and nodular, is wholly schematic and is not based upon pathological observation. A cirrhotic liver is usually small, but it may be of normal size or enlarged, even in cases of long duration, and there is no proof that the large cirrhotic livers are to be regarded as early stages of the contracted form. The left lobe is often disproportionately reduced in size, so as to appear as a mere membranous appendage of the right. The surface is nodular; but in exceptional cases it may be smooth. The nodules vary in size from a pin's head to a pea, and may be larger. Some livers are coarsely nodular, others finely granular, although the size of the nodules or granules varies much in the same liver. The nodules are usually of a yellowish or greenish color, and consist of one or more hepatic lobules in which the liver-cells are bile-stained and usually contain oil-globules. The nodules are separated from each other by grayish-white bands of newly-formed connective tissue. This nodulated aspect has given rise to the term hob-nailed as applied to some examples of cirrhosed liver. The consistence of the liver is firm, sometimes almost cartilaginous. The cut surface is dry and anæmic.

The microscopic examination shows that the most important change is an increase of the connective tissue which accompanies, under the name of the capsule of Glisson, the medium-sized and small branches of the portal vein. This increase of the interlobular tissue is not uniform, but is in the form of bands or masses which surround usually a number of lobules. The new growth of connective tissue is not always confined to that surrounding the ramifications of the portal vein; but it often invades the lobules, and also appears around the central vein, so that the lobules may be split up into larger or smaller portions by bands of fibrous tissue. The new tissue is usually rich in lymphoid and spindle cells, and with age it generally has a tendency to become fibrous. It invades the walls of the interlobular veins, so that they lose their elasticity and many of them become obliterated. The most important symptoms are referable to this destruction of a number of branches of the portal vein. The new tissue, however, is not poor in vessels, many of which are probably newly formed and derived from the hepatic artery. The branches of the hepatic artery and the bile-ducts suffer much less from the compression of the new tissue than the thin-walled portal vessels.

This development of connective tissue is attended by atrophy of hepatic lobules and destruction of a large number of liver-cells. The atrophy of the hepatic parenchyma has usually been attributed to the compression of the liver-cells by the fibrous tissue, but an equally important factor is the lack of nutrition consequent upon the obliteration of the interlobular veins. The remaining hepatic cells usually contain oil-globules and are stained with bile-pigment.

There is much plausibility in the view first advanced by Weigert, and now adopted by several authors, that the primary change in cirrhosis is degeneration and necrosis of a certain number of liver-cells. This view is based upon investigations which show that in similar chronic interstitial inflammations in the kidney and elsewhere the primary change is degeneration of the epithelium or parenchymatous cells, and the new growth of connective tissue is secondary to this.

In many cases of cirrhosis of the liver a number of branching, narrow channels, containing a single or double row of cubical epithelial cells and resembling the interlobular bile-ducts, are met with in the connective tissue. They are too many to consist of only pre-existing interlobular ducts, and there has been much discussion as to their origin. Three theories have been advanced: one, that they are offshoots by new growths from the old bile-ducts; a second, that they are intralobular bile-capillaries which have acquired an epithelial lining; and a third, which is perhaps the most probable, that they are rows of liver-cells altered in shape and arrangement by the growth of connective tissue into the lobule.

The capsule of the liver is thickened, and often presents little villous projections of vascularized connective tissue, which develop into the fibrous adhesions so frequently connecting the liver with the diaphragm and abdominal wall.

Cirrhosis may be combined with extensive fatty infiltration and with amyloid degeneration of the liver. Especially in the latter case the liver may be of very large size.

The changes in other organs are chiefly referable to the portal obstruction and consequent damming back of blood upon the rootlets of the portal vein.

The effects of this obstruction are only imperfectly relieved by the establishment of collateral channels. The spleen in the great majority of cases is enlarged and firm. This enlargement is due to congestion, and sometimes also to hyperplasia of its stroma—a condition which may be referable to the same cause as the cirrhosis. The size of the organ is not usually more than twice or thrice the normal. The increase cannot always be made out during life. The mucous membrane of the stomach and intestine is usually thickened, pigmented, and coated with more or less muco-pus; in other words, in the condition of chronic inflammation. Ascites and, in the later stages, general œdema are present. Hemorrhages into the stomach and intestine, and in other parts of the body, as in the retina, may be found after death.

The small kidney and other forms of chronic diffuse nephritis occasionally complicating cirrhosis of the liver are probably effects of the same cause.

Cirrhosis is to be regarded as a chronic inflammatory affection involving the interstitial tissue of the liver.

CLINICAL HISTORY.—Cirrhosis, as a rule, gives rise to few or no symptoms which point to the liver as the seat of disease prior to the occurrence of hydro-peritoneum. Preceding this event and afterward, pain in the region of the liver exists in only a small proportion of cases. Dropsical effusion is usually the first symptom to suggest the existence of the affection. The dropsy may occur when the patient supposes himself to be in his habitual state of health, or it may be preceded and accompanied by indefinite ailments. The dropsical accumulation increases, usually with considerable or great rapidity. It is rare for the quantity of liquid to remain at a moderate amount for a considerable period. Œdema of the lower limbs in a moderate degree is observed in a considerable number of cases before the patient's attention has been directed to enlargement of the abdomen. It is, however, probable that an amount of dropsical effusion not sufficient to attract attention always precedes the œdema of the lower limbs. After considerable abdominal enlargement has taken place the œdema increases. The limbs often are largely swollen, and the genitalia may become more or less œdematous. The œdema in these situations is attributable to pressure of the peritoneal effusion upon the iliac veins. Œdema of the face and upper extremities does not occur except in cases in which cardiac or renal disease coexists. Œdema of the lower limbs does not occur in all cases.

Jaundice is present in a small proportion of cases, and is rarely great. The lesion does not necessarily occasion obstruction of the biliary ducts, although it must diminish the secretion of bile. Pallor of the prolabia and skin exists in the majority of cases. In general, the anæmic aspect is marked in proportion as other symptoms show the condition of the patient to be unfavorable. Febrile movement does not accompany the disease in its progress. The pulse may be not increased in frequency, or it may be more or less accelerated. It is generally soft and feeble. When the pulse is frequent, the other characters denote diminution of the vital forces, or asthenia.

The obstruction of the portal circulation is compensated, in a measure, by new channels of communication between the portal and the systemic veins. The most important of these are anastomosing branches which pass to the liver between the folds of the falciform ligament and in the ligamentum teres, communicating with the veins of the abdominal parietes through the epigastric and the internal mammary veins. The course of the blood in the anastomosing branches in cases of cirrhosis is the reverse of that in health, being in the latter in a direction toward, and in the former from the liver. Owing to this, the abdominal veins become dilated, and those superficially situated in some cases are seen to be more or less enlarged. A collateral circulation, in a measure, is also established through communications between the inferior mesenteric and the hypogastric veins in the hemorrhoidal plexus; by anastomosing branches between the vena portæ and the veins of the serous covering of the liver; between the coronaria ventriculi and the œsophageal veins; and by newly-formed vessels in the tissue by which the liver adheres to the diaphragm. The size of the superficial abdominal veins on either side of the median line is sometimes enormous. The dilated network of radiating veins around the umbilicus gives rise to the appearance called the caput Medusæ. The conservative effect of compensatory development of veins communicating with the two circulations is shown by the fact that examples of great enlargement of the vessels upon the abdomen may be observed in patients who are free from dropsy, notwithstanding a notable amount of cirrhosis.

Hemorrhage from the stomach and bowels is an occasional event. Gastrorrhagia occurred in 6 of about 40 cases which I have analyzed, the histories not being complete in all the cases. It preceded the hydro-peritoneum in 3 cases. Hæmatemesis may proceed from varices at the lower end of the œsophagus. Enterorrhagia coexisted with gastrorrhagia in 2 cases, and occurred without it in 2 cases. Vomiting is occasionally a prominent symptom, and diarrhœa is prominent in a small proportion of cases.

At an advanced period of the disease hemorrhage is liable to occur from the nostrils, sometimes from the gums, and also into the retina. In these situations the hemorrhage is attributable, not to the portal congestion, but to an altered state of the blood. The appetite is usually impaired. A sense of fulness after taking food is generally complained of if the dropsy be sufficient to distend the abdomen. Progressive emaciation attends the progress of the disease, and in an advanced stage the attenuation of the upper portion of the body, the distended abdomen, and the lower limbs enlarged by œdema render the general aspect highly characteristic.

Albuminuria is rare in cases of cirrhosis. Of 28 cases in which the condition of the urine in this respect was noted, in only 1 case was albumen present. The urine is generally scanty, but in some cases it is abundant. The coloring matter of the urine is abnormally abundant—a fact attributable to the deficient secretion of bile; and the blood-pigment which serves for the production of the bilirubin, not being expended in the liver, is eliminated in the kidneys. The abundance of the coloring matter may be such as to give to the urine the appearance of being bloody. The urates are usually deposited in great quantity, and are notably red from the abundance of pigment.

In the great majority of cases there are no important symptoms pertaining to the nervous system. The mode of dying is usually by slow asthenia. If, however, the abdomen be greatly distended or if the dropsical accumulation take place very rapidly, death may be due in part to the extent to which the respiratory function is compromised. The mental faculties are generally preserved up to the last moments of life. To this rule, however, there are exceptions. In 3 cases I have noted the occurrence of delirium several days before death; in 1 case the delirium was hilarious; in 1 case the patient appeared bewildered; and in 1 case the patient lapsed from childlishness into imbecility. These cases ended in coma. In 3 other cases the patients died comatose. In 1 case convulsions occurred, followed by coma.[1] The symptoms of icterus gravis may appear in the last stage.

CAUSATION.—Cirrhosis in the vast majority of cases is due to spirit-drinking. Hence the significance of the term *gin* or *whiskey liver* applied by British writers to this affection. In most cases an inquiry into the mode of drinking will show that the habit has been to take raw spirits at different periods of the day, before breakfast and at other times, on an empty stomach, a little water being generally drunk after the spirits. In this mode the greatest amount of effect from a given quantity of alcohol is obtained. Occasionally patients declare that they have been accustomed to drink only wine or beer. If the use of alcohol in any form be denied, it may perhaps be said, in connection with the disease, that etiological laws are more trustworthy than human testimony. In a considerable number of cases the statements of patients in regard to the quantity of spirits taken are not reliable.

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[1] This account of the symptomatology is based on an analysis of 46 cases of hydro-peritoneum, cirrhosis existing in nearly all the cases. (Vide *Clinical Report*, already referred to.)

The disease is sometimes attributed to other causes than alcohol—namely, to malarial poisoning, syphilis, chronic peritonitis, chronic phosphorus-poisoning, and occlusion of the common bile-duct. The efficiency of these causes is open to doubt. At all events, if the diagnosis of cirrhosis of the liver be made, the inference of spirit-drinking is always warrantable. As to the action of alcohol in producing this affection, the explanation is that, passing readily into the portal blood from the stomach and carried at once to the liver, it excites by contact inflammation of low grade in the interlobular spaces, and hence the production of adventitious tissue; or, according to Weigert's view, it causes primarily death of a certain number of hepatic cells. So far as my observations go, they furnish no evidence of the agency of disease of the heart in the causation of this affection.

Cirrhosis very rarely occurs at less than thirty years of age, and in the majority of cases the age exceeds fifty years. Still, cases of typical cirrhosis have been observed in young children. The affection is generally a result of the habitual abuse of alcohol continued for a long period. Males are affected much oftener than females—a fact readily explained by the dependence of the affection on the use of spirits in the manner stated.

DIAGNOSIS.—The diagnosis in most cases of cirrhosis after dropsy has occurred is made without difficulty. Hydro-peritoneum, occurring as a local dropsy—that is, irrespective of general dropsy—in a person addicted to spirit-drinking warrants an inference that this affection exists. Additional proof is afforded by the diminished size of the liver. This is ascertained by percussion. The upper margin of the liver is accurately determined by finding the line of hepatic flatness; and the lower border is determined not so accurately, but approximately, by the line of demarcation between hepatic flatness and the tympanitic resonance due to gas in the transverse colon. The latter is not exact, because tympanitic resonance is conducted for a certain distance above the lower margin of the liver, and, moreover, a distended colon may rise above the lower margin of the liver. The vertical diameter of the liver in health, on the linea mammillaris, is about four inches. Directly after the operation of tapping, while the abdominal walls are relaxed, the lower border of the liver may frequently be grasped by the fingers pressed upward beneath the false ribs, and its indurated, nodulated condition appreciated by palpation. It is to be remembered that a normal or even increased size of the liver would not exclude the diagnosis of cirrhosis.

The physician is rarely called upon to make the diagnosis prior to the occurrence of dropsy. It may be practicable in some cases to determine, by palpation, induration and a nodulated condition of the lower part of the organ before dropsy has taken place; and, this information obtained, the diagnosis is highly probable if the patient be addicted to spirit-drinking. Hemorrhage from the stomach sometimes precedes the occurrence of dropsy, and in a spirit-drinker this renders the existence of cirrhosis probable. Hydro-peritoneum, it is to be borne in mind, occurs in other pathological connections, as well as in cirrhosis of the liver. (Vide p. 587.) When it occurs as a result of occlusion of the portal vein by thrombosis, the peritoneal dropsy occurs rapidly, the spleen may become greatly enlarged, and the liver is undiminished in size. A number of cases of gastrorrhagia from cirrhosis without dropsy have come under my observation. I have known most profuse hemorrhage, proceeding from cirrhosis without dropsy, to be followed by the recovery of a fair condition of health. On the other hand, I have known it to cause sudden death.

Prognosis.—Cirrhosis is to be regarded as an incurable lesion. After it has led to dropsy, in the majority of cases its course is progressively toward a fatal termination. The duration in sixteen fatal cases, dating from the occurrence of dropsy, varied between six weeks and seventeen months, the average duration being about five months; but doubtless in all cases the disease has existed for a considerable period before dropsy occurs. In a certain proportion of cases, the dropsy being removed, the patient may regain comfortable or even good health, and remain free from any manifestations of the disease for months and years; but sooner or later, as a rule to which there are very few exceptions, the dropsy returns, and unless life be destroyed by some intercurrent affection the disease ends fatally. Circumstances which preclude much expectation of improvement are—the coexistence of cardiac, renal, or any other important disease, considerable emaciation, sufficient debility to keep the patient in bed, greatly impaired appetite and digestion, speedy reaccumulation of liquid after tapping, and the occurrence of jaundice.

Treatment.—As regards treatment in cases of cirrhosis and in the great majority of the cases of hydro-peritoneum, the indications are the same. The reader is therefore referred to the Treatment of Hydro-peritoneum, considered in the preceding chapter.

Hypertrophic Cirrhosis of the Liver.

It has already been mentioned that exceptionally in ordinary alcoholic cirrhosis the liver may be large throughout the whole course of the disease, presenting the same microscopical appearances and giving rise to the same symptoms as the more usual contracted liver. French writers[1] were the first to call attention, under the name *hypertrophic cirrhosis with severe icterus*, to a disease which they consider to possess distinctive pathological and clinical characteristics. The liver, as a rule, is considerably enlarged, its surface smooth or finely granular, and its consistence firm. Its free and cut surfaces present little yellowish islands of liver-parenchyma, between which are grayish bands and masses of connective tissue. The lobular markings are indistinct or obliterated. The new connective tissue is more uniformly distributed than in ordinary cirrhosis. It surrounds and penetrates the individual lobules[2] rather than surrounding groups of lobules, and is particularly rich in so-called newly-formed bile-ducts. It is maintained by Charcot and Gombault that this form of cirrhosis begins as an inflammation of the interlobular bile-ducts, and extends to the surrounding connective tissue.[3] This opinion, however, is hypothetical. The spleen is constantly enlarged, usually to a greater extent than in ordinary cirrhosis.

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[1] P. Olivier, Hayem, and especially Hanot, *Étude sur une Forme de Cirrhose hypertrophique du Foie*, Paris, 1876, and Charcot et Gombault, *Arch. de Physiologie*, 1876.

[2] For this reason it is called by Charcot insular or monolobular cirrhosis, in distinction from annular or multilobular cirrhosis, which is the ordinary form.

[3] Hence this form of cirrhosis has also received the name of biliary cirrhosis. There is another form of biliary cirrhosis which is chiefly of pathological interest. This latter form is due to obstruction of the main bile-ducts, usually by a gall-stone. The retained bile causes the death of some liver-cells, and a resulting increase of interstitial tissue.

As a characteristic feature of this form of cirrhosis, marked icterus is present throughout the greater part of the disease. Ascites is less marked than in ordinary cirrhosis, and it appears later or may be absent. Symptoms referable to the digestive tract are present, as in ordinary cirrhosis. In the later stages marked cerebral symptoms—coma, convulsions, and delirium—are frequent. Here, too, the only well-established cause is the abuse of alcoholic liquors. The duration of the disease is usually more than a year, and may be five or six years. The chief points in the diagnosis are the enlargement of the liver, the presence of icterus, the enlargement of the spleen, and the absence of marked ascites. The even surface and uniform enlargement of the liver distinguish hypertrophic cirrhosis from enlargement due to echinococcus, carcinoma, and other new growths. The causation is also to be considered in making a diagnosis. In some cases of otherwise typical hypertrophic cirrhosis with icterus, ascites has been a marked feature.

Syphilitic Hepatitis.

There are several forms of syphilitic disease of the liver. Diffuse syphilitic hepatitis is observed as a frequent manifestation of congenital syphilis, but very rarely if ever in the adult. In this condition the liver is enlarged, smooth, firm, and anæmic. The lobular markings are obscured. Upon microscopical examination there are found an increase of the connective tissue, both within and without the lobules, and a quite uniform infiltration with small round cells.

A second form of syphilitic hepatic disease is characterized by the presence of gummata in the liver. These sometimes appear in new-born children as very abundant, yellowish, miliary nodules scattered throughout the substance of the liver. In the adult, gummata generally appear in the liver as scattered nodules varying in size from a pea to a hen's egg, with a dry, yellowish, caseous centre, surrounded by a grayish zone of fibrous tissue which encroaches upon the hepatic parenchyma.

One of the most characteristic forms of syphilitic disease of the liver, the one often called the *syphilitic liver*, is that in which the liver is greatly distorted in size and shape by coarse bands of fibrous tissue, which pass from the thickened capsule into the substance of the organ and cause deep cicatrix-like depressions. In this condition the surface of the liver is coarsely lobulated, and presents an absolutely characteristic appearance. In the fibrous bands gummata may be present, and it is held by the majority of writers that the fibrous tissue is developed only around gummata or as cicatricial tissue in their place. Not infrequently, however, gummata are absent in these livers, and Virchow, denying that the caseous products of a gumma can ever be wholly absorbed, regards this disease of the liver as a chronic interstitial hepatitis developed in a circumscribed form, either with or without the presence of gummata.

Finally, amyloid degeneration of the liver, either occurring independently or complicating one of the previously described conditions, is one of the manifestations of the syphilitic taint.

Syphilitic disease of the liver, as is the case with all forms of visceral syphilis, appears chiefly in the tertiary stage. Although in itself not common, it is one of the most frequent forms of visceral syphilis.

Syphilitic disease of the liver, especially the gummatous form, may give rise to no evident symptoms. The lobulated syphilitic liver may remain latent, so far as symptoms are concerned, but it usually is accompanied with some or all of the symptoms of ordinary cirrhosis, from which it is to be distinguished by the syphilitic history of the patient and the characteristic feel of the liver. The coarse nodules which can be felt on these livers resemble very much those present in carcinoma of the liver, and the distinction between them is not always easy. The umbilication, which can sometimes be felt on the cancerous nodules, may be of assistance in the diagnosis. The spleen is usually enlarged in syphilitic liver, and is not enlarged in cancer of the liver. The history of the patient is, however, the capital point in the diagnosis, as bearing upon the presence or absence of cancer or of syphilitic disease in other parts of the body.

Acute Yellow Atrophy of the Liver.

This affection is characterized, as the name denotes, by a rapid reduction in the size of the liver. Its anatomical characters were first recognized by Rokitansky in 1842. The same disease has been known under the names *malignant* or *fatal jaundice*, *hemorrhagic icterus*, and *diffuse parenchymatous hepatitis* (Frerichs). It is a comparatively rare disease.

ANATOMICAL CHARACTERS.—In typical cases the liver is much diminished in size in all directions, but especially as regards its thickness. The volume and likewise the weight may be reduced one-half or even two-thirds. Occasionally the size of the liver is not below the normal. This happens especially when the disease attacks a liver previously enlarged or when the affection is of unusually short duration. The consistence is soft and flabby, so that the organ when resting on its concave surface is flattened, or when supported on its edge it folds up from its own weight. The capsule is wrinkled in consequence of diminution in bulk of the liver-substance. In the living body the shrunken liver may sink back toward the vertebral column, so that coils of intestine find place over its anterior surface.

Upon its cut surface the liver appears intensely jaundiced. The color may be yellow throughout, but the prevailing yellow color is usually mingled with some reddish-brown patches. The reddish color may preponderate over the yellow. Great variety exists in the relative proportion and the distribution of the yellow and the red substance. The yellow substance is soft and friable. Its color is due to bile-staining and the presence of fat. The red substance is of lax texture, but of tough consistence, and on section it is seen somewhat depressed below the level of the yellow. It appears homogeneous, without any trace of the hepatic lobules, or at the most presents the faint outlines of very small lobules. In the yellow substance the outlines of the lobules are very much obscured. If seen at all, the lobules are found to be larger than in the red substance, but as a rule they are smaller than normal. The disease is often farther advanced in the left lobe than in the right. The former may be almost uniformly red, while the latter is yellow mottled with some red spots.

The microscope shows that the atrophy is due to an actual destruction of the hepatic cells by the process of fatty degeneration. Different stages of the process can usually be found in the same liver. At first the hepatic cells are swollen, cloudy, and bile-stained; but later their outlines are obscured and their substance is occupied by fat in the form either of granules or of large and small globules. There is reason to believe that the fat is formed directly from the albuminous material of the cell. Finally, there is left no trace of the liver-cells, but in their stead we find a mass of granular detritus and drops of fat, with crystallized and amorphous bile-pigment. All these conditions may be observed in different parts of the yellow substance, the degree of destruction of liver-parenchyma varying much in different cases. In some instances the detritus resulting from the disintegration of the cells seems to be more albuminous than fatty.

The red substance is usually regarded as representing a later stage of development of the yellow portion. Its color was formerly supposed to be due to blood-extravasations which may be present in the liver of acute yellow atrophy. Upon microscopical examination, however, the red substance is found generally to be composed chiefly of connective tissue, blood-vessels, and probably newly-formed bile-ducts. Some granules and small drops of fat, together with bilirubin crystals, may still be found in the connective tissue, but the greater part of the disintegrated liver-cells has been absorbed. Cells which can be recognized as hepatic cells are entirely absent or are present in small number. Leucocytes, sometimes in abundance, are found in the connective tissue. The increase in connective tissue is partly apparent, in consequence of disappearance of the liver-cells and of their fatty detritus, and partly real, by means of a new growth of the interlobular tissue. In this red substance are often found narrow, branching channels filled with a single or double row of small polyhedral cells. These epithelial canals resemble the small interlobular bile-ducts; but they are too many to consist only of pre-existing ducts. They are considered by Waldeyer, Zenker, and others as offshoots by new growth from these bile-ducts, and intended for the regeneration of the destroyed liver-parenchyma. Klebs and Perls, however, regard the cells in these canals as rows of pre-existing liver-cells altered in shape. Attempts at artificial injection of the blood-vessels of the liver after death fail in consequence of the escape of injecting material between the liver-cells.

The larger bile-ducts are usually empty. They may be inflamed and contain a secretion of muco-pus, or they may contain bile-stained fluid. The gall-bladder contains a mucous fluid either colorless or bile-stained. It may contain apparently normal bile. Leucin and tyrosin crystals often form in the substance of the liver after death. Rod-shaped bacteria and micrococci have been found by Klebs and others in the bile-ducts and in the atrophied liver-tissue. Their accumulation in large numbers in the bile-ducts has led to the belief that they have made their way there from the intestine, but whether before or after death is a matter of uncertainty.

The changes in other organs are similar to those found in certain acute infectious diseases. The tissues are jaundiced. The blood is fluid or loosely coagulated and contains leucin and tyrosin. The cardiac muscle and the renal epithelium usually show decided fatty degeneration, not approaching, however, in degree that of the liver. The stomach and intestine frequently show evidences of inflammation. The epithelial cells of the gastric tubules undergo parenchymatous degeneration. The spleen is found swollen and soft, unless its size have been reduced by hemorrhage from some of the rootlets of the portal vein. Multiple hemorrhages occur in the majority of cases. These occur especially from vessels of the portal system. Blood is often found in the contents of the stomach and intestine, also in the mucous membrane of the bladder, pelvis of the kidneys, beneath the serous membranes, in the lungs, etc. Hemorrhages very rarely take place in the brain.

CLINICAL HISTORY.—The affection is either developed abruptly or is preceded by a prodromic stage. The premonitions are symptoms denoting gastro-duodenitis and are not significant of the affection. Without or with premonitions lasting usually from three to five days, but sometimes much longer, jaundice occurs. This is an almost constant symptom, but it may be absent. It is usually marked, but not intense, and does not differ, as regards the coloration of the skin, from ordinary jaundice. Intense cephalalgia and delirium, occurring either simultaneously with the jaundice or after a period varying between two and twenty-one days, become prominent symptoms. The delirium is often active, patients crying out and sometimes requiring restraint; but in some cases the delirium manifests itself only in quiet wandering. In a third of the cases convulsions occur. These in some cases are epileptiform, in some cases they consist of general tremor or rigor, and in some cases they are limited to a few muscles. Trismus has occasionally been observed. To the delirium and convulsions stupor is added, eventuating in profound coma. The pupils are sometimes contracted, but are usually dilated, and in some cases they are unaffected, responding readily to light. When the jaundice first appears the pulse is slow, but when delirium occurs the pulse is more or less frequent. During the progress of the disease the pulse fluctuates remarkably as regards frequency. At one time it may rise to 120, at another time falling to 80 or 90. The pulse varies also as regards other characters. In the latter part of the disease it becomes persistingly rapid, increasing progressively in frequency and becoming more and more attenuated and feeble. There is generally a moderate rise of temperature in the early stage, but at a later period fever may be absent, except that sometimes shortly before death a considerable rise (104° or 105°) occurs. Hemorrhages take place in various situations, generally into the stomach and intestines, in females from the uterus, and occasionally there is hæmaturia. Extravasations are not uncommon, giving rise to petechiæ and to ecchymoses found after death in various situations. Pains in the epigastrium are common, extending to the right hypochondrium. Tenderness on pressure exists in these regions. The bowels are generally constipated. The stools are sometimes clay-colored, sometimes green and bilious, and sometimes dark from blood. The breathing is likely to be early accompanied by sighing, and after coma occurs the respirations are irregular and stertorous. The urine has been found to contain biliary salts by a number of observers. The small quantity present and the difficulty in the detection of these salts may explain some of the negative results of other observers. The amount of urea is notably diminished, especially in the latter part of the disease, when it may entirely disappear. Leucin and tyrosin are found in the great majority of cases. Leucin in the form of globules somewhat resembling fat is found only after evaporating the urine. Tyrosin in the form of needles appears as a spontaneous sediment. Albumen is found in a moderate quantity. Coolness and dryness of the surface exist, except under the excitement of delirium and convulsions, when the heat of the skin is sometimes increased. Enlargement of the spleen in a greater or less degree exists in most cases of this disease.

The duration of the affection is usually short. In 28 cases, a fatal termination took place in 13 within a week, in 6 during the second week, in 5 during the third week, and in 4 during the fourth week.

The termination in the great majority of cases is fatal.

PATHOLOGICAL CHARACTER.—As regards the anatomical changes in the liver, according to many authors they denote a diffuse parenchymatous inflammation. As opposed to this view, the alterations formerly regarded as evidence of parenchymatous inflammation are now attributed to degenerative processes. The theory, based upon the presence of leucocytes in the interstitial tissue, that the inflammation is interstitial, is not satisfactory. The wandering cells are most abundant in the red substance, and their presence may therefore be regarded as a secondary change. The greater number of pathologists at the present time regard the changes in the liver as primarily regressive and produced by an acute fatty degeneration.

As to the pathological character of the disease, there have been many speculations, most of which possess only historical interest, such as that the changes in the hepatic cells are due to an over-production of bile, to paralysis of the bile-ducts, to disturbances in the innervation of the liver, etc. The hypothesis now most strongly advocated is that acute yellow atrophy of the liver is an infectious disease. The changes in the liver may be regarded as local manifestations of a general infection, or the infectious principle may be considered to act primarily upon the liver and secondarily upon the blood and other organs. According to the latter view, the special poison makes its way into the system through the liver, perhaps by means of the ingress of the infectious material (bacteria?) from the intestine into the bile-ducts.

The symptomatic phenomena which form the clinical history of the disease are in a greater or less degree effects of the action of the infectious principle, the existence of which may be assumed. Certain of the grave phenomena, however, are attributable to the non-elimination of excrementitious principles which it is a function of the liver to excrete. The retention of these in the blood is denoted by the term acholia. The physiological discovery that the cholesterin in bile is an excretory product derived chiefly from the destructive metamorphosis of nerve-tissue is sustained by, and affords a rational explanation of, the phenomena in this disease which are referable to acholia. That these are effects of cholesteræmia is evidenced by clinical observations showing in this disease a notable accumulation in the blood of cholesterin, and by experiments showing the production of phenomena analogous to those of this disease in animals by the injection of cholesterin into the blood.[1] Uræmia is doubtless in many cases a pathological element, but the clinical history shows clearly toxical effects other than those referable to that element.

CAUSATION.—Of 143 cases analyzed by Thierfelder, 88 were women and 55 were men. Of the 88 women, 30 were attacked during pregnancy and 3 were in childbed. Of the 143 cases, 93 were between the ages of twenty and of thirty-nine; 2 were less than one year of age, and 5 were more than fifty. Aside from the influence of age, sex, and the condition of pregnancy, as shown by the above statistics, we have no knowledge of causative agencies. If, as held by many, the disease be specific or infectious, it involves, of course, a special cause.

DIAGNOSIS.—The symptoms are not diagnostic of this disease prior to the occurrence of the grave toxæmic phenomena—namely, delirium, hemorrhages, etc.—in connection with jaundice. The fevers—typhus or typhoid, remittent, pyæmia, yellow fever—are excluded by the absence of the diagnostic features which belong to them severally, and sometimes by the absence of a febrile temperature. Diminution of the size of the liver while the case is under observation, in connection with the clinical history, renders the diagnosis positive; but this effect does not always take place. Moreover, it is sometimes difficult to determine accurately the vertical diameter of the liver by percussion, owing to the fact that the tympanitic resonance from the colon may be conducted upward, so as sometimes even to meet the pulmonary vesicular resonance when the size of the liver is normal. There is, therefore, a liability to error in deciding that the liver is reduced in size. This difficulty applies to cases in which the transverse colon is distended with gas; but generally it may be correctly ascertained whether the size of the liver has diminished. The presence of leucin and tyrosin in the urine is diagnostic. The sex of the patient and the existence of pregnancy have some weight in the diagnosis, as well as the gravity of the disease, as denoted by the symptoms and the rapid progress toward a fatal termination.

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[1] Vide "Experimental Researches into a New Excretory Function of the Liver," by A. Flint, Jr., *Am. Journ. of Med. Sciences*, 1862; also, Flint's textbook of *Human Physiology*.

Prognosis.—It is questionable whether this disease ever ends in recovery. Cases of recovery have been reported, but there is perhaps more probability that in these cases there was an error in diagnosis than that the disease really existed and terminated favorably.

Thierfelder gives the following statistics in regard to the duration after the development of grave symptoms, based on an analysis of 118 cases: twenty-four hours or less in 10; from twelve to forty-eight hours in 46; from two and a half to three days in 26; from three and a half to four days in 17; from five to seven days in 15; nine days in 2; and ten and fourteen days, of each 1 case.

Treatment.—A few words will embrace all that is to be said with reference to treatment. With our present knowledge nothing is to be expected, beyond palliation, from remedies. Drastic purgatives and emetics have been advised, but the propriety of these or of other perturbatory measures is very doubtful. Symptoms which furnish indications for palliative remedies are vomiting and hemorrhages. For the vomiting and gastrorrhagia ice in small pieces, bismuth, and anodynes may be prescribed. The mineral acids are appropriate. The symptoms referable to the circulation—namely, the frequency and feebleness of the pulse, with coolness of the surface—indicate tonics and stimulants. Inasmuch as the convulsions and coma may be due to uræmia, it would be desirable, if possible, to excite the action of the kidneys; and if this be not practicable the hot-air bath and perhaps hydragogues suggest themselves as means of effecting the vicarious elimination of urea. Hemorrhage from the bowels is to be controlled by cold applications and opiate enemas. Revulsive applications over the site of the liver by means of fomentations and stimulating embrocations or rubefacients may be of some service. Quinia in full doses deserves further trial.

CHAPTER XII.

DISEASES OF THE LIVER (CONTINUED).

Fatty Liver.—Parenchymatous Degeneration of the Liver.—Waxy Liver.—Cancer of the Liver.—Other New Growths in the Liver.—Hydatid Tumors of the Liver.—Multilocular Echinococcus.—Other Parasites in the Liver.—Pigmentary Deposits in the Liver.—Hypertrophy and Atrophy of the Liver.—Changes in the Position and in the Shape of the Liver.—Congestion of the Liver.—Portal Thrombosis.—Adhesive and Suppurative Pylephlebitis.

HAVING considered inflammatory affections of the liver, I shall take up, next in order, structural affections of this organ. The more important of these are incident to an abnormal accumulation of fat, to the waxy or lardaceous alteration, to carcinoma, and to hydatid tumors. These will be considered in this chapter.

Fatty Liver.

The liver is one of the normal situations for the deposition of fat. In well-nourished, healthy individuals, the liver-cells in the periphery of the lobules contain some oil-drops. As there is great variation in the quantity of fat contained in the livers of healthy persons, it is by no means easy to determine the amount which is to be regarded as abnormal.

It is customary to distinguish between fattty infiltration and fatty degeneration of the liver. The reader is referred to Part I. p. 54 *et seq.* for the grounds upon which this distinction, as adopted in this work, is based. The distinction may be here repeated: In fatty infiltration or fatty growth the protoplasm is displaced by the fat, but does not suffer materially in its integrity, except by a slow process of atrophy from compression. If the fat be derived from the albuminous constituents of the cell, these are, in great part at least, renewed. In fatty degeneration or fatty atrophy the cell-substance is directly converted into fat, and is not at all or is insufficiently regenerated. There are important exceptions to the rule that in fatty infiltration of the liver the fat is present in the form of large drops, whereas in fatty degeneration it appears as small drops or molecules. The most typical example of fatty degeneration of the liver is acute yellow atrophy of this organ, to which no further reference will be made in this connection, as it has been already considered. Some writers regard all the other forms of fatty liver as examples of fatty infiltration. It is true that, as a rule, they present the gross and microscopical appearances which have been considered characteristic of infiltration; but, as explained in Part I., these morphological appearances do not suffice for the diagnosis between infiltration and degeneration.

A fatty liver is usually more or less enlarged, its margins rounded, and its surface smooth. The consistence is less than in health. The specific gravity in extreme cases is so light that the organ floats in water. The weight, however, owing to increase in volume, is usually greater than in health. The color is yellowish, and the lobular markings are often obscured. Drops of oil adhere to the knife in making a section. The organ is anæmic. While the amount of fat which can be extracted by ether from a normal liver is estimated at from 3 to 5 per cent., that present in extremely fatty livers may rise as high as 40 or 45 per cent.

In the physiological accumulation of fat in the livers of women during lactation the fat is present chiefly in the central cells of the lobules. In most other cases of fatty infiltration the fat appears primarily in the hepatic cells in the periphery of the lobule, and extends, as the fatty accumulation increases, to the cells in the centre. The fat appears first as small drops, which increase in size and coalesce, until finally one large drop occupies nearly the whole of the space within the cell-contour. An hepatic cell thus altered is spherical in shape, its nucleus is pushed to one side, its protoplasm forms a narrow ring around the oil-drop, and the whole cell resembles an adipose-tissue cell. The fat may also exist between the hepatic cells, either free or in stellate cells in the intercellular spaces.[1]

The essential condition for the development of fatty metamorphosis of the liver is that more fat be either introduced or produced than can be oxidized. This failure of oxidation may depend either upon an excessive introduction or production of fat in the organ, or upon a diminution in the oxidizing processes due usually to a deficient supply of oxygen to the cells. We thus find fatty livers in conditions apparently the most opposite—namely, in cases of great obesity and in cases of extreme emaciation. The causes of fatty liver may be summarized as follows:

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[1] Perls, *Lehrbuch der allgemeinen Pathologie*, Bd. 1, p. 177; Platen, *Virchow's Archiv*, Bd. 74, p. 268.

1. Lactation. The accumulation of fat in the centre of the lobules before and during lactation is to be regarded as physiological rather than pathological. 2. Obesity. Persons who take little muscular exercise, whose food contains an excess of hydrocarbonaceous substances, and who have a predisposition, either hereditary or acquired, to the deposition of fat, may develop very large fatty livers as well as an excessive panniculus adiposus. 3. Continued and excessive indulgence in alcoholic stimulants. This leads often, but not constantly, to fatty infiltration of the liver as well as to cirrhosis. 4. Pulmonary phthisis, and less frequently other cachectic conditions, such as cancer, chronic dysentery, profound anæmias, etc. The largest fatty livers met with are those developed during chronic pulmonary tuberculosis, especially in females. 5. Phosphorus-poisoning. Although the liver here presents the appearance usually considered as characteristic of fatty infiltration, it is probable that poisoning by phosphorus leads to fatty degeneration of the liver, as well as of the kidneys, muscles, etc. Arsenic and some other poisons also occasion fatty metamorphosis of the liver. 6. The acute fatty degeneration of the new-born described by Hecker and Buhl.[1] The liver takes part with other organs in the general fatty degeneration. 7. Yellow fever. The fat is present in large drops, but the process is fatty degeneration. The hepatic cells usually contain fat in cirrhosis and in chronic passive congestion of the liver. The cause here is local and referable to the interference with the circulation.

Our present knowledge is not sufficient to enable us to determine the pathological importance of fatty liver. The pressure of the fat appears to be sufficient to render the organ anæmic, but not to occasion obstruction enough to give rise to peritoneal dropsy. If the latter coexist with fatty liver, cirrhosis is combined, or there exists some other cause for the hydro-peritoneum. The spleen does not become enlarged. There may be some obstruction to the passage of bile in the minute hepatic ducts, but this is not sufficient to occasion jaundice. The function of the secreting cells must be more or less impaired when these are filled with fat, but we are unable to refer any symptoms to this condition.

The diagnosis of fatty liver can be made only when the organ is enlarged. There are no subjective local symptoms other than those dependent on the increase of size. The enlargement may be determined by percussion and by manual exploration below the false ribs. The blunt or rounded lower margin may sometimes be ascertained. The smoothness of the surface is appreciable, and sometimes also the diminished consistence; or, at all events, the absence of induration may be ascertained. An enlarged liver presenting these characters, occurring in a patient affected with pulmonary phthisis, an intemperate person, or a person of luxurious, indolent habits, may be considered as probably fatty. The probability is greater in females than in males. Simple enlargement, in this pathological connection, is not sufficient for the diagnosis of fatty liver, inasmuch as waxy liver is not infrequently associated with pulmonary phthisis; and it is not always easy to determine the physical characters which distinguish these two forms of enlargement from each other.

Fatty liver occurring in cases of pulmonary phthisis does not claim treatment. In fact, the treatment of phthisis, which in certain cases is highly useful—namely, with cod-liver oil and alcoholics—may be supposed to favor the deposit of fat in the liver. Nor, as a general remark, with our present knowledge, does it claim treatment when associated with other important affections. Occurring in persons addicted to the use of alcohol and to overfeeding, especially as regards fatty articles of diet, it is to be treated by a reformation of the habits of living—namely, by temperance or total abstinence in respect to the use of alcoholic beverages, by restricting the diet in a great measure to articles not abounding in fat, and, if the strength will permit, by increased muscular exercise. The remedies indicated are those which will tend to strengthen and invigorate the system. Fatty liver combined with chronic alcoholic gastritis offers a bad prognosis.

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[1] *Klinik der Geburtskunde*, 1, 1861.

Parenchymatous Degeneration of the Liver.

By the terms parenchymatous degeneration and cloudy swelling of the liver is designated a condition of the organ often met with after death from fevers, diseases involving severe blood-poisoning, and affections accompanied with a high temperature. The liver is sometimes enlarged and the outlines of the lobules are indistinct. The liver-cells are swollen and filled with granules which are coarser and more abundant than normal. These granules at first are albuminous, and they disappear on the addition of acetic acid. They may, however, give place to fatty granules and drops. Some regard parenchymatous degeneration as an essential stage in the process of fatty degeneration. The clinical importance of parenchymatous degeneration of the liver is not well understood.

Waxy Liver.

The liver is one of the organs frequently attacked by waxy degeneration. This degeneration, called waxy, amyloid, lardaceous, or albuminoid, consists in the deposition of an albuminous substance, the characters of which have been considered in Part I. of this work (p. 55). It may be repeated here that this substance strikes with a solution of iodine a reddish-brown or mahogany-brown color; with iodine and sulphuric acid, a blue color; and with methyl-violet, a red color. The reaction with iodine alone suffices as a test.

In cases of advanced waxy degeneration of the liver the organ is much enlarged. The enlargement is in some cases enormous. The weight may be twelve or fourteen pounds, and is not infrequently double the normal weight. The form of the organ is not materially altered. The surface is smooth, and the edges are usually, but not always, blunted or rounded. The consistence is notably stiff and board-like, so that thin slices are easily taken off. Notwithstanding its firm consistence, the organ cuts easily, like uncooked bacon. The interior of the liver is dry and anæmic. The cut surface, especially when examined in thin slices held to the light, presents a characteristic translucent appearance compared to uncooked bacon, wax, smoked salmon, and tissues soaked in glycerin. The lobular markings are obscured, but even in extreme cases opaque yellowish-white lines can be seen usually about the periphery and in the centre of the lobules. The amyloid or waxy change is not, as a rule, uniformly distributed, but some lobules are more affected than others; and with the naked eye, even in advanced degrees of the degeneration, lobules and parts of lobules, usually of a yellowish color from fatty metamorphosis, can be seen without the characteristic translucence. In the lesser degrees of the waxy change the organ is not at all or but slightly enlarged, and the affected parts can be distinguished only by the chemical tests mentioned. The earliest stages are to be recognized only by microscopical examination of sections stained with iodine or with iodine and sulphuric acid or methyl-violet.

Under the microscope the amyloid material appears as a white, structureless, translucent, refracting substance. The amyloid degeneration begins first in the terminal branches of the hepatic artery and in the walls of the capillaries within the lobule. If we imagine the lobule to be divided into three concentric zones, the amyloid deposit appears first chiefly in the middle zone, and then gradually extends to the centre of the lobule, while the peripheral part remains longest unaffected. The peripheral hepatic cells usually contain oil-drops, and the central cells are often stained with bile-pigment. The hepatic vein and its branches and the interlobular branches of the portal vein may become waxy. In the arteries and veins the waxy material first replaces the muscular fibres of the middle tunic, and may then extend to the other coats. In the interior of the lobules the waxy material is deposited along the capillaries between their endothelium and the liver-cells. The capillaries appear to be changed into canals with very narrow and finally obliterated lumen and thick refracting walls, between which the liver-cells are compressed and finally disappear. It is uncertain whether the waxy material be ever deposited within the hepatic cells. A large number of liver-cells are destroyed by pressure when the whole lobule becomes waxy. Usually, when the liver is the seat of this morbid change other organs are also affected, especially the spleen and kidneys, then the intestine, lymphatic glands, heart, large arteries, etc. Sometimes, even in comparatively slight degrees of the degeneration, the waxy substance is deposited in the liver as a homogeneous nodule, of the size of a walnut or even larger, and more like a tumor than a centre of infiltration or degeneration.

The affection is rare in infancy and in old age. Of 116 cases analyzed by Frerichs and E. Wagner, 24 occurred at between ten and twenty years of age; 37 between twenty and thirty years; 31 between thirty and fifty years; 16 after the age of fifty; and 8 prior to the age of ten years. It is met with in cachectic patients who have suffered from caries or necrosis of bones. It is incident to prolonged suppuration, especially of bone, as in hip-joint disease, Pott's disease, etc.; also to cachexia from tumors, slowly-growing cancers, and other causes. It occurs in persons affected with syphilis. It has been observed to follow malarial fever. It accompanies pulmonary phthisis, especially when there are suppurating cavities. In all these connections it is a secondary affection. In a small proportion of cases it is not preceded by any other disease. Cohnheim has demonstrated that three or four months suffice for the change to be brought about. It occurs in the male sex oftener than in the female.

The pathological importance of waxy liver and its clinical history are to be based on the study of cases in which the affection is not associated with other serious affections. Such cases are not easily collected, for if the affection be not secondary, the kidneys, spleen, and other parts are liable to be similarly affected. It is undoubtedly an important affection. The functions of the liver must be compromised in proportion to the amount of the deposit. It may impede the portal circulation through the liver sufficiently to give rise to peritoneal dropsy. Jaundice very rarely occurs. Pain referable to the liver is wanting. If the kidneys be affected, general dropsy is likely to occur.

The DIAGNOSIS of this affection is to be based on the existence of enlargement and the exclusion of other affections of the liver involving increase of size. From fatty liver it is to be distinguished by the greater resistance on palpation and by the margins being in a less degree blunt or rounded. The existence of peritoneal dropsy in some cases aids in the exclusion of fatty liver. From cancer of the liver it is distinguished by the absence of tumor and the smoothness of the surface. The absence of subjective local symptoms, especially pain, aids in the exclusion of cancer. Cirrhosis generally leads to contraction of the organ; but this affection is excluded by the smoothness of the surface when it can be felt, and by either the absence of peritoneal dropsy or the existence of only a small amount of effusion. The coexistence of enlarged spleen, and the evidence of disease of the kidneys afforded by albuminuria, general dropsy, and perhaps uræmic symptoms, strengthen the diagnosis. A capital point in the diagnosis is the coexistence of affections to which, as clinical observation shows, this affection is secondary—namely, prolonged suppuration, necrosis of bones, syphilis, etc.

The PROGNOSIS is unfavorable. The lesion must be regarded as, in most cases, incurable. There seem to be some data for believing, especially in cases of cured hip-joint disease, that patients may regain good health after waxy degeneration of the liver has been established, but it is doubtful whether the waxy material be absorbed. Frerichs, Graves, and Budd, however, have observed diminution of the size of the liver in cases in which this affection was supposed to exist. Generally, the fatal termination is to a greater or less extent due to the diseases with which the affection is associated.

The TREATMENT must have reference to the morbid conditions under which the affection is developed. The iodide of potassium and the iodide of iron are considered by Frerichs as sometimes successful in cases in which syphilis is involved. In scrofulous or tuberculous patients the therapeutic measures should be directed especially to the cachexia which is to be considered as standing in a causative relation to the affection. Budd recommends the muriate of ammonia, from five to ten grains three times daily. Frerichs attaches importance to mild alkaline remedies and the use of the sulphurous mineral waters. Dietetic and other hygienic measures are of the first importance. The body should be well nourished, the surface properly protected against vicissitudes, and as much out-of-door life as the strength will permit is advisable. Disorders of any of the functions are to receive appropriate attention. Diarrhœa occurs not infrequently, and this will claim astringent remedies and perhaps opium in small doses. Albuminuria and general dropsy furnish indications the same as when these symptoms occur in connection with any of the renal changes which, collectively, are called Bright's disease.

Cancer of the Liver.

Of the various forms of tumor which may occur in the liver, cancer is the one of greatest importance. While primary cancer of the liver is a very rare disease, the liver is one of the most frequent seats of secondary cancer. The statistics of Leichtenstern show 74 cases in which the cancer was primary, and 358 in which it was secondary. Primary hepatic cancer may develop either from the epithelium of the bile-ducts or from the liver-cells. In secondary cancer of the liver the primary tumor may be seated in various organs of the body, but it will be found most frequently in the stomach,[1] and then, in order of frequency, in the intestine, including the rectum, and the uterus. It has been demonstrated that the secondary cancerous nodules may be developed from emboli of cancer-cells transmitted from the primary growth along the portal vessels or the lymphatics into the liver, and it is believed that this is the most frequent mode of propagation. A cancer in an adjoining organ, especially the stomach, may invade the liver by continuous growth.

Primary carcinoma of the liver usually presents itself in the form of a single large tumor, more or less distinctly circumscribed, and often surrounded with several smaller cancerous nodules. Primary cancer of the liver may also occur in an infiltrated form, and in this case the liver sometimes bears considerable resemblance to a large cirrhotic liver, but the parts corresponding to the lobules are larger and of a yellowish-white color, and they present the microscopical appearances of cancer. Secondary carcinoma of the liver is almost invariably presented in the form of multiple circumscribed nodules of varying size and number. These are usually many, but the whole organ may be studded with them, while sometimes only a few nodules are present. The cancerous nodules have generally a whitish color, with scattered yellowish dots and lines. Extravasations of blood into the cancerous mass are occasionally observed. The consistence is sometimes that of scirrhous, but more frequently that of medullary carcinoma. The tumors are present as well on the surface as in the interior of the organ. A quite characteristic appearance of those nodules which reach the surface is the presence of a cup-shaped depression in the centre. This so-called umbilication is due to degenerative and wasting changes in the central or oldest part of the tumor. Partial peritonitis, causing adhesions, is generally developed when the tumors project beyond the surface, and in some cases the peritonitis becomes general.

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[1] Of 1574 cases of cancer of the stomach analyzed by Welch, the liver was the seat of secondary cancer in 475 (30.2 per cent.).

The type of the secondary cancer conforms generally to that of the primary growth. As a rule, the cancerous alveoli are large and close together, and contain irregularly-shaped, polygonal cells. The cells may, however, be of the cylindrical epithelial type. So-called melanotic carcinoma of the liver is secondary, usually, to similar primary tumors of the choroid coat of the eye or of the skin. They are usually melano-sarcomata.

The cancerous affection usually gives rise to more or less enlargement of the liver. The increase in size and weight is sometimes enormous. The weight has been known to reach twenty-five pounds. Primary as well as secondary cancer of the gall-bladder and larger bile-ducts has been repeatedly observed.

Cancer here, as in other situations, rarely occurs in early life. Of 472 cases analyzed by Leichtenstern, the ages in 37 were between twenty and thirty years; in 61, between thirty and forty years; in 250, between forty and sixty years; and in 33, more than sixty years. Of 756 cases, 334 were men and 422 were women.[1]

It is a singular fact that cancer in this situation is more infrequent in tropical than in temperate climates.

Cancer of the liver being generally secondary, the symptoms in most cases are combined with those referable to other affected organs. It is much oftener secondary to an affection of other abdominal viscera than to a primary cancer of a part not connected with the portal circulation; and when developed secondarily under these circumstances the hepatic affection is liable to be overlooked.

The general manifestations of the cancerous cachexia—namely, waxy pallor, progressive emaciation, and debility—are usually present sooner or later; but in this as in other situations the affection sometimes makes considerable progress before cachectic phenomena are apparent. The affection in some cases remains for a longer or shorter period quite latent. The general symptoms may be manifest without local phenomena pointing to the seat of the affection; but in the majority of cases the affection speedily gives rise to both general and local symptoms. Pain is usually more or less prominent, referable to the liver and shooting in different directions. Tenderness on pressure is a frequent symptom. Jaundice occurred in 67 of 146 cases analyzed by Leichtenstern. In the cases in which it occurs the jaundice persists until death. Peritoneal effusion occurs in about the same proportion of cases—namely, 78 of 154 cases. This may be due to peritonitis excited by the cancerous tumors or to pressure upon the portal vein. In the latter case the effused liquid is serum without lymph; in the former, more or less lymph is exuded. Occasionally blood, extravasated into the cancer, escapes into the peritoneal cavity. The accumulation of liquid may be so great as to call for tapping. The appetite and digestion are early impaired, and at a later period diarrhœa not infrequently occurs. Œdema of the lower extremities is liable to occur in the latter part of the disease. Emaciation and loss in weight are progressive, and death takes place by slow asthenia if the patient be not carried off by some intercurrent disease. A fatal termination may be hastened by peritonitis induced by the local affection, by abundant hemorrhage, by coexisting cancerous affections elsewhere, and by various accidental complications. Aside from these circumstances the progress of the affection is sometimes rapid and sometimes slow. The duration varies from a few weeks to a year, rarely, if ever, exceeding the latter. The average duration is twenty weeks.

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[1] Vide *Ziemssen's Handbuch.*

In most cases the DIAGNOSIS is made without difficulty. The enlargement of the organ, the presence of one or more nodules or tumors appreciable by the touch, with pain and tenderness, and frequently the knowledge of the existence of cancer in some other situation, render the nature of the affection sufficiently clear. The hepatic affections to be excluded are—abscess of the liver, cirrhosis, waxy liver, hydatids, and syphilitic growths. For their diagnostic characters the reader is referred to the articles treating of these affections. Tumors situated near the liver are to be discriminated—namely, aneurism, cancer of the stomach, peritoneal or omental cancer, a distended gall-bladder, and fecal masses in the ascending or transverse colon. The antecedent and coexisting symptoms, taken in connection with the local signs, are in most cases sufficient for the differential diagnosis. Progressive loss in weight and the appearances which denote the cancerous cachexia point to a malignant disease.

The diagnosis is difficult when no evidence is obtained by palpation. If the cancerous growth be confined within the liver, and the organ be simply enlarged—still more, if the size be normal or below the size in health—the existence of the affection can only be conjectured in view of more or less pain and tenderness over the liver, conjoined with a cachectic condition and the existence of cancer elsewhere. The diagnosis is impossible in the few cases in which there are no physical signs and when the affection is latent as regards local symptoms.

The age of the patient, peritoneal effusion, persisting jaundice, and hereditary predisposition are to be taken into account in the diagnosis.

The TREATMENT has reference only to the palliation of symptoms and the prolongation of life. Pain will often require the habitual use of opium. An advantage of an early diagnosis consists in the avoidance of injurious medication. Every debilitating measure will tend to shorten life. The system is to be supported, as far as practicable, by tonic remedies, nutritious diet, and other hygienic measures. The object in this, as in other chronic incurable affections, is to endeavor to aid the system in resisting as long as possible the fatal termination.

Other New Growths in the Liver.

A brief enumeration of the more common of the other tumors found in the liver must suffice, since they generally possess no clinical interest. Primary sarcoma is not known to occur in the liver. Secondary melanotic sarcoma has been mentioned under the head of Cancer. Spindle-celled sarcomata and lympho-sarcomata have been observed as secondary growths in the liver.

Tubercle appears in the liver in the form of countless miliary nodules of small, often microscopic, size in the course of general tuberculosis and in chronic pulmonary tuberculosis. Large, solitary, cheesy tubercles may occur in the liver, as in the brain and spleen, but they are pathological curiosities in this situation. Lymphomata are present in many cases of leucocythæmia and pseudo-leucocythæmia. Little nodular accumulations of lymphoid cells in the portal canals, so-called miliary lymphomata, are often observed after death from typhoid fever, and in cases of diphtheria, smallpox, and scarlatina. Adenoma presents itself in two forms—either as a nodular hyperplasia of liver-substance or as an atypical growth of bile-ducts in a nodular form. Cysts of retention developed from bile-ducts, and cysts apparently of new formation, are not infrequent, combined sometimes with cystic disease of the kidney. These cysts are sometimes lined with ciliated epithelium. Little nodules of cavernous tissue are often observed in the liver. Gummata have been mentioned under the head of Syphilitic Hepatitis.

Echinococcus or Hydatid Tumors of the Liver.

The echinococcus represents the larval stage of tænia echinococcus, a tapeworm about one-sixth of an inch in length, which infests the small intestine of the dog. The embryos developed from the eggs of the tænia which have been introduced into the human alimentary canal make their way through the blood-vessels and lymphatics into the various organs of the body, where they undergo further development. It is held that these embryos may enter the liver also by way of the bile-ducts. An hydatid tumor or echinococcus cyst consists of a membranous bladder, varying in size from that of a pin-head to a child's head, enclosed in a fibrous capsule developed from the surrounding tissue. The hydatid vesicle is not adherent to the surrounding sac. Its wall is composed of an outer cuticular layer, which appears, under the microscope, clear and translucent, with exceedingly characteristic parallel lines, indicating a lamellated structure, and an inner granular, germinal layer provided with cilia. In *simple* echinococcus cysts minute whitish particles, called *brood-capsules*, which contain several *scolices* or tapeworm heads, project as outgrowths from the inner germinative layer into the cavity of the sac, which is filled with fluid. A scolex has in front four suckers and a double row of from thirty to forty hooklets. It is capable of invaginating the anterior portion of the head, so that the hooklets may appear to be in the dilated posterior part. This scolex undergoes no further development in man, but, finding its way into the alimentary canal of certain animals, especially the dog, it becomes a fully-developed tapeworm. Simple echinococcus cysts are not common in man. Usually there is an endogenous proliferation of so-called daughter-vesicles, so that the main or mother-cyst contains floating globular or ovoid cysts varying in size from that of a pea to a hen's egg, and in number from a few to several thousand. The daughter- (and sometimes grand-daughter-) cysts produce from their inner germinal layer brood-capsules containing scolices, in the manner described for the mother-cyst. Very rarely in man, but frequently in the ruminants, there is an exogenous instead of an endogenous growth of daughter-vesicles, which then appear as small sacs outside of the mother-cyst. The fluid contained in an echinococcus cyst, free from regressive or inflammatory changes, is clear, somewhat opalescent, neutral or slightly alkaline, and with a specific gravity between 1007 and 1015. It is devoid of albumen or has only a trace, is rich in chloride of sodium, and it frequently contains grape-sugar, inosite, succinic acid and succinates, and some less important ingredients. (For a description of the modifications, some of a rather complex nature, which may occur in the structure of echinococcus cysts, the reader is referred to works treating specially of entozoa.[1]) Brief reference, however, must be made to the regressive changes and to the multilocular echinococcus. The fluid may be absorbed or thickened, the vesicles and scolices may undergo fatty and calcareous metamorphoses, and the fibrous capsule may shrink so that nothing is left but a fatty calcareous nodule, usually containing cholesterin and hæmatoidin crystals. The hooklets are preserved among the *débris* and aid in the diagnosis. In this way spontaneous cure may ensue. It is maintained that the destruction of echinococci is sometimes due to the entrance of bile into the mother-cyst. Inflammation, resulting in suppuration, of an echinococcus cyst may take place. Sometimes no scolices are developed. Such cysts, with or without daughter-vesicles, are called *acephalocysts*.

Echinococci occur in the liver oftener than in all other parts collectively. They may be present in any organ of the body. Usually there exists a single tumor, but there may be a dozen or more. Similar tumors sometimes coexist in other organs, especially in the lungs and spleen. Oftener found in the right than in the left lobe, they may exist in any part of the organ. The growth of the echinococcus is very slow, extending usually over years; but it is sometimes continuous, or it may become arrested at any stage by the death and consequent regressive changes of the parasite.

The echinococcus disease is less frequent in the United States than in Europe and some other countries. It is said to be particularly common in Iceland, where the inhabitants live in close community with dogs. Osler has collected 61 cases occurring in this country. Of these, fully one-third, and probably more, were in foreigners. Echinococci are said to be not infrequent in hogs and cattle in this country.[2]

The clinical history offers nothing which points to this or any affection of the liver until the tumor attains a sufficient size to occasion inconvenience by pressing on the adjacent parts. As long as the tumor is contained within the gland it is usually completely latent. It gives rise to neither pain nor tenderness, and the hepatic functions are not appreciably disturbed. Hydatids of considerable size are not infrequently discovered unexpectedly in autopsical examinations. If, however, the cyst be situated near the periphery and project considerably beyond the organ, it is likely to give rise to local symptoms directing attention to the part. Usually, under these circumstances pain is slight or wanting, but there is a sense of fulness and uneasiness in the neighborhood of the liver. When pain is present it is due mainly to inflammation developed by the pressure of the tumor. The pressure upon adjacent parts gives rise to symptoms which differ according to the direction in which the tumor extends. Extending into the thoracic space, it gives rise to dyspnœa on exertion, cough, and palpitation. Pressure on the stomach and intestine is liable to produce vomiting and constipation. If the vena cava be compressed, œdema of the lower extremities follows. The growth of the tumor is attended by little or no constitutional disturbance. There is no febrile movement, and the nutrition of the body may be unimpaired.

Important local and general symptoms, however, are incident to the bursting of the cyst or the discharge of its contents by ulceration. These symptoms will depend on the direction in which the opening occurs. Pleuritis, pericarditis, and peritonitis are results of the rupture respectively into the pleural, pericardial, and peritoneal cavities, and the two latter affections thus induced are uniformly fatal. The first of these three affections is very likely to prove fatal. Opening into the vena cava, it gives rise to the symptoms of obstruction of the pulmonary circulation from embolism. If the lung be perforated without general pleuritis, cough and expectoration are prominent symptoms, and in the latter are contained, from time to time, hydatids, together with bile in some cases. If perforation of the lung ensue after the evacuation into the pleural sac, the phenomena of pneumo-hydrothorax are developed. Perforation of the stomach or colon is usually attended by severe pain, and is followed by the discharge, either by vomiting or stool, of hydatids. The discharge of the hydatids into the biliary passages gives rise to jaundice. Jaundice, irrespective of this cause, is rare in cases of hydatid tumor. Dropsy of the peritoneum does not belong to the clinical history of the affection. Both jaundice and hydro-peritoneum, however, are occasional effects of the pressure of the tumor on the biliary passages without the liver and on the vena portæ.

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[1] The following works may be consulted: Davaine, *Traité des Entozoaires*, 2me édition, Paris, 1877; Leuckart, *Die menschlichen Parasiten*, i., Leipzig, 1863; Heller, in *Ziemssen's Cyclopædia of the Practice of Medicine*, vol. iii.; Cobbold's *Treatise on Parasites*.

[2] Osler, *Am. Journ. of the Med. Sciences*, Oct., 1882.

A DIAGNOSIS is impracticable so long as the growth does not extend beyond the gland. The affection can be recognized only when either the tumor is discoverable by physical examination or hydatids are discharged through some outlet. A tumor extending from the anterior surface or from the inner or lower margin of the liver, after it has attained a certain size is appreciable by the touch. The diagnosis now involves its discrimination from other tumors. When situated over the liver it is to be discriminated from cancer and hepatic abscess. This discrimination may generally be made without much difficulty. From cancer it is distinguished by its smooth and globular form, its elasticity and the sense of fluctuation, the freedom from pain, and the absence of the cancerous cachexia. From hepatic abscess it is distinguished by the slowness of its growth, absence of pain and tenderness, and by its being unattended by the constitutional symptoms to which suppuration generally gives rise—namely, chills, fever, etc. Extending from the left border, it is to be discriminated from cancer of the stomach or of the pancreas, and from an aneurismal tumor. The connection of the hydatid tumor with the liver, and the disconnection of other tumors, may generally be ascertained by palpation and percussion. Cancer of the stomach or of the pancreas is usually accompanied by pain, and in the former situation by notable gastric symptoms. The cancerous cachexia may be apparent. A cancerous tumor is generally irregular or lobulated. An aneurismal tumor is distinguished by its anterior and lateral pulsation, the frequent occurrence of murmur, and generally by persistent gnawing pain referable to the back.

A physical sign sometimes obtained by percussion is highly distinctive of a hydatid tumor. If percussion be made upon the tumor, the fingers of the left hand or the whole hand being placed over the tumor, the collision of the floating hydatids with each other causes a characteristic tactile vibration known as the hydatid fremitus. The sensation is compared to that felt when percussion is made upon the hand resting on a mass of jelly. By placing the stethoscope over the tumor and practising percussion, a peculiar sound is elicited, which in some cases has a musical intonation like the sound from a violoncello. These signs are obtained in only a certain number of cases.

An hydatid tumor encroaching more or less upon the thoracic space gives rise to flatness on percussion and absence of respiratory murmur from the base of the chest upward in proportion to the height to which the tumor extends, with perhaps more or less displacement of the heart. The signs are those of pleuritis with effusion. Attention to the following point will suffice for the discrimination in many if not in most cases: In pleuritis the level of the liquid effusion when the patient is sitting or standing is denoted by flatness extending upward on the anterior aspect of the affected side of the chest to about a uniform height; but the flatness due to a tumor extending into the thoracic space generally has an irregular limit; that is, the flatness extends higher at some than at other points. Moreover, the test of the presence of liquid effusion afforded by the results of percussion when the position of the body is changed from the vertical to the recumbent is not available in the case of a tumor.

The diagnosis in cases in which the tumor opens externally or its contents are discharged by expectoration, vomiting, or stool, is established by the discovery of hydatids, either entire or the remains left after their destruction. If entire, they are easily recognized with the naked eye. If not entire, the microscope is to be employed to discover shreds of the characteristic membrane and hooklets of echinococci.

The most certain means of arriving at a positive diagnosis in cases in which the contents of the tumor are not discharged through some outlet externally is by withdrawing a portion of the liquid by means of an exploring canula. If the fluid withdrawn be clear, free from albumen, and rich in chloride of sodium, there can scarcely be room for doubt as to the nature of the tumor. The presence of succinic acid or of glucose is evidence in favor of echinococcus. If the contents have become mixed with blood or inflammatory products, the chemical examination is not conclusive. If scolices or their hooklets, or portions of the finely-striated but otherwise homogeneous echinococcus membrane, be found in the fluid, the diagnosis is established.

Hydatid tumors are not in themselves serious, but they do harm by pressure upon adjacent parts. They prove serious also by opening into certain situations in which grave or fatal diseases are induced by their presence. The prognosis in these cases has been already stated. If the opening occur into a situation from which the contents are discharged from the body—namely, through the integument into the bronchi or into the alimentary canal—recovery may be hoped for or expected according to the circumstances in individual cases. If recovery do not take place, the fatal termination is preceded by protracted irritation and progressive exhaustion, due generally to suppuration within the sac.

In the TREATMENT of hydatid tumors of the liver there are two objects to which measures may be directed. One object is the evacuation of the tumor, and the other is to arrest its growth and promote absorption of its contents. Medicinal means are applicable to the last-named object only; and the first object is of course to be effected exclusively by surgical interference.

Several internal remedies have been proposed as capable of destroying the hydatids. On the death of the parasite the tumor ceases to grow, and, gradually diminishing in size, it may finally disappear. This is the mode in which a spontaneous cure not infrequently takes place. Calomel, common salt, and the iodide of potassium have been thought to have the power of effecting the destruction of the hydatids, these remedies acting by being absorbed and exerting a poisonous effect upon the parasites. Their efficacy, however, does not appear to have been established by clinical observation.

An effectual surgical measure in certain cases is to make an incision into the tumor sufficiently large to remove both the liquid and the hydatids which it may contain. This operation is warrantable when the tumor extends in an outward direction, provided adhesions have taken place around a space in which the opening is made, so that there is no danger of the escape of the contents into the peritoneal sac. A method of determining whether adhesions exist or not is to ascertain if the tumor or the liver be depressed by a forced inspiration or remain fixed in the same position. This point is readily ascertained by marking on the chest or abdomen the situation of the tumor, or the lower margin of the liver, or its upper margin, successively at the end of an expiratory and an inspiratory act. After evacuating the tumor cicatrization may be expected to follow, but this result may be preceded by suppuration within the sac, which may be continued so as to involve danger from constitutional irritation and exhaustion.

A simple method of treatment which often suffices for the cure is to withdraw a portion of the liquid by aspiration, puncturing the sac with a fine hollow needle or trocar. The destruction of the parasite may be in this way effected, and the risk of the escape of the liquid into the peritoneal cavity is extremely small. The injection of medicated liquids into the sac is unnecessary, and increases the liability to suppuration. If suppuration occur, a free opening may be required. In order to avoid danger from a free opening, Simon recommends the following procedure: "The tumor is punctured with a fine trocar and the canula allowed to remain; in twenty-four hours peritoneal union will have taken place. Eight days afterward a second trocar is thrust in at some distance from the former, and the canula again allowed to remain. After about twenty-two hours the two punctured wounds may be united by an incision."[1] For the destruction of the parasite electricity, transmitted through needles introduced into the tumor, has been effective.

The prophylaxis of hydatids in the liver and elsewhere is to be based on the source—namely, the ova of the tænia echinococcus developed in the intestine of the dog. Hence it is important, as far as practicable, to avoid the possibility of the introduction of ova from the excreta of this animal into the human intestine.

Multilocular Echinococcus.

The echinococcus multilocularis represents a peculiar form of development of this parasite. With very few exceptions it has been found only in the liver. It forms a hard, smooth tumor, often of very large size, which takes the place of the liver-parenchyma. Upon section this tumor bears a striking resemblance to a colloid cancer, with which it was formerly confounded. In the meshes of a firm, fibrous stroma are small cavities containing a gelatinous material in which are present the characteristic echinococcus membranes, rarely in the form of vesicles. Fully-formed scolices are rarely found, but their hooklets are more frequently observed. The tumor has a tendency to form in its central part ulcerative cavities sometimes filled with pus. Virchow, who first recognized the nature of the disease, holds that the echinococcus multilocularis develops in the lymph-vessels; and Friedreich, that it grows in the blood-vessels and bile-ducts.

The disease has been observed almost exclusively in South Germany and Switzerland. Prudden describes a case occurring in a German who had been in this country five years.[1] The symptoms are usually more pronounced and graver than those of ordinary echinococcus. They are—jaundice, often intense and rarely absent, gastric irritation, enlargement of the spleen, sometimes pain and tenderness in the region of the liver, progressive emaciation, and, at the last, often hemorrhage from different mucous membranes. The prognosis is very grave. The enlarged liver presents usually a smooth, hard surface, and not the nodular swellings of ordinary echinococcus and cancer. The duration, too, is longer than that of cancer, which is not usually accompanied with enlarged spleen or such intense jaundice. There is no effectual treatment.

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[1] Vide Heller, in *Ziemssen's Cyclopædia*, Am. ed., vol. iii.

[1] Delafield and Prudden's *Handbook of Path. Anat.*, New York, 1885, p. 354.

The other parasites which are found in the liver are of little clinical importance, at least in this climate, on account either of their infrequency or their innocuousness. The cysticercus cellulosæ is a rare parasite in the liver. Various species of distoma, of which the most common in this climate are distoma hepaticum, or liver-fluke, and distoma lanceolatum, produce serious disturbances in dogs, sheep, and some other animals, but they are infrequent in man. They are found in the gall-ducts and in the gall-bladder. In India, and especially in China, certain species of distoma, known as distoma crassum and distoma spathulatum, or sinense, are said to be common and to cause serious hepatic disorders. The distoma hematobium, found by Bilharz in Egypt, occurs in the portal veins, but causes no lesion of the liver.

The pentastoma denticulatum, the larval stage of pentastoma tænioides, infesting the nasal cavities of dogs, horses, and some other animals, has been frequently found, especially in parts of Germany, in the human liver, where it usually occurs in small calcareous cysts. The pentastoma constrictum has been observed, especially in Africa. A specimen of this parasite, which had been expectorated by a patient under his care, was sent to me by Dr. M. M. Campbell of Missouri. From seventy-five to one hundred of these parasites had already been expectorated. The liver was much enlarged, and the primary habitat of the parasites was probably in this organ.[1] The pentastomes are not known to interfere with the hepatic functions.

The ascaris lumbricoides sometimes enters the common bile-duct from the intestine. It may cause obstruction of the ducts and jaundice, but this occurrence is very rare. The psorosperms which are so common in the rabbit's liver have been occasionally found in the human liver, where they generally prove harmless.

Pigmentary Deposits within the Liver.

A deposit of dark-brown granules in the liver, giving to the organ a steel-gray, chocolate, or bronzed appearance, is observed not infrequently after death from malarial fevers. A similar deposit generally coexists in the spleen, and may be found in other organs. It is found in the blood, especially within the portal vessels. The source of the pigmentary matter, together with its pathological relations and effects, cannot be considered as settled. (Vide Part I. p. 78.) With our present knowledge, the pigment-liver is hardly entitled to be reckoned in the list of hepatic affections. It will be referred to hereafter as entering into the anatomical characters of the periodical fevers.

A certain amount of bile-pigment is often present in normal livers, and this amount is increased in diseases causing obstruction to the flow of bile.

In severe anæmia, particularly in pernicious anæmia, the liver contains a large number of colorless granules which are stained greenish-black by treatment with sulphide of ammonium, and which therefore contain iron. These ferruginous particles are probably the result of an extensive destruction of red blood-corpuscles.

Hypertrophy and Atrophy of the Liver.

Hypertrophy of the liver was supposed to be not infrequent before the several degenerative affections to which this organ is liable were as well understood as now. While the liver may be greatly increased in size by tumors, abscesses, fatty and amyloid degenerations, congestion, and certain forms of cirrhosis, there is no evidence that a simple hypertrophy of the liver with preservation of its normal structure occurs as a pathological condition. The size of the liver varies considerably within normal limits.

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[1] Vide *N. Y. Med. Record*, Jan. 13, 1877; also Aitken's *Science and Practice of Medicine*.

It has been proven by experiments that the liver possesses to a moderate degree the power of regeneration of its tissue when this is destroyed to a limited extent. We may assume that this regenerative power is active after such degenerations of liver-cells as sometimes occur in phosphorus-poisoning, typhoid fever, cholera, and certain other diseases.

Simple atrophy of the liver occurs as a senile change and in diseases accompanied with prolonged marasmus. The liver is uniformly diminished in size, and the liver-cells often contain a considerable amount of reddish-brown pigment (brown atrophy).

Changes in the Position and in the Shape of the Liver.

Aside from congenital malformations, which are of no special clinical interest, the liver may undergo changes of position which it is important to understand for diagnostic purposes.

The liver is readily displaced downward by pressure from above, as by large pleuritic effusions, and is displaced upward by fluid accumulations in the peritoneal cavity, by abdominal tumors, and by tympanitic distension of the intestines. It possesses a certain degree of mobility upon its transverse axis, and, in consequence of this, pressure from below, as by the distended colon, may cause the liver to rotate, so that its anterior surface becomes superior, and the normal liver-flatness in front almost or wholly disappears. Its mobility may be abnormally increased by relaxation of its ligaments, and sometimes, especially in women with flabby abdominal walls who have borne many children, it is considerably dislocated and is very movable (hepar mobile). Sometimes coils of intestine, particularly the transverse colon, may pass between the liver and the anterior abdominal walls, so as to substitute a tympanitic resonance for the normal hepatic flatness.

The liver may be variously distorted by pressure. The most important of these distortions is that produced by tight-lacing. In consequence of constriction of the lower part of the chest it is compressed from side to side, and a circular furrow or depression is produced which may be so deep as to almost divide the organ, especially the right lobe, transversely into two parts, of which the lower may even be tilted up over the upper division. Corresponding to the tight-lace furrow the liver-substance is atrophied and the capsule is thickened and opaque.

The liver may be dislocated downward by the traction of peritoneal adhesions below the organ. In an instance observed at Bellevue Hospital the lower margin was a little above the crest of the ileum. In a case reported to the New York Medical and Surgical Society by Willard Parker the dislocation was an effect of a fatal injury. Dr. Alex. Y. P. Garnett has reported a case in which dislocation followed a violent muscular exertion, a spontaneous return of the organ to its normal situation taking place within three days.[1]

Restoration in cases of dislocation—or, as it has been termed, wandering liver—is to be effected, if practicable, by pressure from below, and the organ should be retained in place, by a properly-adapted abdominal supporter.

Congestion of the Liver.

Congestion of the liver may be either active or passive. Active congestion is caused by an increased flow of blood to the liver through the hepatic artery or the portal vein. Passive congestion is the result of some obstruction to the outflow of blood from the hepatic veins.

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[1] Vide *Am. Journ. of Med. Sciences*, Jan., 1881.

A physiological *active congestion* of the liver occurs during digestion, and it is believed by many writers that this form of congestion may become abnormal in degree or in persistence in those who indulge excessively in the pleasures of the table and who lead sedentary lives. It is held also that highly-spiced food and immoderate use of alcohol tend to produce active hyperæmia of the liver. Active congestion is said to be more common in tropical than in temperate climates, and is attributed to the use of highly-seasoned food, to telluric influences, and to malarial infection.

Congestion of the liver formerly played a more important rôle in pathological speculations than at the present time. It entered into the conception of so-called abdominal plethora, and in connection with an abnormal secretion of bile it was regarded as an important cause of the so-called bilious disorders. A sense of weight in the right hypochondrium, impaired appetite and digestion, sometimes nausea and vomiting, a bitter taste in the mouth, pain in the head, a coated tongue, and a sallow complexion, are symptoms often attributed to hepatic congestion. It is by no means certain that this pathological condition is represented by the foregoing symptoms. Active congestion doubtless occurs, but the agencies producing it and its morbid effects are not understood. The diagnosis with our present knowledge is not practicable, and the physician, therefore, in treating it acts upon the suspicion of its existence.

Chronic passive congestion, on the other hand, is a well-defined condition, the causation of which is understood. Anything which obstructs the current of blood through the hepatic veins, through the inferior vena cava above the entrance of the hepatic veins, through the lungs, or through the heart causes passive congestion. The obstruction is usually seated either in the heart or in the lungs. The various forms of uncompensated valvular lesions of the heart, particularly mitral and tricuspid lesions, insufficiency of the power of the heart's action due to myocarditis and obstructions in the pulmonary circulation, such as those resulting from emphysema, chronic pleurisy, fibroid phthisis, or tumors of the mediastinum, are the most frequent causes of chronic passive congestion.

In consequence of the damming back of blood upon the hepatic veins, first the central veins of the lobules, and next the lobular capillaries immediately surrounding these veins, become over-distended with blood. Thus the centre of the lobule presents a dark-red color, increased usually by the presence of brownish pigment-granules in the central hepatic cells. In contrast with this the periphery of the lobule is pale and yellowish from the comparative anæmia in that situation and from the presence of oil-drops in the peripheral liver-cells. A liver presenting the appearance described is called a nutmeg-liver. It is usually more or less enlarged, and is firm in consistence.

When the congestion is long continued and severe, the tissue at the centre of the lobule consists of little else than dilated blood-vessels, between which the liver-cells have become atrophied from compression. Whole lobules may be reduced to this cavernous tissue, which appears on section dark red and depressed. This change is called red or cyanotic atrophy of the liver. It is thus a late stage of the nutmeg-liver. The organ is diminished in size, the consistence is firm, and the capsule is usually thickened and opaque. Cyanotic atrophy is often associated with more or less increase of the interlobular connective tissue.

When the condition of chronic passive congestion is well marked during life, it is possible by means of palpation and percussion to make out enlargement of the liver. It may extend a hand's breadth below the free border of the ribs. There may be considerable variation from time to time in size, corresponding to variations in the amount of obstruction to the circulation.

The SYMPTOMS referable to the nutmeg-liver are not well defined. There may be a feeling of weight, but rarely of actual pain, in the right hypochondrium. A slight icteroid hue of the conjunctivæ and of the skin may be present, and this, combined with the cyanotic color, gives a characteristic aspect to many cases of heart disease and of emphysema. The obstruction to the flow of blood through the liver leads to congestion in the rootlets of the portal vein, causing passive congestion of the spleen, of the stomach and intestine, and of the peritoneum. In this way digestive disturbances and a certain amount of ascites are produced. These effects, however, belong to the general venous congestion which is present in most cases of nutmeg-liver, and only exceptionally are they out of proportion to other symptoms of general venous congestion. In the atrophic stage of the nutmeg-liver the symptoms of portal congestion become more marked.

The TREATMENT in cases of chronic passive congestion of the liver is directed not to the condition of this organ, but to the causes which obstruct the circulation; that is, to heart disease, emphysema, or some of the other causes already mentioned. Saline laxatives are useful in relieving temporarily the portal congestion.

Portal Thrombosis—Pylethrombosis—Adhesive and Suppurative Pylephlebitis.

Thrombosis of the main trunk of the portal vein or of its larger branches may be due to pressure from without, caused by enlarged glands, abscesses, tumors, impacted gall-stones, fibrous bands resulting from localized or general peritonitis or from a chronic inflammation of the surrounding connective tissue (portal phlebitis). It may be caused by foreign bodies within the vein or calcific plates resulting from chronic endophlebitis. It may be secondary to cirrhosis. A blow upon the abdomen has been known to cause portal thrombosis. In rare instances the thrombus is due to general weakness of the circulation in consequence of marasmus.

In recent cases the thrombus is grayish-red or yellowish in color and more or less adherent to the inner coat of the vessel. It either partially or completely occludes the vessel. In the so-called adhesive pylephlebitis the presence of the thrombus leads to secondary changes in the wall of the vessel, resulting in what is regarded as an organization of the thrombus. (Vide Part I. p. 29.) The thrombosed vessel is converted into a fibrous cord, either solid or canalized. In the latter case the circulation may be partially or completely re-established, and recovery takes place. If the main trunk of the portal vein be occluded, life is not prolonged sufficiently for the organizing process to take place.

The SYMPTOMS of adhesive pylethrombosis or pylephlebitis are referable to the mechanical obstruction of the portal circulation. If the occlusion be complete or nearly so, it gives rise to peritoneal dropsy. The rationale is the same as in dropsy caused by cirrhosis of the liver. From the latter it is distinguished by a more rapid accumulation of liquid, and by its occurrence in persons not addicted to spirit-drinking, this being the cause, *par excellence*, of cirrhosis. Hemorrhage from the stomach or intestine may occur, as in cases of cirrhosis, and sometimes this precedes the occurrence of dropsy. The spleen is more or less enlarged. The superficial abdominal veins may become dilated. Anorexia and loss of digestive power are symptoms occurring without much delay. Diarrhœa occurs frequently. The lower limbs and genitals become greatly œdematous. There is progressive loss of strength without fever, and death may take place within a few weeks by asthenia. Rapidly fatal asthenia may follow a profuse hemorrhage. Jaundice is often present. Its occurrence may be explained by the diminished pressure in the branches of the portal vein, or in some cases by a coincident obstruction of the bile-ducts from the same causes which induce thrombosis of the veins. In general, however, the substance and the functions of the liver are little disturbed by a simple portal thrombosis. This is due to the fact that, as has been shown by the experiments of Cohnheim and Litten, the blood carried by the hepatic artery is sufficient for the nourishment of the liver and for the secretion of bile.

The TREATMENT embraces, as an important palliative measure, paracentesis abdominis, repeated as often as the dropsical accumulation recurs. Measures to arrest hemorrhage are indicated when this occurs. Additional measures are those to meet other symptomatic indications and to support the powers of life.

Recovery has taken place in several cases under my observation, in which ascites and intestinal hemorrhage were referable to portal thrombosis.[1]

In pylethrombosis, as just described, or adhesive pylephlebitis, inflammation is limited to the site of the thrombus. The phenomena of the disease are referable to occlusion of the portal vein. *Suppurative pylephlebitis*, on the other hand, involves the presence of emboli or thrombi, which act, not only mechanically, but by the agency of a noxious principle. Owing to this principle they give rise to multiple hepatic abscesses. The clinical history is that of pyæmia, often without symptoms pointing directly to the liver. Pain and tenderness in the right hypochondrium, jaundice, and enlargement of the liver may be present. To these symptoms are added those of obstruction if, as may or may not be the case, the portal vein or its principal branches be occluded.

The most frequent causes of suppurative pylephlebitis relate to ulcerative and suppurative processes within the district of the portal circulation. In this category belong abscesses of the spleen, typhlitis and perityphlitis, ulceration of the gall-bladder, stomach, and intestinal tract, particularly dysenteric and sloughing ulcers, abscesses and ulcers of the rectum, such as sometimes follow surgical operations in that neighborhood, and inflammation of the umbilical vein in the newly-born infant. A foreign body in the portal vein may give rise to suppurative pylephlebitis. In an autopsy at Bellevue Hospital by Dr. Janeway a fish-bone two inches long was found, one half in the superior mesenteric and the other half in the portal vein. The portal vein contained puriform matter and the liver multiple abscesses. Another similar case has been reported. A thrombus in the portal vein is sometimes a direct continuation of a thrombus primarily formed in some of the rootlets of the vein at the diseased part. Sometimes the thrombosis follows the lodgment of an embolus broken off from a thrombus situated in some of the afferent branches of the portal vein.

In suppurative pylephlebitis the thrombus breaks down into a puriform mass from which emboli are detached, the vascular walls become infiltrated with pus-cells, and the suppurative process extends to the surrounding connective tissue and the hepatic parenchyma, the result being the production of abscesses in which the presence of bacteria can be demonstrated. The abscesses are usually not of large size, but they are many, and their distribution can sometimes be seen to follow the course of the portal vessels within the liver.

The DIAGNOSIS of suppurative pylephlebitis is to be based on the local symptoms already stated, in conjunction with the symptoms of pyæmia and the antecedent existence of some one of the causative conditions which have been named. Certain of the latter are not discoverable before death, and there are cases in which the post-mortem examination sheds no light on the etiology. The affection is then said to be idiopathic.

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[1] Vide paper entitled "Early Tapping in Cases of Ascites." (See note, p. 592.)

There is no ground for encouragement in the way of prognosis, and the treatment has relation solely to symptomatic indications and to support.

CHAPTER XIII.

DISEASES OF THE BILIARY PASSAGES.—JAUNDICE.

Inflammation of the Biliary Passages.—Dilatation of the Gall-bladder: Cancer of the Gall-bladder.—Jaundice, or Icterus.—Functional Affections of the Liver.

Inflammation of the Biliary Passages.

THE most frequent form of inflammation of the biliary passages is *simple inflammation*, or, as it is often called, catarrhal inflammation. The seat of the inflammation is usually the lower part of the ductus communis choledochus, particularly the part contained in the wall of the duodenum. The inflammation is in the great majority of cases simply a continuation of a similar inflammation affecting the stomach and the duodenum.

The anatomical changes in simple inflammation of the common bile-duct consist in hyperæmia and swelling of the mucous membrane and increased secretion of mucus. As in similar inflammations elsewhere, these changes may not be well marked after death, the congestion and swelling especially being often absent upon post-mortem examination.

The importance of acute simple inflammation of the common bile-duct consists in the obstruction which is offered to the flow of bile by the swelling of the mucous membrane and the accumulation of mucus. Inasmuch as the force which propels the bile through the biliary passages is slight, being chiefly due to the continued secretion of bile by the liver-cells and the movements of the diaphragm, even an insignificant obstruction suffices to impede its flow. The obstruction offered to the passage of bile causes an accumulation of bile in the ducts within the liver and an absorption of accumulated bile by the lymphatics. The bile thus absorbed enters the blood-current by way of the thoracic duct and causes jaundice.

The jaundice which is produced by simple inflammation of the intestinal part of the common bile-duct is usually called simple jaundice or catarrhal jaundice. Simple jaundice, caused in this way, is a very common affection. Being secondary to simple gastro-duodenitis, simple jaundice is usually preceded for a few days by moderate disorder of the gastric functions, such as anorexia, bad taste in the mouth, belching of gas, lassitude, and occasionally by vomiting. These symptoms continue for a time after the appearance of jaundice. The bowels are usually constipated.

The DIAGNOSIS is based chiefly upon the exclusion of other causes of jaundice, particularly the impaction of gall-stones, and severe structural affections of the liver, such as cirrhosis, acute yellow atrophy, tumors, etc. In general, the diagnosis is easily established.

The DURATION of an attack of simple jaundice is generally from about three to six weeks. Constitutional symptoms disappear, as a rule, a considerable time before the jaundiced color is lost.

The CAUSATION is that of gastro-duodenitis. In some cases there is evidence of some specific or infectious cause. This, at least, is the probable explanation of the occasional accumulation of cases of simple jaundice at certain times and in certain places, giving to the affection an epidemic or an endemic character.

Simple jaundice, if uncomplicated, is not a serious affection, and nearly always terminates favorably. It is to be remembered, however, that some cases of acute yellow atrophy of the liver begin with the symptoms of simple jaundice.

As the natural tendency of simple jaundice is to recovery, active medical interference is not indicated. The patient's diet should be regulated, fats especially being excluded. As the bowels are usually constipated, a cathartic may be occasionally administered, for which purpose some of the laxative saline waters, such as Carlsbad, may be employed. A time-honored remedy is calomel or blue mass, given at night and followed by a dose of salts in the morning. Devices for the displacement of the assumed plug of mucus from the bile-duct are of doubtful efficacy. Such devices are, pressure upon the gall-bladder, faradization over the gall-bladder, and the injection of large enemata of cold or lukewarm water with the intention of arousing intestinal peristole.

Nothing is known of simple inflammation of the bile-ducts within the liver as an independent affection.

Purulent inflammation of the biliary passages may be caused by the presence of gall-stones or of parasites, and is sometimes, though rarely, a complication of infectious diseases, such as pyæmia, typhoid fever, and cholera. Sometimes the inflammation is secondary to suppuration or necrosis of the tissues in the neighborhood of the ducts. Purulent inflammation of the bile-ducts outside of the liver may lead to ulceration and perforation of the affected portion, with consequent peritonitis. Purulent inflammation of the ducts within the liver leads to the formation of abscesses containing bile and usually biliary concretions.

Croupous and *diphtheritic inflammations* of the biliary ducts have been described, but they are rare and only of pathological interest.

The various forms of inflammation which have been described may attack the gall-bladder, with or without coincident disease of the bile-ducts. *Inflammations of the gall-bladder*, except those caused by the presence of gall-stones, which have already been described (p. 530), do not call for consideration in works the purpose of which is mainly clinical. Ulceration and perforation of the gall-bladder may occur independently of the presence of calculi. I have met with a case in which an abscess within the walls of the gall-bladder led to perforation and fatal peritonitis.

Dilatation of the Gall-bladder (Hydrops Cystidis Felleæ).

The two most common causes of obstruction of the biliary passages—namely, gall-stones and mucus-plugs—have already been described. Further reference will be made to these, as well as to other causes of obstruction to the flow of bile, in the article on Jaundice. Closure of the common bile-duct causes, in addition to jaundice, an accumulation of bile within the gall-bladder, but this accumulation is not generally sufficient to produce marked symptoms. Exceptionally, however, under these circumstances the gall-bladder becomes enormously distended with bile, as in a case related by Van Swieten in which the gall-bladder contained eight pounds of thick bile.

Some consideration is due to obstructions of the cystic duct. These do not lead to jaundice, but manifest their effects upon the gall-bladder. By far the most common cause of occlusion of the cystic duct is the impaction of one or more gall-stones. Other causes are stenosis from cicatrized ulcers, accumulation of inflammatory products, and pressure from without, as by tumors and peritoneal bands. Congenital stenosis is a rare condition.

As a result of occlusion of the cystic duct the gall-bladder becomes distended with a clear, usually somewhat viscid fluid, which is free from bile. This condition is called hydrops cystidis felleæ, although the fluid is not of dropsical origin, but is a secretion from the glands and mucous membrane of the gall-bladder. The gall-bladder, thus distended, forms a tumor, usually about the size of the fist, but sometimes as large as a man's head or even larger. In the latter case the tumor extends to the pelvis and occupies the greater part of the abdominal cavity.[1] The shape of the tumor produced by hydrops cystidis felleæ is globular or pyriform.

There are usually no SYMPTOMS, other than the presence of a tumor, and this in ordinary cases is not so large as to cause much inconvenience.

The DIAGNOSIS is based upon the situation and the characters of the tumor. The normal situation of the fundus of the gall-bladder corresponds to the median extremity of the ninth or tenth right costal cartilage. It can usually be ascertained by palpation and by percussion that the tumor projects in this situation from the lower margin of the liver. The tumor is generally movable, although it may be fixed by adhesions. It usually follows the respiratory movements of the diaphragm. Unless the tumor be greatly distended a sense of fluctuation can be appreciated. In exceptional cases there may be considerable difficulty in diagnosis, as when the colon makes its way between the upper part of the tumor and the abdominal wall, so that a tympanitic resonance may be found between the liver and the tumor. Cases are recorded in which the tumor has been so constricted as to resemble the kidney in shape, and, in fact, it has been mistaken for a wandering kidney. It suffices to call attention also to the possibility of mistaking a distended gall-bladder for an echinococcus cyst, a cancer, a hydro-nephrosis, or an ovarian cyst; but such errors are not likely to occur. An exploration of the tumor by a hypodermic syringe may be of assistance in diagnosis.

The TREATMENT must have reference to the circumstances in individual cases. In many cases no treatment is required. Aspiration of the gall-bladder may be employed if the degree of dilatation warrant this procedure. The fluid, however, is almost certain to be renewed. If it be decided to make a free opening into the gall-bladder, the same precautions are to be adopted with reference to the formation of peritoneal adhesions and the avoidance of septic inflammation as in making similar openings into abscesses and hydatid cysts of the liver.

Sometimes, instead of a clear, bland fluid, pus accumulates in the gall-bladder (empyæma cystidis felleæ). The affection is then, of course, of a very grave nature, and is likely to terminate fatally. If the diagnosis can be made, the evacuation of the pus by a free incision is indicated.

Primary cancer of the gall-bladder occurs, but in its symptoms and course it cannot be discriminated with any certainty from cancer of the liver, and it therefore does not require separate clinical consideration.

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[1] In a case described by Erdmann the amount of fluid is said to have been from sixty to eighty pounds.

The parasites which are sometimes found in the biliary passages have already been considered (p. 559).

Jaundice, or Icterus.

The presence of bile-pigment in the blood and lymph in sufficient quantity to give to the conjunctiva and to the skin a yellow color, greater or less in degree, constitutes the morbid condition known as *icterus*, or *jaundice*. The term *cholæmia* denotes the presence of bile in the blood, and under this head, in treating of the morbid conditions of the blood in Part I., was embraced the general pathology of the affection to be now considered (p. 77). The terms icterus and jaundice, of which the latter is the more commonly used, relate to yellowness of the surface from the presence of bile-pigment, whereas cholæmia implies the presence of all of the bile-constituents in the blood.

Jaundice is strictly never an individual disease. It is merely an effect or a symptom of disease; but as it is an obvious and striking morbid condition dependent on various affections, and occurring sometimes when its causation is not evident, it is convenient to give it separate consideration. This removes the necessity of much repetition in treating of the manifold affections of which jaundice is a symptom.

The presence of bile in the blood, or cholæmia, is a result of the entrance into the blood of bile after its secretion. The bile-pigment and the biliary salts (the glycocholate and the taurocholate of soda) are formed within the liver, and do not pre-exist in the blood. The bile, however, contains at least one excrementitious principle—namely, cholesterin. The latter constituent of bile accumulates in the blood if the secretion of the liver be from any cause suspended.

Whether the bile-pigment be ever formed in the circulating blood is a matter of uncertainty. Hæmatoidin, a pigment identical with bilirubin, is often formed in old blood-extravasation; and this fact, together with other facts, has given rise to the supposition that in certain cases of jaundice the bile-pigment is formed outside of the liver, probably in the circulating blood. Jaundice thus produced is called hæmatogenous jaundice, in distinction from that caused by the absorption of bile, or hepatogenous jaundice. The hypothesis of a hæmatogenous jaundice is advanced to explain certain cases of jaundice in which none of the recognized causes of the absorption of bile from the liver have been found. A hæmatogenous origin has been urged especially for the jaundice occasionally occurring in certain infectious diseases, such as septicæmia, pyæmia, remittent fever, and certain cases of poisoning, as by phosphorus and the mineral acids, in which destruction of red blood-corpuscles doubtless occurs. It must be admitted, however, that there are serious objections to the acceptance of the theory of a hæmatogenous jaundice, and that this theory has of late lost, rather than gained support. Hæmoglobin, which is set free by destruction of red blood-corpuscles in the circulating blood, is eliminated by the kidneys in the form of meth-hæmoglobin, and there is no proof that it is ever converted within the blood into bile-pigment, although dissolved hæmoglobin increases the formation of bile-pigment within the liver. There is a tendency to refer many cases which some writers have regarded as hæmatogenous jaundice—for instance, the jaundice of many acute infectious diseases—to a mild inflammation of the large bile-ducts; but the anatomical proof of this view is deficient for a considerable number of the cases. Nor is the theory that the absorption of bile in these cases is the result of lowered blood-pressure in the liver to be regarded as established. It must be admitted, therefore, that while a certain number of the cases of jaundice formerly considered as hæmatogenous are in reality of hepatogenous origin, we possess no satisfactory explanation of the cause of the jaundice in the remaining cases.

In all cases in which we possess an adequate explanation of the causation the cause is to be found in some obstruction to the flow of bile in its passage from the liver to the intestine. The causes of such obstruction are various, and they may be classified—*first*, as abnormalities in the interior of the bile-ducts; *second*, as alterations in the walls of the ducts; and *third*, as lesions causing pressure upon the bile-ducts from without.

The most frequent of the first of these causes are plugs of mucus resulting from simple inflammation of the common bile-duct secondary to gastro-duodenitis (simple or catarrhal jaundice) and gall-stones, including concretions of inspissated bile. Lodgment of parasites in the bile-ducts is a rare cause of jaundice. These parasites are lumbricoid worms, the various forms of distoma and echinococcus. Pus, blood, and fibrin may block up the bile-ducts in rare instances.

Of the changes in the walls of the bile-ducts which may encroach on the lumen of these tubes, the most important are inflammatory swelling, either that of acute or of chronic inflammation, cicatricial thickening, and contraction following ulceration and new growths. Congenital stenosis of the bile-ducts is a rare condition.

The most important of the causes which produce jaundice by external pressure on the bile-ducts are tumors and inflammatory products. Cancers of the liver, of the gall-bladder, of the lymphatic glands in the porta of the liver, of the pylorus, of the duodenum, and of the head of the pancreas, may all cause jaundice by compression of the common bile-ducts. Less frequently cancer in other situations and other forms of tumor produce jaundice in a similar way. Hepatic abscesses and echinococcus cysts may compress the main bile-ducts. The pressure of the gravid uterus and of fecal accumulations in the transverse colon belongs in this group of causes. Obstruction may also be due to peritoneal adhesions, which cause traction on the peritoneal fold which contains the common bile-duct. The compression of a large number of intra-hepatic ducts in cirrhosis of the liver leads to a certain amount of jaundice.

The quantity of smooth muscular tissue in the walls of the bile-ducts appears to be too slight to support the theory of jaundice produced by spasm of the ducts. Although it is possible, it cannot be considered proven, that bile may be absorbed, and that jaundice be thus caused by diminution of the blood-pressure in the liver. This, however, is the usual explanation of the jaundice occurring in cases of portal thrombosis. As has already been mentioned, bile is not absorbed in cases of jaundice directly by the blood-vessels, but it is first taken up by the lymphatics in the liver, is carried to the thoracic duct, and thence enters the blood. Another supposed agency in producing jaundice in certain cases is enfeebled respiration. This is based upon the idea that the movements of the diaphragm aid in the propulsion of the bile, so that when these are weakened there may be a sufficient stasis of bile in the bile-ducts for the production of jaundice.

The jaundice which frequently occurs in from two to four days after birth, and is called icterus neonatorum, has not received any explanation which has met with general acceptance. This jaundice is peculiar in the fact that the urine is generally free from bile-pigment, and that the blood after death, as well as the papillæ of the kidneys, and sometimes other organs, contain a large quantity of crystalline pigment.

Yellowness of the conjunctiva and skin takes place after a certain amount of bile has entered the circulation. Prior to the appearance of jaundice the bile-pigment may be found in the blood, the urine, and the liquid of serous cavities. The coloration of the surface depends on the presence of the bile-pigment in the transuded liquid which infiltrates the tissues. Other things being equal, the intensity of the yellow color is proportionate to the degree of the cholæmia. The presence of the bile-pigment in the serum of the blood is shown by the yellow color of the liquid beneath the epidermis if a blister be applied.

The urine in cases of jaundice contains bile-pigment in abundance, very often hyaline casts and sometimes epithelial casts. Not infrequently a small quantity of albumen is present in the urine. The presence of the pigment is apparent to the eye, and it gives to linen a yellow stain. The perspiration contains it, and the body-linen may also be stained yellow, especially from the abundant cutaneous secretion in the axilla. Bile-pigment is rarely, if ever, contained in the saliva, nor, as a rule, is it found in mucus, but it may be found in the sputa of pneumonitis developed in a person affected with jaundice. The milk of nursing women rarely contains it, although its presence in this secretion has been observed. The humors of the eye do not contain it, except in some cases in which the jaundice is unusually intense. The "jaundiced eye" (xanthopsia) is so rare an event that the phrase is to be regarded as a poetical license.

The tissues in the interior of the body are more or less colored. The coloration is seen especially in the areolar tissue, the serous membranes, and the muscles. The substance of the brain escapes, except in cases in which it is unusually infiltrated with serum, that is, œdematous.

Bile in the blood acts as a sedative on the circulation and nervous system. The heart's action is usually diminished in frequency. The pulse frequently falls to 50, 40, or even lower, in cases in which the jaundice is not associated with acute inflammation or fever, and if thus associated the frequency of the pulse is less than if jaundice did not coexist. The effect is attributed to the presence in the blood, not of the pigment, but of the biliary salts. The temperature of the body may be lowered. The respirations diminish in frequency in proportion to the effect on the circulation. The mental faculties are dull and there is a disposition to somnolency. Pruritus of the surface, especially in the axilla and the inguinal region, troublesome particularly at night, is an occasional symptom. It is sometimes a source of great annoyance. An occasional complication is urticaria, denoted by the characteristic elevations on the skin, or wheals, which present a deeper yellowness than the surrounding integument and occasion intense pruritus.

The symptoms referable to the digestive system are frequently due to coexisting duodenitis or gastro-duodenitis. Anorexia, nausea, and sometimes vomiting, thirst, more or less pain in the neighborhood of the epigastrium, and tenderness in that situation, are symptoms denoting subacute inflammation of the gastro-duodenal membrane. In cases of jaundice disconnected from this affection the appetite may be but little or not at all impaired, and certain articles of food are apparently digested without difficulty. Fatty food is imperfectly digested, and chemical examination of the evacuations from the bowels shows fat in more or less abundance. Patients have an antipathy to fatty articles of diet. The nutrition for a time may be but little affected, but sooner or later, loss in weight takes place; and this is progressive if the jaundice continue.

The appearance of the dejections is important, as affording evidence of the completeness or otherwise of obstruction to the passage of bile into the intestine. If complete occlusion exist, the stools are ashy or clay-colored, owing to the presence of fat. On the other hand, they have more or less of the yellowish or brownish color of health if the obstruction be only partial. The feces, when the intestines are devoid of bile, emit an odor of putrescence, and the formation of gas is frequently troublesome, bile in the intestinal canal being an antiseptic as regards the fecal contents. Generally, the bowels are constipated, but this is by no means always the case, even when complete occlusion exists.

Jaundice may exist for a greater or less period without giving rise to notable disturbance. The system sometimes tolerates the accumulation of bile for a long time. Not infrequently, patients are able to be up and about as in health, experiencing but little inconvenience. The symptoms and the gravity will depend in a great measure on the pathological conditions with which the jaundice is associated. If the jaundice depend on obstruction in connection with subacute gastro-duodenitis, the prognosis is always favorable. Simple gastro-duodenal jaundice usually continues for from three to six weeks. If it depend on the impaction of a gall-stone in the common hepatic duct, the recovery will depend on the passage of the stone into the intestine or backward into the gall-bladder. Permanent obstruction from a gall-stone or the pressure of a tumor involves of course persistence of the jaundice. Under these circumstances serious results may not take place for a considerable or even a long period. Cases have been under my observation in which the nutrition of the body was not greatly impaired for one, two, and even four years. In one case under my observation the jaundice had persisted for eight years. Life has continued for a much longer period. Sooner or later, however, the body wastes, the patient becomes anæmic and cachectic, the vital powers gradually fail, and the termination is fatal, irrespective of either the morbid condition giving rise to the jaundice or of intercurrent affections of other organs than the liver. The color of the skin becomes dark or almost black in some cases of long-persisting jaundice. In the majority of cases of jaundice from permanent obstruction, however, the duration of life does not extend beyond several months.

When life is destroyed purely in consequence of the retention of bile, the liver takes on important lesions. The bile-ducts within the liver become dilated; the gall-bladder may or may not become distended with bile; the liver is at first increased in volume from the accumulation of bile, but subsequently its volume is diminished and it becomes flabby and wrinkled. There is often a new growth of interstitial tissue, constituting one of the forms of biliary cirrhosis of French writers. The persisting distension of the hepatic bile-ducts at length leads to an arrest of the secretion of bile. In proportion as the secretion of bile is arrested the jaundice diminishes, and the patient suffers from the retention in the blood of the excrementitious portion of the bile—namely, cholesterin. In some cases death is preceded by convulsions and coma. In these cases *cholesteræmia* may be supposed to exist, but these phenomena in a certain proportion of cases are due to *uræmia*. Post-mortem examinations show an accumulation of granules of bile-pigment in the uriniferous tubes when jaundice has existed for a considerable or long period; and the uræmia proceeds from the morbid condition of the kidneys. Hydroperitoneum may occur in the latter period of the affection, and general dropsy is sometimes a result of the superinduced renal affection.

The DIAGNOSIS of jaundice is readily made if the examination of the patient be made in the daytime, but the yellowness may not be apparent by artificial light. The yellow tint of the surface seen in some cases of cancer, intermittent fever, and chlorosis is readily discriminated from slight jaundice by the absence of yellowness of the conjunctiva, the latter being always present in jaundice; but to determine the causative pathological conditions in individual cases is not always easy or in all cases practicable. The dependence on duodenitis or gastro-duodenitis is to be inferred from the symptoms of these affections preceding and accompanying the jaundice—namely, anorexia, nausea, vomiting, and tenderness over or in the neighborhood of the epigastrium. The presence of a gall-stone may be inferred from the pre-existence and coexistence of the symptoms of hepatic colic. A tumor which presses upon the common duct may be discoverable by manual exploration. From the character of the tumor and the coexisting symptoms its cancerous nature may sometimes be inferred. Occlusion as a result of inflammation of the ducts exclusive of duodenitis, from lumbricoid worms in the common duct, and from other causes, cannot be determined during life. The existence only of obstruction is determinable in such cases. Not infrequently the distended gall-bladder may be felt, and this is positive evidence that the jaundice involves obstruction of the common duct.

In the TREATMENT of jaundice it has been the custom to prescribe active cathartics and emetics under an idea of removing the obstruction by their operation. If, however, the jaundice be connected with duodenitis or gastro-duodenitis, not only are these measures ineffectual, but they can hardly fail to do harm. If the obstruction be caused by a gall-stone, its passage will not be hastened by these measures; and they will weaken the patient without doing good if the jaundice depend on a tumor pressing on the common or hepatic duct. As remedies, then, addressed to the obstruction giving rise to the jaundice, cathartics and emetics are contraindicated.

The treatment of jaundice in the majority of cases is to be addressed to a subacute inflammation of the stomach and duodenum. Laxatives or cathartics are required only for the relief of constipation, and when required the mildest forms are to be preferred. There is no ground for the supposition that mercury exerts any special efficacy, but a few grains of calomel and blue mass frequently prove as mild and effective as any form of cathartic or laxative, followed, if necessary, by a saline draught. The latter alone will often suffice. The saline mineral waters, such as the Congress or Kissingen water, will frequently secure an adequate laxative effect. Remedies to soothe the inflamed mucous membranes are useful, such as small doses of morphia or some of the succedanea of opium—namely, belladonna, hyoscyamus, conium, etc. Conium has been considered as a remedy having special efficacy in jaundice. Its apparent efficacy may be accounted for, in part by its usefulness as a soothing remedy and partly by its supplanting active cathartics and emetics, which are injurious. The diet should be bland and digestible. Fatty articles of food should be excluded, and to these the patient generally has an antipathy. A blister over the epigastrium has seemed to me to be useful. Accidental symptoms will of course claim appropriate palliative measures. If a distended gall-bladder can be felt, pressure upon it will sometimes remove the obstruction, the tumor disappearing under this procedure. This fact I have verified. Pressure, however, should not be made with such force as to incur risk of rupture. Gerhardt has found the induced current of electricity to be useful by exciting spasmodic contractions of the muscular coat of the gall-bladder.[1] A bubbling sound, caused by the passage of the bile into the duodenum, may sometimes be heard occurring with the disappearance of the tumor.

In cases of jaundice connected with the passage of gall-stones the palliation of pain is the chief object of treatment. The occurrence of jaundice does not add indications to those which have been already considered as pertaining to the passage of gall-stones or hepatic colic. (Vide p. 533.)

Jaundice dependent on irremediable causes, such as permanent occlusion of the biliary ducts or the pressure of a tumor, does not admit of cure, and the existence of an irremediable cause is to be inferred when the jaundice persists for several months or years. Yet I have repeatedly met with cases in which, after the persistence of jaundice for many months, recovery at length took place, and I have known recovery to take place after a persistent jaundice for several years. A fecal tumor in the transverse colon and a movable right kidney have been known to give rise to jaundice by pressing on the common duct. A fecal tumor may be removed, but, in general, tumors so situated as to produce obstruction are persisting. In cases not admitting of cure the objects of treatment, as regards the jaundice, are the elimination of bile by the renal and cutaneous emunctories and the support of the powers of life in order to postpone as long as possible the fatal termination. With a view to the elimination of bile, the action of the kidneys may be excited by the vegetable diuretic remedies, and the functions of the skin maintained by the use of the warm bath and by guarding against vicissitudes of temperature. The powers of life are to be supported by a nutritious diet, together with all the hygienic means, moral and physical, of invigorating the system.

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[1] *Ueber Icterus gastro-duodenalis*, von C. Gerhardt, Leipzig, 1871.

In cases of jaundice ending in recovery it is to be borne in mind that the yellowness of the skin continues for some time after the absorption of bile has ceased. The coloring matter, being outside of the vessels, remains when the cholæmia no longer exists. Of the continuance of the cholæmia or of the condition which occasioned the jaundice the urine affords the most reliable evidence. The affection is to be considered as having ended when the coloring matter ceases to be apparent in the urine. I have known yellowness of the skin to persist for several months after the disappearance of bile-pigment from the urine. In the case in which this was observed the jaundice had existed for a year. The absorption is probably slow in proportion as the jaundice has been of long duration.

Functional Affections of the Liver.

Functional affections of the liver have hitherto held a prominent place in pathological speculations, and they are still often mentioned by physicians in conversing with each other and with their patients; but in a clinical point of view, with our present knowledge, the importance of the functional affections of the liver is in striking contrast with the frequency with which this organ is referred to as the source of morbid phenomena.

It is customary with many to refer a certain group of symptoms to congestion of the liver. The nature of these symptoms and the propriety of considering them as dependent upon hepatic congestion have already been discussed in the article on Congestion of the Liver (p. 625).

Physiology teaches us that the liver is endowed with various functions, of which the most important are the secretion of bile, the production and perhaps the transformation of glycogen, and the metamorphosis of the albuminous and other constituents of the products of digestion which are absorbed by the portal vein.

The action of the liver upon the products of digestion which are absorbed by the rootlets of the portal vein, although not well understood, is probably an important one. The liver seems to be an important, although not the sole, seat of the formation of urea. It may be believed, therefore, that structural and functional disorders of the liver interfere with the alterations which the absorbed products of digestion normally undergo in the liver, but our knowledge of this subject is too imperfect to admit of more than profitless speculation.

The views concerning abnormalities in the glycogenic function of the liver have already been considered in Part I. (p. 70). There remain, therefore, to be considered only the functional disturbances in the secretion of bile.

Functional disorders of the biliary secretion are a morbid excess, a morbid deficiency, and a vitiation of the bile. A morbid excess of bile is supposed to enter into the etiology of diarrhœa, colic, and sporadic cholera in certain cases. Deficiency of bile is often treated as a functional affection under the name of *torpor of the liver*, remedies being given to increase the biliary secretion. A whitish or ashy color of the stools probably is evidence of deficient secretion, although this appearance may arise from undue absorption of bile from the intestinal canal. Every practitioner is familiar with this appearance of the stools in young children and after the administration of opium. The production of the so-called *bilious stools* by the use of mercury is well known. Torpor of the liver is a phrase often applied to cases in which the morbid condition is obscure. It affords an acceptable explanation to patients, owing to the popular notion that the liver plays an important part in the ailments referable to digestion and to the nervous system; and, whatever may be the rationale, a mercurial cathartic or laxative often affords relief of the symptoms, which are vaguely called *bilious*. A deficiency of precise knowledge respecting excess and deficiency of the secretion of bile as functional disorders must be confessed, but as regards vitiation of bile the deficiency of knowledge is still greater. Acrid bile is supposed to be sometimes secreted, and the greenish color of the stools, often observed, especially in children, has been regarded as denoting a morbid quality of bile. It is not improbable that a vitiated bile may be secreted, but it is impossible to determine whether morbid changes be due to a faulty secretion or to causes acting within the alimentary canal; and nothing can be more vague than to regard a vitiated secretion of bile as a functional disorder furnishing therapeutical indications.

CHAPTER XIV.

DISEASES OF THE SPLEEN.—DISEASES OF THE PANCREAS.

Diseases of the Spleen.

MOST of the diseases of the spleen are secondary to other diseases, and they are therewith considered. It is desirable, however, to bring together the various affections of the spleen, although few of them require detailed consideration as independent diseases. The spleen bears to the blood-current much the same relation that the lymph-glands do to the lymphatic current. This explains the frequency with which the spleen is involved in the various blood diseases, particularly in those characterized by the presence of infectious substances.

Acute congestion of the spleen is one of the morbid elements in the production of acute splenic tumor, to be presently considered.

Chronic passive congestion of the spleen is caused by mechanical interference with the portal circulation. The most important of its causes are pylephlebitis, cirrhosis of the liver, compression of the portal vein by tumors, cardiac disease, emphysema of the lungs, large effusions in the pleural cavity, and intrathoracic tumors. The spleen is enlarged, dark red in color, firm in consistence, and its capsule is usually thickened. The enlargement can usually be determined by physical examination, and this is often of considerable assistance in diagnosis. There may be complaint of a feeling of weight and tension in the left hypochondrium, but in general no definite symptoms are referable to chronic passive congestion of the spleen.

The spleen, next to the kidneys, is the most frequent seat of *embolic infarctions*. The emboli are usually derived from the left side of the heart, but they may come from any part of the aorta above the origin of the splenic artery. The branches of the splenic artery do not anastomose with each other, so that the mechanical effect of an embolus in this organ is the production of an infarction. Splenic infarctions are wedge-shaped, with the apex directed toward the hilum of the spleen. Splenic infarctions may be hemorrhagic, white, or mixed infarctions. Most frequently the centre is white and the periphery is hemorrhagic. They are on the average of larger size than renal infarctions. It is said that sometimes the lodgment of an embolus in the spleen is attended by a sensation of pain, but this is not proven; nor in fact are we able to refer any symptoms to the bland infarctions of this organ. If life be sufficiently prolonged, an embolic infarction terminates in the formation of a fibrous cicatrix which may contain blood-pigment and lime salts.

If the embolus contain infectious substances, generally in the form of micro-organisms, it produces, in addition to its mechanical effects, suppuration leading to the formation of an abscess. In rare instances a gangrenous focus is produced by an infectious embolus.

Embolic *abscesses* are produced in many cases of acute ulcerative endocarditis and in pyæmia. Such abscesses are generally of small size and do not admit of positive diagnosis. The symptoms are those of the diseases named. These diseases are usually attended by more or less enlargement of the spleen, whether abscesses be present in this organ or not. Embolic abscesses may, however, attain a large size, especially by the coalescence of a number of abscesses. Splenic abscess may be produced independently of emboli. It may be caused by wounds of the spleen. It rarely follows severe injuries without wounds which involve this organ. It occurs sometimes as a complication of various infectious diseases, such as relapsing fever, typhoid fever, and intermittent fever, where an embolic origin cannot be demonstrated. Abscess may be caused by perforation of a gastric ulcer into this organ or by extension of suppuration from adjoining parts. Sometimes no cause can be determined for an abscess in the spleen. The size of the abscess may be enormous. Sometimes all of the splenic tissue is destroyed and only the capsule remains. The abscess may burst in different directions, as into the peritoneal cavity, the stomach, the intestine, the chest, and externally. Except when the abscess presents itself as a fluctuating tumor a positive diagnosis from symptoms and signs is impracticable. An exploratory puncture may aid in the diagnosis by demonstrating the presence of pus. The prognosis and treatment in cases of splenic abscess are essentially the same as in abscess of the liver. (See p. 598.)

Acute splenic tumor is one of the most important pathological changes of the spleen. By this name is designated the enlargement of the spleen which forms one of the most frequent and essential lesions of many acute infectious diseases. Nearly every acute infectious disease may at times be accompanied by splenic enlargement, but there are certain of these diseases in which swelling of the spleen is more constant than in others. Acute splenic tumor is nearly always present in relapsing fever, typhoid fever, typhus fever, and intermittent and remittent fevers. It is usually or frequently present in pyæmia and septicæmia, including puerperal fever, in epidemic cerebro-spinal meningitis, erysipelas, acute lobar pneumonitis, diphtheria, acute yellow atrophy of the liver, acute miliary tuberculosis, the acute exanthematous fevers, etc. It has been observed during the primary and the secondary periods of syphilis.

The spleen is enlarged, often twice or thrice its normal size; its consistence is soft, its color dark red or bluish-red; and upon section the pulp swells above the surface. The Malpighian bodies are generally invisible, but they may be distinct or even enlarged. The enlargement is due to active congestion and to an increase in the number of cells in the spleen, some of which, at least, are produced by proliferation of the pre-existing cells. The lesion, therefore, in acute splenic tumor is an acute hyperplastic splenitis. In splenic swellings of short duration probably the change does not pass beyond active congestion.

The acute splenic tumor of infectious diseases is believed to be due to the accumulation in the spleen of noxious substances present in the blood, these substances acting as an irritant and causing congestion and inflammation. That these substances, in many cases at least, are micro-organisms seems certain. The accumulation of these organisms, as well as of fine particles in general, is explained by the peculiarities of the circulation in the spleen, which render this organ a kind of filter for the blood, as the lymphatic glands are for the lymph.

Clinically, the chief importance of acute splenic tumor is the aid which its recognition affords in diagnosis. In rare instances the swelling of the spleen has led to rupture of the organ and fatal hemorrhage into the peritoneal cavity. This is an accident which has occurred in typhoid fever, intermittent fever, and other acute infectious diseases.

Under the name of *chronic splenic tumor* are included enlargements which occur in a variety of chronic diseases, of which the most important are malarial fever, leucocythæmia, and pseudo-leucocythæmia. A similar enlargement is met with in some cases of syphilis. The enlargement is due to a chronic hyperplastic splenitis, but the morbid appearances vary in different diseases; in some cases the hyperplasia affecting especially the trabecular tissue, and in others affecting uniformly the cells and the trabeculæ. These differences are considered in connection with the different diseases in which chronic splenic tumor occurs.

Chronic interstitial splenitis occurs sometimes without apparent cause, although in most cases it is referable either to malaria or to syphilis.

Enlargement of the spleen, acute or chronic, is not attended with much pain or with tenderness on pressure. If not greatly enlarged, there may be no local symptoms directing attention to an affection of the organ. If much enlarged, its weight occasions a sense of uneasiness and tension in the left hypochondrium. Disconnected from the pathological associations under which it is generally presented, it may exist without any notable general symptoms.

Splenic enlargement is generally ascertained without difficulty, by palpation and percussion. In some cases the enlarged organ extends upward, pushing up the diaphragm, and causing an evident projection of the false ribs of the left side of the chest. Its upper border is accurately indicated by the line of demarcation between pulmonary resonance and flatness on percussion, and the lower border is determined, approximately, by finding the upper limits of tympanitic resonance. In other cases the organ is dragged downward by its weight, and it may descend as low as the brim of the pelvis. Under these circumstances, if the abdominal walls be thin and relaxed, the organ may be grasped by the hand and restored to its normal position. The form will serve to distinguish it from the other abdominal tumors. The notches on the anterior border may sometimes be felt through the abdominal walls. An enlarged spleen is depressed by the act of inspiration and recedes in the act of expiration. Its normal limits in the left infra-axillary region are the ninth and eleventh ribs. When enlarged, its lower border extends downward and anteriorly. Pulsation of an enlarged spleen, in connection with aortic regurgitation, has been observed.

The TREATMENT of acute enlargement involves the indications belonging to the infectious diseases with which it is associated. Associated with malarial fever, experience shows a special influence of large doses of quinia, salicin, and other antiperiodic remedies upon the spleen. These remedies are also indicated in chronic malarial enlargement. There is ground for attributing to quinia, given in full doses, an effect upon the enlargement in other infectious diseases than malarial fever. The treatment in chronic enlargement not due to malaria has reference to the associated diseases—namely, cirrhosis of the liver, leucocythæmia, etc. There is no special course of medication to be pursued. Generally, chalybeate tonics, continued steadily for a long time, are useful. They are indicated in proportion as the symptoms and signs denote anæmia. Changing the form of the chalybeate tonic from time to time is important. DaCosta has reported a case of splenic leucocythæmia in which an apparent cure was effected by the hypodermic injection of ergot.[1]

Extravasation of blood sometimes occurs in connection with vascular engorgement of the spleen, and the hemorrhage may lead to rupture of the capsule and the escape of blood into the peritoneal cavity. This has been observed in cases of intermittent fever, of typhoid fever, and during an epileptic paroxysm. It also occurs in connection with pregnancy, both before and after labor. Death may take place immediately from the loss of blood, and in most of the cases which have been observed the duration of life after the rupture has not exceeded twenty-four hours. The symptoms resemble those which follow intestinal perforation, and the differential diagnosis cannot be made with positiveness. Were it practicable to recognize the accident by the symptoms, the treatment would be the same as in cases of perforation—namely, opium in full doses, with perfect quietude of body. It would be proper to apply cold over the abdomen. Nothing is to be expected from treatment beyond palliation.

The different structural affections which have been considered as seated in the liver are sometimes found in the spleen—namely, waxy degeneration, syphilitic growths, tuberculosis, cancer, and hydatids. These may involve the whole or only a portion of the organ. They occasion enlargement, in some instances giving rise to splenic tumors, properly so called, of greater or less size.

Waxy degeneration of the spleen rarely exists except in connection with a similar affection in other situations, as in the liver, kidneys, and intestinal canal. There are two forms of waxy spleen—namely, the sago spleen and the diffuse waxy spleen. In the former the Malpighian corpuscles are chiefly affected, in the latter the splenic pulp. The enlargement of the organ, as a whole or in part, is sometimes very great. The diagnosis is to be based on the affection of other organs and the causative conditions—namely, suppurative disease of bones or cachexiæ, especially syphilis.

Tuberculous disease may be manifested anatomically either by miliary granulations or by cheesy collections called solitary tubercles. The former are almost invariably present in the acute miliary tuberculosis of adults and in tuberculous disease affecting children. The spleen is very rarely the seat of a primary tuberculous affection. The organ becomes in some cases moderately enlarged. Pain and tenderness are rarely present, and the enlargement is generally the only evidence that this organ participates in the tuberculous disease.

Cancer of the spleen is never primary, and its occurrence is infrequent. It is always secondary to cancer of the liver or of some other of the abdominal organs. Cancer is to be inferred from the development of splenic enlargement or tumors in the course of cancer seated elsewhere.

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[1] Vide *Am. Journ. of Med. Sciences*, January, 1875.

Syphilitic affections of the spleen in the form of gummata, causing enlargement or appreciable tumors, are to be diagnosticated by taking into account other antecedent or coexisting evidences of syphilis, and by excluding waxy degeneration. As stated by Mosler, syphilitic affections in this organ "are more frequently the object of anatomical than of clinical observation."

Hydatid tumors in the spleen are rare. Their history and the means of diagnosis are the same as when they are seated in the liver. (Vide p. 618.)

With reference to the anatomical characters, pathology, prognosis, and treatment of the foregoing structural affections of the spleen, it suffices to say that in these regards they are the analogues of the corresponding hepatic affections.

Our present knowledge does not enable us to understand fully the consequences of the impairment or loss of the functions of the spleen. It is well known that this organ may be removed from inferior animals without the destruction of life or serious injury to health. This fact goes to show that whatever may be its functions, they are adequately performed by other organs in its absence. There is reason to believe that this statement is applicable to man as well as to inferior animals. A case was communicated to me in 1861 by Dr. John L. Alston of Texas, in which, after an abdominal wound by a musket-ball, a portion of the spleen protruded three and a half inches in length and two and a half inches in width. The protruding portion was ligated and it dropped off on the fourth day. Recovery took place rapidly, and the patient was in a short time discharged from the hospital entirely well. Cases have been reported in which the entire spleen, having escaped through a wound, was removed, recovery taking place and the health remaining good for years afterward. There have been several cases recorded of congenital absence of the spleen without apparent detriment to health. Within the last twenty-five years several surgeons have ventured upon the removal of this organ for great enlargement. In the majority of instances patients have died from the immediate effects of the operation. In view of the danger the operation is not warrantable.

The spleen is sometimes *dislocated* downward as an effect of tight-lacing or other causes not always apparent (wandering spleen). The organ may or may not be enlarged. It is important to distinguish between the dislocated organ and simple enlargement. If dislocated, the organ is to be restored by pressure with the hand and retained in its normal situation by an appropriate mechanical supporter.

Affections of the Pancreas.

Owing to their infrequency and to their obscurity as regards diagnosis, affections of the pancreas require in this work but a brief notice. This gland may be the seat of inflammation, acute or chronic. Of all the glandular organs—liver, ovaries, testes, kidneys, etc.—the pancreas is perhaps the least liable to become inflamed. Acute *pancreatitis* has been found, on examination after death, to occur in cases of continued fever, of puerperal fever, of pyæmia, and apparently as a result of the employment of mercury. The morbid appearances denoting acute inflammation are hyperæmia, softening, enlargement, and suppuration. Gangrene has been observed, and even the discharge of necrosed portions of the pancreas by the rectum. The symptoms which have been observed are, pain referable to the epigastrium, vomiting, diarrhœa, chills, and more or less febrile movement. The data for determining the clinical history of the affection are insufficient. A discharge, by vomiting and stool, of a liquid resembling saliva, supposed to be the pancreatic secretion, has been thought to be a diagnostic symptom, but there is no reliable evidence of the correctness of this opinion. The presence of fat in the stools has been supposed to be a diagnostic symptom. The diagnosis, with our present knowledge, is impracticable. An abscess of the pancreas has been known to open into the stomach. Chronic pancreatitis is, if possible, even more obscure, as regards diagnostic symptoms, than the acute form of the disease. In a case observed by Wilks death followed extreme emaciation without any symptoms pointing to the seat of the disease.[1] From what has been stated with regard to the diagnosis, it follows that it is impossible to form an opinion as to the proportion of cases in which either acute or chronic pancreatitis ends in recovery.

The pancreas may be the seat of either primary or secondary *cancer*.[2] Primary cancer most frequently begins at the head of the pancreas. It is usually of the scirrhous variety. Medullary cancer, colloid cancer, and cylindrical-celled epithelioma also occur in this situation. Enlargement of the head of the pancreas from chronic inflammation, cancerous disease, or the formation of cysts may constitute a tumor discoverable by manual exploration. The diagnosis involves its discrimination from other tumors in the same situation. It is most likely to be confounded with aneurism of the aorta and cancer of the pylorus. A pulsation may be communicated to the tumor from the aorta, and this is suggestive of aneurism. The diagnostic symptoms and signs of aneurism of the abdominal aorta are to be sought for, and this affection may be excluded by their absence. A positive discrimination between cancer of the pancreas and cancer of the pylorus can scarcely be made. The main points which weigh in favor of cancer of the pancreas are, less prominence of gastric symptoms than in cancer of the stomach (vide p. 431), the presence of jaundice, fatty stools, and sugar in the urine. The connection between structural disease of the pancreas and fatty diarrhœa has been considered in treating of the latter. (Vide p. 510.) The presence of free fat in the alvine dejections, taken in connection with a tumor supposed to be an enlarged pancreas, is undoubtedly a point of much weight in the diagnosis. The absence of fatty dejections, under these circumstances, is not proof against, but their existence is strong evidence for, the supposition that the tumor is pancreatic. Glycosuria has been observed in several cases of cancer of the pancreas.

Enlargement of the head of the pancreas may give rise to serious results from pressure upon adjacent parts. The duct leading from the pancreas is sometimes compressed, and dilatation of its branches within the organs ensues. Pressure upon the ductus communis choledochus, involving obstruction to the flow of bile, is followed by the retention of this secretion, causing persistent jaundice, dilatation of the bile-ducts, and at length disorganization of the liver. An enlarged pancreas may cause obstruction of the pylorus or duodenum, and this renders it difficult to differentiate the affection from cancer of the pylorus. Pressure upon the portal vein may exist to such an extent as to give rise to hydro-peritoneum.

Disease of the pancreas, particularly atrophy, has been found associated with glycosuria sufficiently often to render not improbable a pathological connection with the latter disease in some although not in all cases.

An abnormal deposit of fat occurs in the pancreas in two forms: first, as *fatty degeneration* of the glandular cells of the pancreas; and second, as *lipomatosis*, or development of adipose tissue between the acini, which may be thereby obliterated. We know no symptoms referable to lipomatosis of the pancreas.

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[1] *Pathological Anatomy.*

[2] For an analysis of 37 cases by DaCosta, vide *Proceedings of the Pathological Society of Philadelphia*, 1858.

Zenker[1] and Klebs have described cases of sudden death in which the only important lesion found was hemorrhage within and around the pancreas, combined with fatty degeneration. Similar cases have been reported under the name of acute hemorrhagic pancreatitis, in which the interstitial tissue of the gland was infiltrated with lymphoid cells. Necrosis of adipose tissue in and about the pancreas has also been observed in several cases. Sudden death has also been known to attend carcinoma of the pancreas.[2] The symptoms in some of these cases have been severe epigastric or abdominal pain, sometimes icterus, vomiting, subnormal temperature, and collapse. The explanation offered by Zenker for the sudden death is the occurrence of a reflex paralysis of the heart from a coincident affection, by pressure or otherwise, of the cœliac plexus and semilunar ganglion.

Finally, *calculi* sometimes form within the pancreatic ducts, varying in size from that of a pea to an almond. These have been found in considerable number. In their journey along the excretory passage to the duodenum they may occasion severe pain resembling that caused by biliary calculi. Paroxysms of pain due to the passage of pancreatic calculi or attacks of pancreatic colic are doubtless sometimes attributed to gall-stones, and it is not practicable, with our present knowledge, to make the differential diagnosis. The differential diagnosis is not, practically, very important, since the indications for treatment are the same in either case. Clinical observation with reference to the presence of free fat in the dejections may show this symptom to be present in cases in which, without special attention, it is overlooked; and it may be found that this symptom is of much value in the diagnosis of affections of the pancreas.[3]

A cystic tumor of the pancreas may attain to a large size. Dr. N. Senn has reported a case in which occlusion of the common duct led to the formation of a tumor containing three quarts of liquid. Laparotomy and the removal of the contents of the cyst were followed by complete recovery.[4]

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[1] *Tageblatt d. 47ten Versammlung Deutscher Naturforscher.*

[2] Haber, *Deutsches Archiv f. klin. Med.*, Bd. 15, p. 455.

[3] For a full consideration of affections of the pancreas, the article by Friedreich, in *Ziemssen's Cyclopædia*, Amer. ed., vol. iii., and the article by Humbert Molière, in the *Nouveau Dictionnaire de Médecine et de Chirurgie* (1873), may be referred to; also, Chvostek, "Klinische Beiträge zu den Krankheiten des Pancreas," *Wiener med. Blätter*, 1879, No. 33 *et seq.* Vide, also, an elaborate article on "Calculus and Other Affections of the Pancreatic Ducts," by George Woodruff Johnson, M. D., in the *Am. Journ. of Med. Sciences*, Oct., 1883.

[4] Vide article in the *Journal of the Am. Med. Association*, Oct. 3, 1885. Six additional cases, being all to be found in medical literature, are reported in this article.

SECTION FIFTH.

DISEASES AFFECTING THE NERVOUS SYSTEM.

CHAPTER I.

DISEASES RELATING TO THE CEREBRAL AND THE SPINAL CIRCULATION.

Cerebral Hyperæmia.—Cerebral Anæmia.—Cerebral Embolism and Thrombosis.—Cerebral Hemorrhage.—Localization of Cerebral Diseases.—Meningeal Hemorrhage.—Insolation, or Sunstroke.—Hyperæmia and Anæmia of the Spinal Cord and its Meninges.—Spinal Hemorrhage.

OF the diseases affecting the nervous system, the greater number relate to the nervous centres—the brain and spinal cord. Important affections, however, are seated in the nervous trunks or their branches; that is, in the peripheral portion of the cerebro-spinal system. I shall consider first those diseases of the brain and the spinal cord, together with their meninges, which, as regards pathological character, are common to all the anatomical systems—namely, hyperæmia, hemorrhage, embolism and thrombosis, inflammation, and various structural lesions—taking up afterward the diseases peculiar to the nervous system, or the *neuroses*. This chapter will embrace hyperæmia, anæmia, embolism and thrombosis, hemorrhage, and insolation, or sunstroke, these diseases forming a group in which are involved morbid conditions pertaining especially to the circulation within the skull and the spinal canal.

Cerebral Hyperæmia.

From the assumption that the contents of the cranium are incompressible by any amount of force which the heart can exert through the arteries, and from the fact that they are enclosed in an air-tight case, Monroe, in the last century, drew the conclusion that the quantity of blood within the skull can never vary. The incorrectness of this conclusion, however, has been abundantly established, particularly by Magendie on rational grounds, and by the direct observations of Donders. Magendie demonstrated that the cerebro-spinal fluid is an important regulator of the cerebral circulation, inasmuch as it recedes from the cranial cavity when the blood-vessels become distended, and is increased when the quantity of blood in the intracranial vessels is diminished. It has since been shown that a similar and an equally important rôle is played by the lymph in the perivascular lymph-spaces. Donders adopted the ingenious device of observing in animals the degree of fulness of intracranial vessels through a glass plate accurately adjusted and hermetically sealed, in an opening made through the skull by trephining. He and afterward other observers were able to prove conclusively by this method that the amount of blood in the intracranial vessels under various influences increases and diminishes. While the possibility of cerebral hyperæmia is now universally admitted, there is not the same unanimity as to its frequency, importance, and etiology. Within the last few years there has been a decided tendency, especially among neurologists, to deny the clinical importance of this affection; but while it is to be admitted that in former times the diagnosis of cerebral congestion was made too frequently and upon insufficient evidence, and while our knowledge of its pathogeny and symptomatology is still imperfect, there remains sufficient evidence of its occurrence and importance to justify its careful consideration.

The morbid appearances of congestion of the brain are to be sought chiefly in the brain-substance, and not in the meninges. The mistake is too often made of deciding upon the existence of cerebral congestion merely from fulness of the meningeal veins. Distension of these vessels with blood is no index of cerebral hyperæmia during life, being dependent upon the position of the body after death, the length of time during which the blood remains fluid, the manner of dying, and whether the head or the trunk be opened first at the post-mortem examination. It is probable that transient hyperæmia of the brain, as of other organs, may leave no trace of its existence after death. When, however, the congestion is intense and has lasted some time, there are found flattening of the convolutions from turgescence of the brain, a rosy color mixed with the gray of the cortical substance, and red points (*puncta vasculosa*) disseminated over the cut surface of the white matter. The vessels of the meninges, especially the smaller vessels, are also injected. The perivascular spaces are diminished in size or they disappear. Cerebral hyperæmia, especially of the passive or venous variety, may be followed by transudation of serum into the meshes of the pia mater, the choroid plexuses, and the ventricles. The presence of a large amount of reddish-brown amorphous pigment in the walls of the vessels and in the perivascular spaces has been held as an evidence of long-standing congestion. The presence of a certain amount of this pigment is physiological rather than pathological.

Congestion of the brain, as of other parts, may be active or passive. In active congestion an increased amount of blood is brought to the cerebrum by the arteries; in passive congestion the blood accumulates in the brain in consequence of some obstacle to its return by the veins.

It is thought that a full or plethoric habit predisposes to active determination of blood to the brain. Increased force of the heart's action, especially when resulting from simple hypertrophy of the left ventricle, may cause active cerebral congestion. Hypertrophy of the left ventricle is, in the great majority of cases, developed in compensation of some obstruction to the circulation, and has no tendency to produce cerebral congestion; but in the hypertrophy accompanying certain forms of chronic Bright's disease, and in the rare cases of so-called idiopathic cardiac hypertrophy, active cerebral congestion is sometimes developed.

Paralysis of the vaso-motor nerves supplying the cerebral arteries and irritation of vaso-dilator nerves are to be reckoned among the causes of active cerebral hyperæmia. It has been experimentally demonstrated that division of the sympathetic in the neck causes hyperæmia of the intracranial vessels.[1] It may be suspected that strong mental emotion may cause hyperæmia by an affection of the vaso-motor nerves. Nitrite of amyl produces cerebral congestion in this way. There are reasons for believing that one form of migraine or hemicrania is the result of unilateral vaso-motor paralysis causing cerebral congestion.[2]

Collateral fluxion of the cerebral vessels, resulting from some obstruction to the flow of blood from the aorta into other branches, has been considered as giving rise to cerebral hyperæmia. As causes of this collateral fluxion, have been regarded stenosis of the aorta at the point of entrance of the ductus Botalli, compression of the abdominal aorta and its branches by distended intestine, tumors and exudations, and obstruction to the cutaneous circulation by the action of cold. The adequateness of these causes, however, is doubtful. Excessive mental or emotional activity and alcoholic stimulation are efficient causes. Cerebral hyperæmia is an element, but probably not the most important one, in insolation.

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[1] Ackermann, *Virch. Arch.*, 1858, Bd. 15, p. 431.

[2] Möllendorff, *ibid.*, Bd. 41; Berger, *ibid.*, Bd. 59.

Rapidly-induced *active congestion* of great intensity may produce sudden coma which may disappear after a few hours or may prove speedily fatal. This is the pathological explanation of apoplectic attacks in the cases which have been distinguished as cases of *congestive apoplexy*. An instance of sudden death from active congestion incident to simple hypertrophy of the heart has fallen under my observation. When the encephalic functions are thus suspended, this effect is attributable to intracranial pressure. The coma is produced by the pressure of the blood within the vessels, as it is by the extra-vascular pressure in cases of meningeal hemorrhage. The pressure of the blood in the large vessels interrupts the circulation in the brain-substance; and hence the suspension of the cerebral functions. It is apparently a paradox that an over-accumulation of blood within the skull should cause coma, and perhaps destroy life by depriving the mass of the brain of a due supply of arterial blood, yet the fact is sufficiently intelligible. Apoplexy attributable to active hyperæmia is a rare event, but of its occurrence there can be no doubt.

The SYMPTOMS of active hyperæmia of more or less intensity, but falling short of a degree sufficient to produce coma, are as follows: Pain in the head varying in degree, sometimes intense, with a sense of heat, fulness, bursting, or weight. The pain is not limited to one side or to any particular part of the head. There is inability to sleep, or if sleep be obtained it is incomplete and unrefreshing. Delirium sometimes occurs, and if present it is active. This symptom occurs especially when the congestion is due to excessive alcoholic stimulation. Convulsions occasionally occur in the adult. They are less infrequent in infancy, but active congestion as a primary affection is not frequent in infantile life. The countenance is usually but not invariably flushed, the eyes are suffused or injected, with intolerance of light and sometimes scintillations. The head is hot; the carotids and the temporal arteries pulsate strongly; and the patient is conscious of a throbbing sensation in the head. Inability to control mental operations, mental confusion, and momentary loss of consciousness are occasional symptoms, and also vertigo, tinnitus aurium, and some embarrassment in speech. The symptoms are more or less marked in proportion as the hyperæmia is greater or less in degree.

Simple hyperæmia is merely a functional affection. It involves no lesion; and if the congestion be removed without much delay the structures are left intact. In a slight or moderate degree it involves no immediate danger. As already stated, however, it may be developed rapidly in a sufficient degree to produce apoplectic coma, which sometimes proves speedily fatal. Congestion may prove an exciting cause of cerebral hemorrhage if the minute arteries of the brain be the seat of miliary aneurisms or other lesions. Without morbid changes in the coats of the vessels cerebral hyperæmia, however intense, is very rarely causative of hemorrhage.

The DIAGNOSIS of congestive apoplexy involves the discrimination from sudden coma caused by cerebral or meningeal hemorrhage, cerebral embolism, alcoholic intoxication, and uræmia. The differential points cannot be stated without anticipating the clinical histories of these affections. Suffice it to say here that apoplexy from cerebral hemorrhage and embolism is excluded by the absence of hemiplegic paralysis, and that the symptoms pointing to congestive apoplexy are those which denote a determination of blood to the head, such as a flushed countenance, strong pulsation of the carotids, etc. Circumstances causative of active hyperæmia are also to be considered, such as simple hypertrophy of the left ventricle, alcoholic excess, mental excitement, etc.

Intense active hyperæmia calls for bloodletting. Relief is obtained most promptly by this measure. If the symptoms do not denote a degree of congestion sufficient to call for bloodletting, relief may be obtained by the cold douche, ice to the head, stimulating hot pediluvia, dry cups applied to the neck, and an active purgative. The bromide of potassium and the other bromides are potential remedies, the rationale of their operation being that they diminish the amount of blood within the cranium. They may be employed when the degree of congestion does not indicate either bloodletting or active purgation. Faradization of the cervical sympathetic nerve and ergot relieve active congestion by inducing contraction of the intracranial arteries. Antimony relieves by its sedative effect upon the heart, but it should not be given in doses which produce vomiting. Sinapisms and other excitants of the skin relieve by revulsion.

These measures are for the removal or diminution of existing congestion. The treatment, in addition, should embrace, if possible, the removal of the causes, and if the patient be of a full habit, regulation of diet and occasional saline laxatives are advisable.

Passive congestion differs from the form just considered. Intracranial pressure from over-distended vessels exists alike in both, but in active congestion there is an overplus of arterial blood, and in passive congestion there is an excess of venous blood. This difference accounts for disparity in the symptoms.

In a degree to constitute a morbid condition passive congestion may be produced by any mechanical impediment to the return of blood from the head. This may be due to pressure on the jugular veins in cases of goitre, or of enlargement of the lymphatic glands of the neck, and by the pressure of an aortic aneurism or other tumors on the vena cava. Prolonged efforts of expiration, as in some forms of spasmodic cough and in playing on wind instruments, have this effect. Diseases of the lungs occasioning interference with the pulmonary circulation, such as pleuritis with large effusion and emphysema, give rise to passive congestion by leading to an overloading of the right cavities of the heart. It is incident to certain diseases of the heart, especially to obstruction at the right side of the heart from valvular lesions on that side, but more frequently from dilatation of the right ventricle and auricle, together with tricuspid insufficiency as a result of mitral lesions. Obstruction or obliteration of the cerebral sinuses by coagula induces passive congestion in the communicating veins. Diminished force of the arterial circulation—that is, weakness of the *vis a tergo*—is also a cause.

The SYMPTOMS denoting passive congestion are somnolency, dulness of mind, blunted perceptions, and sometimes in children convulsions. Throbbing of the carotids and temporal arteries, active delirium, increased heat, and other symptoms denoting active congestion are wanting. The veins of the head are turgid, and the face not infrequently is cyanosed. The symptoms are due in part to intracranial pressure, and in part to the excess of venous or the deficiency of arterial blood.

Passive congestion of the brain is to be considered as not an individual affection, but a morbid element entering into various affections. When dependent on a mechanical impediment to the return of blood from the head, caused by cardiac lesions, tumor of the neck, etc., the object of treatment is, if possible, to diminish the obstruction. It can do no good to diminish the mass of blood by bloodletting. It is doubtful if aught is to be effected by revulsive measures. Anything which weakens the force of the circulation tends to increase the congestion. If dependent on feebleness of the circulation, the therapeutical indications are to remove, if possible, the causes of exhaustion, and to increase the force of the circulation by digitalis and other cardiac tonics, stimulants, and nutritious diet. The degree of toleration of great venous congestion in some instances—for example, in dilatation of the right side of the heart or when an aneurismal tumor presses on the descending vena cava—is worthy of note, as showing that symptoms are probably often incorrectly attributed to this cerebral condition.

In the foregoing account of cerebral hyperæmia reference has been had to this condition as general; that is, affecting the entire encephalon, and not limited to certain portions of the brain. Hyperæmia more or less circumscribed occurs in connection with tumors which may produce obstruction of vessels or cause a determination of blood within a limited area. Of these two modes, the former gives rise to passive, the latter to active congestion. Passive congestion, limited more or less in extent, is a result of thrombosis of veins and also of both thrombosis and embolism of arteries.

Cerebral Anæmia.

In cerebral anæmia the brain is pale. The puncta vasculosa in the white substance are few or absent. The contrast in color between the gray and white substance is less marked than normal. The perivascular spaces are dilated. Cerebral anæmia is often accompanied by increase in the amount of cerebrospinal fluid in the subarachnoid space and by serous transudation into the ventricles. This œdematous condition of the brain is often found in persons who have died of wasting diseases and who may not have presented any cerebral symptoms during life. A wet brain, as it is called, is often found as the chief pathological lesion in individuals who have died of acute or chronic alcoholism, and who may have been comatose during the last hours of life.

The quantity of blood sent to the brain is diminished whenever the mass of blood in the body undergoes diminution, as after hemorrhages, and in all diseases in which the constituents of the blood are expended without a proportionate renewal by means of alimentation. An over-accumulation of blood in other organs than the brain has the same effect. Mitral obstruction or regurgitation also occasions a defective supply of blood to the brain. An increase of the subarachnoidian liquid and œdema of the brain, which may be effects of venous congestion, must diminish the amount of arterial blood by pressure on the cerebral arteries. An obstacle to the passage of blood in the distributing arteries may arise from their spasmodic contraction excited by influences exerted through the vaso-motor nerves; and certain mental emotions which are accompanied by notable pallor of the face and a tendency to syncope produce this effect. Anæmia from vaso-motor spasm exists in the first stage of an epileptic paroxysm.

A deficiency of arterial blood in either the whole or a part of the brain is an important pathological element in certain affections of the nervous system which are to be presently considered, the deficiency arising from plugging of a cerebral artery (embolism and thrombosis), the pressure of a clot, of the products of inflammation (fibrin, serum, and pus), and of tumors of different kinds. Inasmuch as the red globules are the chief constituents of the blood on which depends the normal condition of the nervous centres, a paucity of these as regards morbid effects is equivalent to a diminished supply of arterial blood to the brain. Cerebral anæmia, therefore, is incident to impoverishment of the blood. Thus produced, it is an important element in many affections. Cephalalgia, mental depression, hysteria, and the phenomena referable to nervous asthenia are often attributable to a deficient supply of fully oxygenated blood to the brain in consequence of its impoverishment, or, in other words, a morbid deficiency of the carriers of oxygen—namely, the red globules. Impoverishment of the blood, together with feebleness of the circulation, resulting from intestinal transudation, occasions in children that condition of semi-coma described by Marshall Hall under the name "hydrencephaloid affection"—a condition involving, with deficiency of arterial blood or of red globules, venous congestion.

The immediate effect of a sudden and considerable diminution of arterial blood in the brain is syncope. Consciousness is lost, the face presents a death-like pallor, and convulsions may occur. Syncope occurs in cases of abundant hemorrhage or after a copious venesection, and it may be produced by feebleness of the heart's action without any loss of blood. The phenomena of syncope are due to a certain deficiency of arterial blood in the brain. It is especially liable to occur when the mass of blood in the body is deficient; and impoverishment of the blood also predisposes to it, inasmuch as the efficiency of blood in maintaining the functions of the nervous system depends chiefly on a normal quantity of the red globules. Epileptiform convulsions were produced by Kussmaul and Tenner in rabbits by tying the arteries which supply the brain. Convulsions are not infrequently symptomatic of the anæmia induced by severe post-partum hemorrhage. Coma and paralysis in cases of cerebral hemorrhage, embolism, etc. are attributable to an interruption of the free circulation of arterial blood in the substance of the brain. Notable diminution of the arterial blood sent to the brain may be tolerated in a remarkable degree. This fact was exemplified in a case which came under my observation in which were absence of arterial pulsation in both upper extremities and an extremely feeble pulsation in both carotids, the autopsy showing complete embolic occlusion of the innominate artery, with complete occlusion of the left subclavian artery, and the left carotid artery so nearly occluded as to admit of the passage only of a fine probe. There were aortic lesions and the heart was found enlarged. The patient suffered from frequent attacks of vertigo and blindness, and was incapable of taking any active exercise. Pressure on the carotids produced syncope. There were no other noteworthy cerebral symptoms, and death was due to an attack of pneumonia.

The TREATMENT of syncope embraces measures to increase the quantity of oxygenated blood sent to the head. Recumbency, with the head low to secure the effect of gravitation, is important. Internal stimulants to increase the force of the heart's action are indicated. Dashing cold water upon the face is an efficient measure which acts by causing deep inspiratory efforts. The application of the vapor of ammonia to the nostrils has the same effect. These measures generally are promptly successful, unless the syncope be caused by an excessive hemorrhage or the loss of blood continue. If not successful, another efficient measure consists in compressing the abdominal aorta and the arteries of the upper limbs. In this way, advantage is taken of collateral fluxion.

Cerebral anæmia, as incident to impoverishment of the blood, calls for the remedies and hygienic measures which have been already considered in treating of anæmia as one of the diseases of the hæmatopoietic system (p. 369). The importance of recognizing this condition, with reference not only to a correct interpretation of pathological phenomena pertaining to the brain, but to disorders in other organs, and to its morbid agency when it is an element of nervous affections, cannot be too strongly enjoined.

It is to be noted that a deficiency of arterial or oxygenated blood may coexist with venous congestion within the skull. The former may lead to the latter in consequence of the contents of the skull being free from atmospherical pressure; and on the other hand congestion of the cerebral veins due to some impediment to the return of blood from the head may be an impediment to freedom of circulation in the distributing arteries, and thus occasion a deficiency of arterial blood.

Cerebral Embolism and Thrombosis.

The fact has been pointed out in Part I. of this work (p. 30) that the presence of an embolus or a thrombus in an artery, the branches of which freely anastomose with each other—as, for example, in an artery of a voluntary muscle—produces, as a rule, no appreciable disturbance of the circulation; while the occlusion by embolus or thrombus of an artery belonging to the terminal arterial system—that is, of one whose branches do not inosculate—is followed by an infarction, either hemorrhagic or simply necrotic, in the district supplied by the occluded vessel. It has been demonstrated by Heubner and by Duret that the arteries supplying the so-called basal district of the brain—that is, the region occupied chiefly by the nucleus caudatus, internal capsule, nucleus lenticularis, optic thalamus, and adjoining cerebral substance—are terminal arteries, and that those supplying the cortex cerebri freely anastomose with each other in the pia matter, in which they become much reduced in size before they enter the gray matter. We are thus able to explain why an embolus lodging in one of the cortical arteries in the pia mater (whither, however, it rarely finds its way) produces, as a rule, little permanent disturbance, while an embolus or thrombus occluding one of the basal arteries beyond the circle of Willis is followed by destructive changes in the region from which the blood-supply has been cut off.

The source of the emboli conveyed into the cerebral arteries in the great majority of cases is the left ventricle of the heart. They consist usually of masses of fibrin which have been deposited on the aortic or mitral valves in consequence of acute or chronic endocarditis, and which are washed away by the blood-current into the circulation. Thrombi formed in the heart from any other cause may also give origin to emboli. Cerebral emboli may also be derived from the pulmonary veins, the first part of the aorta, the carotid and the vertebral arteries. Next to disease of the left ventricle, aortic aneurism and atheroma of the aorta form the most frequent source of emboli. The emboli usually consist of portions of a thrombus, but they may be calcareous concretions, fragments of connective tissue, bits of new growth, bacteria, other vegetable parasites, fat, etc. (Vide p. 30.)

An embolus may lodge in any artery of the brain, but in the majority of cases it occludes the middle cerebral artery not far from its origin. Next in order of frequency comes embolism of the internal carotid, and then embolism of the basilar artery. The statistics compiled by Gelpke[1] showed that out of 100 cerebral emboli more than 90 affected the arteries which supply the basal ganglia. An embolus enters the left middle cerebral artery more frequently than the right. The explanation of this is afforded by the difference in angle at which the left common carotid and the innominate come off from the aorta; but the predominance in frequency of embolism of the left cerebral arteries over that of the right has been exaggerated. According to

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[1] *Arch. d. Heilkunde*, 1875, p. 501.

Gelpke, of 131 emboli of the carotid artery or of its branches, the left side was affected 64 times, the right side 54 times, and both sides 13 times.

An embolus usually leads to the formation of a thrombus about it. The cause of spontaneous thrombosis of the cerebral arteries is to be sought in some change in the walls of the affected vessels. The most frequent change is atheroma of the arteries of the brain, but other forms of chronic arteritis, especially syphilitic endarteritis, may also occasion thrombosis. Other and rarer causes are—simple fatty and calcareous degenerations of the arteries, compression of the arteries as by a tumor, extension of a thrombus primarily formed in one of the carotid arteries, and possibly mere slowing of the circulation from weakened heart's action.

Cerebral thrombosis occurs chiefly in old age, because the arterial changes to which it usually owes its origin are most frequent at this period of life. When it is present in the earlier periods of life, it is due usually, but not necessarily, to chronic alcoholism or to syphilis. Embolism, on the other hand, may occur at any age.

The effect of the occlusion by an embolus or a thrombus of an artery of the brain belonging to the terminal system is softening of the portion of brain the blood-supply of which has been cut off. For reasons which have already been given, embolic softening in the vast majority of cases is situated in the caudate nucleus, the internal capsule, the lenticular nucleus, the optic thalamus, or the island of Reil. According to the size of the artery occluded, a greater or less portion of this region of the brain will be affected. It is especially to involvement of the internal capsule in the softening that we attribute the hemiplegia which attends embolism of the middle cerebral artery; for in this narrow band of white matter, situated between the lenticular nucleus externally and the caudate nucleus and optic thalamus internally, are compacted together the motor-fibres which supply the opposite half of the body. Embolism of a cortical artery of the brain may do no permanent damage, or it may be followed by softening of the district supplied by the artery. Many of the depressed, soft yellowish patches (*plaques jaunes*) found in the cerebral cortex are of embolic or of thrombic origin.

The first effect of embolism of a cerebral artery is anæmia of the district supplied by that vessel. If a sufficient collateral circulation be established, this anæmia passes off without permanent damage; but if the branches of the artery do not anastomose, the anæmia is followed by a gradually progressive necrobiosis or death of the part, which may or may not be accompanied by hemorrhage. A true hemorrhagic infarction, like that which follows embolism of one of the branches of the pulmonary artery or of the splenic artery, does not occur in the brain, but a certain amount of extravasation of blood in a punctate form is not infrequently present in the anæmic district. It is not quite clear why in some cases embolic softening is accompanied by so much blood-extravasation as to resemble a primary cerebral hemorrhage, while in other cases no blood is extravasated. It is probable that increase of pressure in the veins coming from the anæmic spot favors a regurgitant current with hemorrhagic softening, and that low venous pressure opposes it. When a spot of embolic softening is stained with extravasated blood, the process is called red softening; if there be simply anæmic necrosis without hemorrhage, it is spoken of as yellow softening by some and white softening by other writers. It may be mentioned here that the term cerebral softening has ceased to have any exact significance as a pathological process, and that each of the varieties—red, yellow, and white softening—may occur from various causes and indicate various pathological conditions.

In the early stages of embolic softening (and what is said in this connection of embolic applies equally to thrombic softening) the volume of the affected portion of brain is somewhat increased; its consistence is diminished; its boundaries are not distinctly marked off from the surrounding healthy tissue; and its color is red or yellow or whitish-yellow. At a later period the softened spot is diminished in volume and its consistence may be nearly fluid. The final stage of the process is not always the same. Frequently a cyst with fluid contents remains. If the first stage have been red softening, the cyst-wall and the fluid contents are stained with blood-pigment, and the cyst cannot be distinguished from one following a primary cerebral hemorrhage. If hemorrhage have not attended the process of softening, the cyst-wall and its contents are unstained and the embolic origin of the cyst is evident. Sometimes, instead of a cyst there is left a reticulum of connective-tissue fibres and blood-vessels enclosing in their meshes fluid or semifluid contents, frequently of a white color. This is called the cellular infiltration of Durand-Fardel. Other terminations are in a cicatrix and in a dry caseous mass. Finally, the affected tissue may remain indefinitely as a spot of yellow or white softening. In the sequence of changes a spot of primary red softening becomes one of yellow softening in consequence of pigmentary alterations in the extravasated blood.

The microscopic examination reveals, as the most characteristic element throughout the stages of softening, the so-called granular corpuscles, formerly called the inflammatory corpuscles of Glüge. These are large, usually spherical cells, so densely filled with fat-globules as to appear dark by transmitted and white by reflected light. They are usually present in large numbers. They probably have different origins. They consist of emigrant white blood-corpuscles which have taken up the fatty matter resulting from the disintegration of nerve-elements, or they may represent neuroglia-cells, ganglion-cells, adventitious cells of the blood-vessels, or endothelial cells distended with fatty globules. Similar granular cells are found in pathological processes accompanied with fatty degeneration in various other parts of the body, as in ovarian cysts, pneumonia, etc. In the early stages the disintegrated nerve-elements can be recognized in the shape of broken nerve-fibres, drops of myelin, and fatty ganglion-cells. These gradually disappear. Corpora amylacea and red blood-corpuscles, and later amorphous and crystalline hæmatoidin and crystals of the fatty acids, may be detected.

Certain secondary degenerations in the spinal cord which may follow softening of the internal capsule and some other parts will be noticed under the head of Cerebral Hemorrhage.

The preceding description of the anatomical changes resulting from cerebral embolism has been based on the assumption that the embolus is bland in its nature, which is usually true. If, however, the embolus be infectious, it leads to the formation of an abscess here as in other situations; or if an embolus come from a gangrenous focus, such as may be present in the lung, gangrene of the affected portion of the brain or necrosis accompanied by decomposition follows.

CLINICAL HISTORY.—Cerebral embolism is one of the several pathological conditions which are causative of sudden coma or apoplexy. This effect does not occur as a rule, but it is not infrequent. The apoplectic attack is not preceded by premonitions. If antecedent cerebral symptoms have existed, they had no connection with the occurrence of the embolism, the latter depending entirely on the dislodgment of the embolus at its source, and being wholly independent of any previous affection within the brain.

The apoplectic coma may be complete or the patient may be aroused to consciousness by more or less effort. There may be stertor, infrequency of the pulse, vomiting, and immobility or inequality of the pupils; but these symptoms are rare in embolic apoplexy as compared with apoplexy from cerebral hemorrhage. Convulsive movements occasionally occur.

Hemiplegic paralysis almost always is an effect of embolism of the middle cerebral artery, and this effect is coincident with the apoplexy if the latter occur.

The duration of the coma varies between a few moments and many hours. A fatal termination, the coma existing until death, is rare. The hemiplegia, with few exceptions, persists for a longer or shorter period after the coma has disappeared. It may be permanent. It may or may not include the muscles of the face. It affects the side opposite to the hemisphere in which the embolism is seated. It is chiefly or exclusively motor. Owing to the fact that the embolus is seated oftener in the left than in the right hemisphere, the hemiplegia in the majority of cases affects the right side.

It is not easy to give a satisfactory pathological explanation of embolic apoplexy. The localized anæmia, limited to the district supplied by the occluded artery, and the increased pressure by the arteries dilated in consequence of arterial fluxion, seem *a priori* inadequate to account for the sudden coma, and other causes are not apparent.

In a considerable, perhaps the larger, proportion of cases embolic hemiplegia is not preceded by an apoplectic attack. The hemiplegia occurs suddenly without premonitions. The paralytic, like the apoplectic, stroke takes place unexpectedly, the patient being free from any cerebral symptoms up to the instant of its occurrence.

Cerebral thrombosis very rarely causes apoplectic coma. Coma, if it occur, is developed gradually, and is due to an extension of the thrombus by fresh deposits, thus enlarging the district from which the arterial supply is cut off. Hemiplegia takes place, not by a sudden stroke, but by degrees, not infrequently becoming greater by successive steps instead of by a continuous increase. Moreover, the occurrence of coma or of hemiplegia is preceded by premonitions, such as vertigo, impairment of mental faculties, difficulty in speech, etc. These premonitions may in some instances be due to embolism, which causes a partial obstruction, the occlusion being rendered complete by a thrombus added to the embolus. The so-called softening of the brain of old people, which is accompanied by increasing dementia, is usually the result of thrombosis.

Disturbances of speech often occur in cases of cerebral embolism and thrombosis. Loss of speech, not from any affection of the organs concerned in phonation or from mental imbecility, is expressed by the term *aphasia*. The terms *aphemia* and *alalia* have the same significance, but are not generally used. Aphasia is to be distinguished from *aphonia*, a term denoting inability to speak in consequence of either laryngeal disease or paralysis of the laryngeal muscles. Aphonia is the loss of voice, and aphasia is the loss of speech; the former relating exclusively to the vocal organs, the latter proceeding from a cerebral affection. In aphasia the patient is mute from an inability to use words expressive of the ideas which are in the mind. The loss of speech may be complete or partial, and the patient in some instances is absolutely dumb. Sometimes a single word can be spoken, such as "yes" or "no," and this is uttered whenever the attempt to speak is made; in other instances the vocabulary is larger, but limited to a few words; and in different cases there is every degree of incompleteness in the aphasic condition. A difficulty of speech may consist in an inability to use the proper words to express the mental ideas. Things are not called by their right names, and the language in sentences either spoken or written is incongruous and unintelligible. This difficulty is sometimes distinguished as *paraphasia*.

An important distinction is denoted by the terms *amnesic* and *ataxic* *aphasia*. In ataxic aphasia the mind recognizes the proper signification of words, and the difficulty consists in an inability to give utterance to the words. The name ataxic implies that this inability proceeds from a lack of power to co-ordinate the muscles concerned in speech. Something more than simple ataxia, however, seems to be involved. A lack of co-ordinating power would be manifested by movements responding to, but not governed by, the will, and the result would be inarticulate phonetic sounds. This evidence of ataxia is sometimes manifested, but generally the patient will not make any effort to speak—that is, if the aphasia be complete—and if a few words be uttered, these may be spoken with distinctness. In amnesic aphasia there is inability to recollect words as the symbols of ideas. Language is lost to the memory.

If the aphasia be purely ataxic, the ability to use language correctly in writing is not lost, and may not be impaired, provided the intellectual faculties be intact. The ability to read is retained, and language spoken by others is comprehended. So far as can be judged, in some cases of ataxic aphasia absolute mutism is not incompatible with undiminished intelligence. Frequently, however, the cerebral affection which causes the aphasia impairs, in a greater or less degree, the mental faculties. In amnesic aphasia, on the other hand, the loss of speech is accompanied by inability to use written language (agraphia). The loss of the memory of words does not necessarily carry with it inability to understand their meaning when they are heard or seen. The patient may understand both spoken and written language. Inability in these respects involves, in a greater or less degree, mental imbecility.

In ataxic aphasia the ability to interpret signs or gestures is preserved. If this be lost in either ataxic or amnesic aphasia, there is imbecility in a greater or less degree. Patients affected with ataxic aphasia, even when it is evident that the intelligence is well retained, cannot repeat words after dictation. Patients with purely amnesic aphasia are able to do this, provided the intelligence is not too much impaired. The inability to repeat words is evidence either that the aphasia is not purely amnesic or that there is imbecility.

The ataxic and amnesic forms of aphasia may be combined. The patient can then express ideas neither by spoken nor written language, and is unable to repeat words after dictation. In a form of aphasia which has been called *word-blindness* the ability to read and write is lost, although speech is not affected. I have met with two instances in which the aphasia was exclusively of this character, recovery taking place in each instance. Hemiplegia was wanting in both these instances.

In cases of either ataxic or amnesic aphasia it is not an easy problem to determine whether or not, or to what extent, the mental powers are impaired. This problem is sometimes presented to physicians in medico-legal cases. The instances must be rare in which persistent aphasia, associated with hemiplegia, does not involve more or less impairment of mind. Ataxic aphasia as an isolated affection may exist with the intellect apparently unaffected. It is doubtful if this can be said of amnesic aphasia. Words are the instruments of thought as well as the expression of ideas; and it is difficult to believe that the inability to recollect words is possible without deterioration of the reasoning faculties. The inability to understand spoken or written language and signs is proof of impaired intellect.

A large accumulation of clinical observations within the last few years has established the connection of aphasia, in the vast majority of cases, with lesions localized in a particular part of the brain—namely, the island of Reil, and especially the posterior part of the third frontal convolution. These lesions are not alone those incident to embolism and thrombosis, but those caused by cerebral hemorrhage, morbid growths, abscess of the brain, etc. Aphasia is therefore a symptom belonging also to the clinical history of diseases which are to be hereafter considered. In a small number of cases lesions have been found in the similar parts of the right hemisphere, or the aphasia has been associated with left-sided hemiplegia. In a certain proportion of the latter cases it was ascertained that the patients were left-handed. It may be doubted if in any case of well-marked persistent aphasia an autopsy has shown conclusively absence of any lesion affecting the island of Reil or proximate parts in one or the other hemisphere. (For statistical facts on which these statements are based the reader is referred to other works.[1])

The theory which, with our present knowledge, furnishes the most satisfactory explanation of these facts assumes the following postulates: The third convolution and the island of Reil are in a direct physiological relation to language. This is true of the left, and not of the right, hemisphere in most persons, owing to the habitual use of the left instead of the right hemisphere in the exercise of the function of language. As exceptions to the rule some persons habitually use the right instead of the left hemisphere in the exercise of the function of language, and these persons are generally left-handed. Injury of the parts in the left hemisphere which are in relation to language do not in these persons cause aphasia, but this result follows injury of the parts in the right hemisphere.[2] The parts in the right hemisphere which are in a physiological relation to language, although their functional activity has been in abeyance owing to non-use, may assume in some instances activity after the parts of the left hemisphere have been incapacitated by lesions; hence, improvement or recovery from aphasia may sometimes take place, the lesions in the left hemisphere remaining unchanged.

Aphasia attributable to embolism occurs without apoplexy or paralysis. The following case is an example: A young man having ridden all day in an open wagon, the weather being cold, after eating a hearty supper was suddenly unable to utter a word. He went to bed, hoping that the ability to speak would return in the morning. He slept perfectly well, but on awakening he was still unable to speak. In the course of the day he began to use some words, often using wrong words, but in the course of a few days he recovered his speech, although ever since he has had some difficulty in selecting right words. He saw me a year after this occurrence. Meanwhile, nothing had occurred to denote any cerebral disease, except that he was conscious of an uneasy sensation in the left side of the head. He had an aortic regurgitant and a mitral direct murmur, with considerable enlargement of the heart. It can hardly be doubted that in this case the temporary aphasia was due to embolism of one of the branches of the middle cerebral artery.

Cases have been reported of the occlusion, after an interval, of the middle cerebral artery on the two sides, giving rise to double hemiplegia. These cases have furnished examples of left hemiplegia without aphasia, followed by right hemiplegia with aphasia.

DIAGNOSIS.—The diagnosis in most cases of embolic apoplexy requires the exclusion of apoplexy from cerebral hemorrhage. The latter is presently to be considered. If the apoplexy be due to occlusion of the middle cerebral artery, it is accompanied by hemiplegia. This is true also of apoplexy due to cerebral hemorrhage in the situations where it is most likely to take place. As a rule, the other pathological conditions which stand in a causative relation to apoplectic coma do not give rise to hemiplegia. The differentiation, therefore, in the great majority of cases is between embolism and hemorrhage in cases of apoplexy with hemiplegia. The following are the differential points:

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[1] Vide the very comprehensive article on Aphasia, by Kussmaul, in *Ziemssen's Cyclopædia*, vol. xiv.

[2] Vide cases reported in *Trans. London Clinical Society*, vol. xiv.

Embolism may occur at any period of life, and it is as frequent in early as in middle or advanced age. Cerebral hemorrhage is rare before the middle period of life. An apoplectic seizure, the patient not having reached the latter period, is therefore more likely to be due to embolism than to hemorrhage. The occurrence of hemiplegia on the right side weighs somewhat in favor of embolism. The existence of endocarditis or of valvular lesions in the left side of the heart renders embolism probable. The absence of symptoms denoting cerebral congestion which often, but by no means uniformly, precede cerebral hemorrhage, has some weight in the diagnosis. The absence of cerebral prodromes, which applies to all cases of embolism, is not as constantly applicable to cases of hemorrhage. Vomiting, an infrequent pulse, stertor, changes in the pupils, and convulsions are more frequent and more marked in cases of apoplexy due to hemorrhage. The diagnosis of embolism is strengthened by the occurrence of symptoms denoting embolism of the spleen and kidneys. Unilateral amaurosis from embolism of the central artery of the retina, preceding or accompanying the attacks, is diagnostic of embolic apoplexy.

These differential points suffice often for a highly probable diagnosis. They do not warrant a positive diagnosis. The physician who ventures upon the latter incurs a risk of being convicted of an error at the autopsy. If, however, the patient speedily emerge from the apoplectic condition, rapid improvement as regards the paralysis subsequently taking place and ending in complete recovery, the diagnosis of embolism is very probable.

Embolic hemiplegia is to be differentiated from hemiplegia due to cerebral hemorrhage and to other conditions—namely, morbid growths, abscess, etc. The difficulty in the diagnosis, however, is chiefly in excluding hemorrhage. The other conditions generally involve antecedent symptoms of cerebral disease, whereas the paralysis caused by embolism is devoid of these; and this is often true of hemorrhage. Both embolism and hemorrhage may give rise to sudden hemiplegia without apoplexy, but in much the larger proportion of cases this is true of embolism. In sudden hemiplegia without apoplexy, therefore, the chances are that it is embolic. Other differential points are embraced in the list of those which relate to the differentiation of apoplexy from embolism and hemorrhage. Rapid improvement is much more likely to take place in paralysis from embolism, and complete recovery, which is not infrequent in embolic hemiplegia, is rare in paralysis from hemorrhage. Increase of the body-heat two or three days after an apoplectic or paralytic stroke has been regarded as denoting the presence of a cerebral clot, but observations have shown that an equal increase sometimes follows when the apoplexy and paralysis are due to embolism.

For the differentiation of embolism from thrombosis the most important point has been stated—namely, the effects of arterial occlusion occur by the former suddenly, and by the latter more or less gradually. This point of distinction, however, cannot always be relied upon. A thrombus may exceptionally form so rapidly as to simulate embolism in the abruptness of its effects. An attack of hemiplegia, either with or without apoplexy, may occur during sleep, and under these circumstances this differential point may not be available. Again, as already stated, an embolus may cause only a partial obstruction, complete occlusion afterward being produced by a thrombus formed at the seat of the embolus.

Other differential points are as follows: The conditions for thrombus belong to middle or advanced age. The youth of the patient is therefore a point in evidence for embolism. The existence of endocarditis or valvular lesions of the heart points to embolism, and their absence to thrombosis. Calcification of the radial arteries is a ground for inferring that the cerebral arteries have undergone the same change, this being the most frequent of the conditions which are causative of thrombus.

Embolism and thrombosis of the basilar artery may produce coma, either suddenly or more or less slowly, without hemiplegia. Death takes place in some cases within a few hours. In other cases patients recover, although the artery remains occluded. These statements have been verified by cases under my observation.

PROGNOSIS.—An apoplectic attack caused by embolism of the middle cerebral artery is less likely to prove immediately fatal than apoplexy from cerebral hemorrhage; hence the probable diagnosis has a bearing on the prognosis. In most cases, after a few hours the coma disappears, hemiplegia remaining.

The persistence of hemiplegia, whether preceded or not by apoplexy, depends on the occurrence of softening from necrosis and extravasation of blood as effects of the arterial obstruction. If the circulation within the district in which the blood-supply was furnished by the occluded artery be restored within two or three days, so as to prevent the changes incident to defective nutrition, rapid improvement takes place and is followed by complete recovery. These changes are to be inferred from the persistence of the paralysis. There may be more or less improvement, which will be in proportion to the favorable course of the local process as described in connection with the Anatomical Characters. Complete recovery from the paralysis is not to be expected. If the local process be not favorable, more or less impairment of the mental faculties is added to the persistent paralysis. The duration of life is indefinite. Death is generally caused by antecedent or intercurrent disease.

The prognosis if hemiplegia be caused by thrombosis is less favorable than if it be due to embolism, as the patients are more enfeebled. The prognosis is extremely unfavorable if coma supervene upon hemiplegia.

The prognosis as regards the continuance of aphasia is a matter of much interest to patients and their friends. The restoration of speech may accompany recovery from the paralysis, but it may be otherwise. In a hospital case under my observation the hemiplegia entirely disappeared, the general health was excellent, and the mental faculties were apparently not in the least affected, but complete mutism persisted. The patient made no attempt to speak; and when asked why he was silent he placed his finger on his lips and shook his head. He was discharged in this condition. If, owing to the pathological changes in the affected district of the brain, the paralysis persist, aphasia generally continues. There may, however, be more or less improvement. There are no means of judging beforehand what degree of improvement will take place. Adopting the views stated in regard to the duality of the parts which stand in a physiological relation to language, if the injury destroy the functional capacity in the hemisphere to which the exercise of the function has been limited (usually the left hemisphere), the recovery of speech or the degree of improvement will depend on the ability to develop functional activity of the corresponding parts in the other hemisphere, which had previously been in abeyance. The probability of success is greater in proportion to the youth of the patient. If the aphasia continue without improvement for several months, none is to be expected.

The prognosis is favorable when aphasia occurs without apoplexy, hemiplegia, or other evidence of cerebral disease.

TREATMENT.—The object of treatment in the early stage of embolism and thrombosis is the re-establishment of the circulation within the district deprived of blood-supply, in order to prevent the changes incident to defective nutrition. Bloodletting, cathartics, and any measures which weaken the heart's action contravene this object, and are therefore contraindicated. On the contrary, it is often important to increase the strength of the heart's action by tonic remedies, a nutritious alimentation, and alcoholics. If there be feebleness and irregularity of the heart's action in connection with valvular lesions and dilatation, digitalis is indicated. All forms of counter-irritation are contraindicated. Cold applications to the heart are inappropriate. Warm applications may be useful.

This plan of treatment is to be pursued for several days until it be determined whether or not the patient can escape the local changes which ensue if the circulation be not re-established in the affected district of the brain. If softening take place, the objects of treatment relate to the general condition of the system and to the hemiplegia.

The first of these objects embraces measures for improving and maintaining the nutrition of the body—namely, tonic remedies, a good diet, and an invigorating regimen. In cases of embolism diseases of the heart will often furnish indications for treatment.

Hemiplegia incident to the brain lesions resulting from embolism and thrombosis claims the same treatment as when it is due to a blood-clot in the brain. (Vide Cerebral Hemorrhage.) It consists of passive movements, together with rubbing and kneading the paralyzed limbs, the employment of electricity, and as much voluntary exercise of the muscles as practicable. These measures are important in order to prevent degenerative changes in the muscles and nerves of the paralyzed limbs, to promote the circulation and nutrition in these parts, and to develop as far as possible their functional capacity.

The treatment which has special reference to aphasia consists in persistent efforts to regain the memory of words and their vocal utterance. If the mutism be not complete, perseverance in exercises for these ends may sometimes accomplish much, and their importance is to be enjoined.

Thrombosis of the Cerebral Sinuses.

Thrombosis of the cerebral sinuses, especially of the longitudinal and lateral sinuses, may be the result of slowing of the circulation from weakened heart's action in patients, particularly children who are greatly enfeebled by some wasting disease. This form is called marantic thrombosis. Of a similar nature is the thrombosis sometimes observed in acute infectious diseases, particularly typhoid fever.

Thrombosis may also be occasioned by inflammation of the walls of the sinus or phlebitis of the sinus, secondary, as a rule, to disease of the cranial bones. Thrombi thus formed are usually infectious or purulent. Caries of the petrous portion of the temporal bone following inflammation of the middle ear or of the mastoid cells may be taken as one of the most frequent and typical causes of this variety of sinus-thrombosis. The lateral, petrosal, and in a less degree the cavernous, sinuses are most frequently the seat of purulent thrombi. Large carbuncles and severe erysipelatous inflammation about the head and neck may lead to sinus-thrombosis. Thrombosis of the cerebral sinuses may be accompanied by no very characteristic changes in the meninges and brain. Venous congestion, with punctate hemorrhages in the membranes and in the cortex cerebri, is often observed. Sometimes more extensive hemorrhages follow. The purulent thrombi are often accompanied with meningitis, and not infrequently abscess of the brain. They may lead also to the formation of embolic abscesses in the lungs and in other parts of the body.

Thrombosis of the cerebral sinuses can rarely be diagnosticated with any approach to certainty. The marantic variety in young children gives rise to symptoms analogous to those attributed to anæmia in the so-called hydrencephaloid affection of Marshall Hall—namely, somnolence and coma, with general and progressive prostration. The symptoms which, added to these, render thrombosis probable are tonic contraction of the muscles of the neck and back, strabismus, and ptosis. In adults indefinite cerebral disturbance gradually eventuates in coma. Turgidity of the external veins limited to certain situations on the cranium is a diagnostic feature in some cases. Thrombosis of the lateral sinus sometimes occasions venous congestion and œdema behind the ear. In a few recorded cases the thrombus has extended into the internal jugular, causing pain and swelling of that side of the neck. Thrombosis of the cavernous sinus has been observed to give rise to congestion of the intraorbicular veins, with prominence of the eyeballs, swelling of the eyelids, and congestion of the retina, with choked disc.

Prominent encephalic symptoms accompanying pyæmia, if preceded by caries of the bones about the ear, by carbuncles or erysipelas of the face, head, or neck, may be referable to purulent thrombosis of the cerebral sinuses.

Capillary Embolism and Thrombosis of the Brain.

Our knowledge of embolism and thrombosis of the cerebral capillaries is too limited for a determination of their pathological effects, symptomatology, and diagnosis. Pigment embolism is incident to malarial fevers, especially in their pernicious forms. Emboli or thrombi may consist of leucocytes, fat-drops, or the salts of lime. Future observations may establish an importance which does not at present belong to occlusion of the cerebral capillaries regarded from the standpoint of practical medicine.

Cerebral Hemorrhage.

ANATOMICAL APPEARANCES.—Intracranial hemorrhage may be conveniently divided into cerebral hemorrhage and meningeal hemorrhage. In cerebral hemorrhage, which will be first considered, the extravasation of blood is into the substance of the brain. Hemorrhages, usually punctate in character, are frequently found in and about tumors, around abscesses, and in foci of embolic softening, but only the so-called *spontaneous* cerebral hemorrhages will be considered in this article.

The ultimate cause of spontaneous cerebral hemorrhage is, probably without exception, disease of the cerebral arteries. Since the publication in 1868 of the investigations of Charcot and Bouchard the bursting of miliary aneurisms has been recognized as the predominant cause of spontaneous cerebral hemorrhage. These aneurisms are for the most part visible with the naked eye as little nodules of the size of a millet-seed or of the head of a pin; but they may exceed these dimensions, or, on the other hand, may be microscopic in size. In the majority of cases the whole circumference of the artery takes part in the aneurismal dilatation. The aneurisms are usually fusiform, but they may be round, and they are sometimes sacculated. The aneurisms are the result of pre-existing disease of the arterial coats. The most frequent form of this disease is chronic deforming endarteritis or atheroma. The aneurisms may also be caused by obliterating endarteritis and by a hyaline degeneration of the middle coat of the artery.

Aneurisms cannot be found in all cases of spontaneous cerebral hemorrhage. Atheroma, fatty degeneration, obliterating endarteritis, hyaline degeneration, mesarteritis and periarteritis, may so weaken the vascular walls as to prove efficient causes of hemorrhage without the development of miliary aneurisms.

In cases of cerebral hemorrhage occurring in the so-called hemorrhagic diathesis, as in scurvy, purpura, leucocythæmia, etc., it is not always possible to demonstrate any alteration in the vascular walls.

All circumstances which occasion increased arterial tension may serve as exciting causes of cerebral hemorrhage, but they never lead to cerebral hemorrhage unless there be disease of the arteries. Such circumstances will be mentioned under the head of Causation. Here may be mentioned, as an important exciting cause of cerebral hemorrhage, cardiac hypertrophy associated with elevated blood-pressure, particularly the cardiac hypertrophy associated with chronic Bright's disease.

Cerebral hemorrhage occurs by far most frequently in and about the basal ganglia of the brain; that is, in the nucleus caudatus, internal capsule, nucleus lenticularis, optic thalamus, and the adjacent part of the centrum semi-ovale. Much less frequent are hemorrhages in other parts of the centrum semi-ovale, in the pons, cerebellum, crura cerebri, medulla oblongata, and cerebral convolutions. The explanation of the frequency of hemorrhages in the opto-striate bodies (corpus striatum and optic thalamus) and in their neighborhood is found in the fact that the arteries which supply these parts are given off from the anterior, middle, and posterior cerebral arteries near their origin, and penetrate directly, as comparatively large vessels, the brain-substance, where they divide as terminal arteries. These arteries are subjected, therefore, to a relatively high pressure; whereas the arteries which supply the cortex cerebri become almost capillary in size before they dip down from the pia mater into the gray substance, and are therefore less exposed to the direct effects of increased blood-pressure. In the majority of cases the hemorrhage is due to rupture of some of the branches of the middle cerebral artery, which are destined for the supply of the lenticular nucleus, the greater part of the internal capsule and of the caudate nucleus, and a part of the optic thalamus.[1] Of special importance is involvement of the internal capsule; as lesions of most parts of the internal capsule are followed by permanent hemiplegia, whereas the hemiplegia which immediately follows hemorrhages confined to the lenticular nucleus or caudate nucleus is usually temporary, or at least is capable of great improvement.

Cerebral hemorrhage occurs in the form of capillary extravasations and in that of so-called apopletic foci in which the extravasated blood is collected into a mass. The *capillary hemorrhages* are of the lesser importance. They appear as isolated small red points, either scattered through the brain or collected together in a small space. The capillary hemorrhages may unite to make apoplectic foci. They are often found around the large extravasations; they are present around tumors, abscesses, centres of softening; and they may result from active congestion of the brain and from diseases attended with severe blood-changes. An *apoplectic focus* or clot is usually about the size of a hazelnut or of a pigeon's egg, but it may be no larger than a pea, or it may occupy nearly the whole of one cerebral hemisphere. If the extravasation take place in the vicinity of one of the lateral ventricles, the blood not infrequently finds its way into the latter. The blood may reach the outer surface of the brain either by breaking directly through the peripheral cerebral substance or by penetrating the third and thence the fourth ventricle, from which it can reach the exterior. Usually only a single hemorrhagic focus is present, but there may be several foci. It is not very uncommon to find remains of a previous hemorrhage.

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[1] According to Duret, the localization of hemorrhage in the region of the basal ganglia depends upon whether the rupture be in the region of the lenticulo-striate arteries, which supply the anterior part of the corpus striatum, or in that of the lenticulo-optic arteries, which supply the posterior part of the corpus striatum and a part of the optic thalamus. Among these arteries, which are branches of the arteria corporis striati, there is one particularly well developed, which supplies the region of the lenticular and the caudate nucleus. This artery, according to Charcot, is so liable to rupture that he designates it as "the artery of cerebral hemorrhage."

If the hemorrhage in the interior of the brain be of considerable size, the corresponding hemisphere is swollen, the convolutions are flattened, and the dura mater is tense. If the hemorrhage be recent, the apoplectic focus consists of a dark-red clot of blood mingled with the *débris* of cerebral tissue, and contained in a cavity the wall of which is composed of lacerated nerve-tissue surrounded by œdematous cerebral substance. Frequently, in proximity to the clot a number of capillary extravasations are present. If the extravasation be large, the substance of the corresponding hemisphere is anæmic. Careful examination by proper methods will usually reveal miliary aneurisms in or around the focus. Care must be exercised not to mistake for these aneurisms hemorrhages into the perivascular sheaths, the so-called dissecting aneurisms which are usually to be found about cerebral extravasations. If life be prolonged, retrograde metamorphoses occur in the extravasated blood. The color changes from dark red to brownish or yellow in consequence of the formation of blood-pigment in the form of granular and of crystalline hæmatoidin out of the red blood-corpuscles. From dissolved coloring matter the surrounding brain-substance becomes stained diffusely yellow or brown. The fibrin and the lacerated nerve-elements undergo disintegration and are absorbed, granular corpuscles making their appearance. While these regressive changes are going on, a connective-tissue capsule is formed by reactive inflammation around the focus. The final result is the production of a so-called apoplectic cyst. This cyst consists of a fibrous capsule enclosing a brownish or yellowish (rarely a clear) fluid. The cyst-wall and the fluid are stained by blood-pigment. Usually delicate threads of connective tissue traverse the cavity between the cyst-walls. Sometimes the walls of the cyst come together and only a pigmented cicatrix is left. It is probable that a cicatrix may also be formed without the intervention of a cyst. It rarely happens that the inflammation around the extravasated blood is sufficient to cause extensive softening, and abscess seems never to follow. When the hemorrhage is situated in the periphery of the convolutions, the changes are essentially the same, except that here the pia mater forms the outer covering of the focus.

The lungs of those dying in the early stages of cerebral hemorrhage are usually congested and œdematous, often containing extravasations of blood, particularly in the lower lobes.

The secondary degenerations which follow after a certain time (from six weeks to several months) hemorrhagic foci situated in certain regions of the brain deserve a brief mention here. These secondary degenerations, first studied exhaustively by Türck, follow not only hemorrhage, but softening and other destructive lesions cutting across the group of motor-fibres which, beginning in the anterior and posterior central convolutions, pass through the corona radiata, the internal capsule, the pes pedunculi, the pons, and the anterior pyramids of the medulla oblongata to the crossing of the pyramids, where the greater portion cross over and descend in the posterior part of the lateral column of the opposite side, a small portion continuing in the median region of the anterior column of the same side. Destructive lesions of the internal capsule especially, and also of the central convolutions, lower portion of the corona radiata and of the pes pedunculi, are followed by atrophy of the motor-fibres below the seat of lesion. The secondary degeneration not only consists in atrophy of the nerve-fibres, but is accompanied by increase of the connective tissue between the fibres. It is at first characterized by the appearance of large numbers of granular cells. The atrophied bundles are visible on fresh specimens as gray bands following the course which has just been described as that of the motor—or, as they are called, pyramidal—fibres. Upon making a cross-section of the spinal cord in a case of secondary descending degeneration a triangular gray patch will be observed in the posterior part of the lateral column on the side opposite the lesion in the brain, and a narrow gray band, not always visible to the naked eye, in the anterior column near the median fissure on the side of the lesion. The contrast in color between the healthy and the degenerated parts becomes more distinct in specimens hardened in chromic acid. Lesions confined to the nucleus caudatus, lenticular nucleus, or to regions of the brain other than those named are not followed by these secondary degenerations. The degeneration does not affect the peripheral motor nerves.

CLINICAL HISTORY.—Of the several pathological conditions which occasion sudden coma or apoplexy, cerebral hemorrhage is the most frequent in its occurrence. Some writers limit the term apoplexy to sudden coma caused by extravasation of blood. In a large majority of cases the apoplectic attack is without premonition. It is, however, sometimes preceded by certain cerebral symptoms, such as a sense of weight or fulness, vertigo, tinnitus aurium, and flushing of the face. These symptoms, however, are not sufficiently significant to warrant a prediction of impending apoplexy, especially if the patient have never had an attack. It is not uncommon for an attack to occur at a time when the person attacked feels unusually well.

In an apoplectic attack the loss of consciousness may be so abrupt that the patient, if standing or walking, falls to the ground as if felled by a blow; but in most instances the coma is developed, not instantaneously, but during a period ranging from a few moments to half an hour or even longer. It has happened to me to witness an attack which occurred when I was interrogating a patient who had just stated that he had felt on that day unusually well. I noticed suddenly a change in his appearance, as if he were mentally agitated; his expression changed and he winked with his left eye only. He made an effort to rise and fell from the chair. The right side was paralyzed. He did not at once become unconscious, but was irrational, attempting to speak, being unable to articulate, and frequently making efforts to get up from the sofa on which he had been placed. The respiration soon became heavy, with slight stertor and puffing of the lips. He vomited freely. He became profoundly comatose in about half an hour. He remained in the comatose state and died within twenty-four hours.

The coma in different cases is more or less profound. When complete, the patient cannot be roused in any degree by efforts to awaken consciousness; but in many cases there is not this total loss of mental faculties. Voluntary movements in some cases are entirely wanting; and except that the movements of respiration continue, the body is as motionless as a cadaver. In the majority of cases, however, the patient exerts the will, although all manifestations of intelligence may be wanting. In this way the existence of unilateral paralysis becomes apparent. The movements are limited to the upper and lower extremity of one side, and it is evident that hemiplegia is added to the apoplexy. The paralysis is generally at first complete, and the paralyzed limbs are motionless as regards volition. The respirations are slow, the rhythm is sometimes irregular, and the inspirations are stertorous or snoring. If the coma be profound, the buccinator muscles and the lips are flaccid and are puffed out in expiration. The pulse is usually diminished in frequency, full, and hard, the artery striking against the finger like a metal rod. Exceptionally the pulse is small and feeble. The face is frequently flushed, and, if respiration be much embarrassed, more or less livid, but it is sometimes pallid. The surface in most cases is warm, but sometimes it is cool. At first the temperature of the body, as determined by the thermometer, is lowered from one to two and a half degrees, but after twenty-four hours there is usually a considerable increase of the body-heat. The pupil may be contracted or dilated, or, without either contraction or dilatation, immovable, and some disparity between the two pupils is not uncommon. The muscles are generally flaccid, but in some cases rigid. Rigidity of the muscles is the rule in intraventricular hemorrhages. Convulsive movements are infrequent, but they sometimes occur, especially when the hemorrhage involves the motor cortical region. These are usually limited to one side, and to the side not paralyzed. To this rule there are exceptions. The limbs are sometimes rigid on the non-paralyzed side. Vomiting is usual at or shortly after the attack.

These are the prominent symptoms which characterize the apoplectic state. Its duration varies. It may last for a few moments only, the patient gradually emerging from it, or unconsciousness slowly disappears after the lapse of a few hours, or the state may continue for days and then pass off. As a rule, if the coma persist without any improvement for eight or ten hours, and especially if during this period the coma become more and more profound, improvement is not to be expected, and the attack will prove fatal. On the other hand, the attack may prove fatal in a few moments, the mode of dying being by apnœa; or death takes place after several hours; or, again, the apoplectic state continues, and proves fatal after the lapse of several days, in the latter case the mode of dying being by asthenia and apnœa combined.

Patients emerging from the coma appear as if awakened from sleep, and they rarely evince surprise or ask what has happened to them. A noticeable appearance, lasting for several days, is a lateral deviation of the eyes, and sometimes there is rotation of the head in a direction opposite to the paralyzed side. With more or less increase of the body-heat pain is referred to the head. Sometimes there is delirium, and spasmodic movements or contractions of the paralyzed muscles may occur. These symptoms denote circumscribed inflammation of the brain surrounding the blood-clot. As a rule, they disappear after a few days.

The symptoms in apoplexy dependent on cerebral hemorrhage are essentially the same as when coma from compression of the brain follows an injury to the head. The extravasation of blood produces a shock or stunning effect upon the brain proportionate to the rapidity and the amount of the hemorrhage, and the pressure affects the circulation in the whole mass of brain. The pressure impedes the circulation in the substance of the brain, and the apoplectic phenomena are measurably due to the want of a proper supply of blood therein. The production of apoplexy depends on the suddenness of extravasation. If it take place gradually, apoplexy is not induced, and if it be not sufficient to destroy life within a brief period, the circulation may become restored sufficiently for the restoration of consciousness.

The symptoms which remain after the disappearance of the coma depend upon the situation of the clot. In the next article will be considered symptoms referable to hemorrhage and other lesions occupying different parts of the encephalon. In the present article the hemorrhages in the usual situation—namely, in the basal ganglia and their neighborhood—will be taken as the basis of description. It is important to distinguish between symptoms due to the destruction of a certain part of the brain by the hemorrhage and symptoms dependent upon pressure, œdema, and other disturbances of nutrition in parts adjacent to the clot. Evidently, the first set of symptoms are permanent, while the second set of symptoms may be, and usually are, recovered from.[1]

Hemiplegia is a constant effect of cerebral hemorrhage when the extravasation takes place in the situations in which it occurs in the great majority of cases. After the apoplectic state disappears the hemiplegia remains; but in a small proportion of cases the hemiplegia occurs without loss of consciousness, this proportion being much smaller than in cases of cerebral embolism. The attack of hemiplegia without apoplexy is often sudden, but the paralysis is sometimes developed gradually. The difference as regards this point probably depends on whether the extravasation take place quickly or slowly. The hemiplegia, however, may be at first complete, although unaccompanied by any impairment of consciousness. The following account of the hemiplegia dependent on cerebral hemorrhage is applicable to cases with or without an apoplectic attack.

The paralysis may embrace both motion and sensation, or it may be limited to motion. In the great majority of cases, if sensation be at first lost, it is shortly regained, the motor paralysis continuing. The paralysis may be complete or incomplete, and if incomplete it may be of every gradation as regards degree. The paralysis may be complete in one and incomplete in the other extremity. If incomplete in both extremities, the degree of paralysis is generally greater in one than in the other extremity, and in the vast majority of cases the greater degree is in the upper extremity.

In cases of complete motor paralysis movements may frequently be produced by pricking the integument, tickling the sole of the foot, or applying a heated iron to the surface. These movements take place without volition, and much to the surprise of the patient if the mental faculties be intact. They are purely reflex. They are produced in the lower, but rarely in the upper, extremity. In most cases of cerebral hemiplegia the tendon reflexes are increased on the paralyzed side, and to a less extent on the healthy side; whereas the skin reflexes, including the cremaster reflex, are diminished on the paralyzed side. Direct mechanical irritation of the paralyzed muscles will often cause marked contractions which are possibly of a reflex nature. There is a notable difference in different cases as regards the condition of the muscles of the paralyzed limbs. They are in some cases soft and flaccid, offering no resistance to the movement of the limbs in different directions; but in other cases they are hard and rigid, resembling sometimes muscles in tetanic contraction, and offering much resistance to movements of the limbs.

The paralysis generally involves the muscles of the face, the side affected, as a rule, corresponding to the paralyzed limbs. Exceptionally, the limbs on one side and the face on the opposite side are affected. This is called crossed paralysis (*paralysie alterne*). Only the muscles of the lower facial district—namely, the muscles of the mouth, cheek, and nose—are affected. On the paralyzed side of the face the expression is abolished, and when the facial muscles are excited to action, as in speaking or smiling, the mouth is drawn to the opposite side. Voluntary power over the frontal muscle and the orbicular muscle of the eye is rarely lost. The patient is generally able to close the eye on the affected side, but he cannot, as a rule, close the eye on the affected side while the other eye remains open. The power over the orbicular muscle in a certain proportion of cases is very obviously impaired. The patient usually winks on the affected side.

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[1] It is possible that symptoms due to destruction of brain-substance may disappear by other parts of the brain—for instance, corresponding parts of the opposite hemisphere—taking on vicariously the functions of the destroyed district. This supposition, however, cannot be considered as proven. There is no way of distinguishing between symptoms resulting from destruction of brain-substance and those dependent on disturbed nutrition of brain-substance, except that the latter disappear more or less rapidly.

If the patient protrude the tongue, the extremity frequently points to the paralyzed side of the face. In the instances in which the tongue has been thought to point to the opposite side there was probably an error of observation. The tongue may appear to be thus deflected, owing to the different situations of the upper lip and angle of the mouth on the paralyzed side. If the mouth on this side be drawn by the finger laterally, so as to be in symmetry with the opposite side, the tongue, which had appeared to be deflected, may be seen to be protruded in a direct line or to point to the paralyzed side. The loss of certain teeth may cause the tongue to protrude in an abnormal direction. The deflection of the tongue to the paralyzed side may be explained by the action of the stylo-hyoid muscles and of the posterior belly of the digastric, which effect the protrusion of the tongue by elevating the base attached to the hyoid bone. When only the muscles of one side act, the base is elevated upon this side only, and the tip of the tongue is protruded to the opposite or paralyzed side; or the deviation of the tongue may be the result of paralysis of the hypoglossal nerve supplying the genio-glossus muscle. The velum pendulum palati on the side of the paralyzed limbs is sometimes involved in the paralysis. This is shown by an abnormal flaccidity of the velum on one side, which remains motionless when the muscles of the other side are seen to contract on depressing the tongue with a spatula. The uvula in these cases is deflected to the unaffected side; but this alone is not evidence of paralysis, as deflection of the uvula to one side is frequently observed in healthy persons. Occasionally there are ptosis, diverging strabismus, and dilatation of the pupil, sometimes on the same side, but in the great majority of cases on the side opposite to that of the paralyzed limbs. These symptoms show that the third cranial nerve is involved. The other nerves distributed to the eye are very rarely involved. The fifth nerve usually escapes. The eighth nerve is unaffected, save in certain cases of hemiplegia with coma about to terminate fatally. The nerves of special sense almost always escape. The muscles of respiration are not usually involved in the paralysis, the respiratory movements on the two sides being equal. Sometimes, however, the respiratory muscles on the hemiplegic side are paretic, as is made evident by a slower and less complete expansion of that side of the chest than of the opposite side. This lessened expansion may explain a tendency of pulmonary complications, which are common in cerebral hemorrhage, to affect the lung on the side opposite to the hemorrhage. The muscles of the neck and trunk in general remain unaffected. It is a law that muscles which usually act co-ordinately or consensually, such as the muscles of the eye and the respiratory muscles, escape in hemiplegia. The trapezius muscle on the paralyzed side is usually paretic, causing a sinking of the corresponding shoulder.

The clinical history of hemiplegia from cerebral hemorrhage presents much diversity in different cases. It is impossible to judge at first of the prospect as regards the amount of improvement which will take place. More or less improvement takes place in the majority of cases. The improvement is usually gradual. The sensibility, if this have been lost, is first restored, and it may become abnormally increased on the affected side. The paralyzed condition of the face may diminish or disappear in a short time. Improvement, as a rule, begins sooner and progresses more rapidly in the lower than in the upper extremity. Frequently the patient is able to walk without much difficulty, while the upper extremity remains almost completely paralyzed. The manner of walking varies in different cases. If the rotating muscles of the thigh be relatively but little affected, the limb is thrown forward so as to describe the arc of a circle; but frequently the body is inclined to the opposite side and the limb is dragged after its fellow. Sometimes it is brought forward with a jerking movement. Tremulousness of the limb in walking is sometimes observed. The patient often experiences difficulty in walking, from a depression of the toes which renders him liable to trip.

A remote effect upon the paralyzed parts is deficiency of volume or wasting, this effect being proportionate to the degree of permanent paralysis. Atrophy of the paralyzed muscles, however, occurs later, and is much less in degree, than in cases of paralysis from affections of nerves or of the spinal cord. The atrophy which takes place is due to disuse of the muscles. Paralyses dependent on cerebral lesions are distinguished by the absence of a trophic disturbance of the affected muscles. The paralyzed muscles also, as a rule, respond to the excitation of electricity; that is, their electro-contractility is preserved. The temperature in hemiplegia becomes less on the paralyzed than on the opposite side; the pulse has less amplitude; and the veins sometimes show congestion. There may be œdema on the paralyzed side. Remote effects relate to the contraction of certain muscles. Permanent contraction of the flexor muscles, especially of the forearm, is frequently observed. It is in some cases due to a contraction sufficient to overcome antagonizing muscles, and in other cases to a greater degree of paralysis of the extensor muscles, the flexors contracting simply because they are not antagonized. In the latter case the flexed parts are restored with a little force; and in the former the contracted muscles offer resistance to efforts for extension. It is customary to distinguish a primary and a secondary contracture. The primary contracture appears a few days after the attack, and is attributed to reactive inflammation. It generally disappears. The secondary contracture appears several months after the attack, and is permanent. It is attributed by many to secondary degeneration of the spinal cord. It is a bad prognostic sign. Weir Mitchell has observed that the growth of the nails on the limbs of the affected side is arrested, and that a resumption of their growth is a forerunner of a return of power over the paralyzed muscles. There is a tendency to the formation of bed-sores, particularly on the nates and over the trochanter of the paralyzed side.

The mental condition in different cases of hemiplegia from cerebral hemorrhage presents great variation. The faculties of the mind are generally, if not invariably, more or less impaired. Patients are moved to tears or laughter by trivial circumstances. A marked change in character is frequently observed. Patients are liable to become impatient and irritable. They lose self-reliance, and are easily influenced by those around them. This fact sometimes has important medico-legal bearings. The tendency is to progressive impairment of the mental faculties, ending at last in imbecility. Cases differ much as regards the rapidity of progress toward this result. The mental deterioration doubtless often depends in no small measure on the deficiency of the exercise of the mental faculties after paralysis has occurred.

Aphasia has been already considered as incident to cerebral embolism and thrombosis. (Vide p. 653.) It occurs in a certain proportion of the cases of hemiplegia caused by cerebral hemorrhage. There may be either total loss or more or less impairment of speech, and the aphasia may be either ataxic or amnesic. As regards the dependence of this symptom on the situation of the clot, the facts stated in connection with Cerebral Embolism and Thrombosis are applicable. Recovery from the aphasia is less likely to take place when dependent on the presence of a clot than when it depends on occlusion of an artery. Aphasia is to be distinguished from the difficulty in articulation arising from paralysis of the tongue and lips.

Causation.—The dependence of cerebral hemorrhage on conditions pertaining to the vessels within the brain has been considered in connection with the anatomical characters, and also its relation to disease of the heart. The local conditions are rarely present prior to the middle period of life; hence, apoplexy and hemiplegia, caused by cerebral hemorrhage, seldom occur in persons less than forty years of age. In the majority of cases the age is more than sixty. Cerebral hemorrhage is somewhat more frequent in males than in females. The various causes of arterio-sclerosis are remote causes of cerebral hemorrhage. Such causes are—alcoholism, syphilis, gout, and hereditary predisposition. As already stated, simple hypertrophy of the left ventricle of the heart may have a causative relation to cerebral hemorrhage by increasing arterial tension, assuming the existence of the lesions of the arteries of the brain which have been described. Hypertrophy associated with mitral stenosis or insufficiency or with aortic lesions has no causative agency. It is doubtful if venous congestion, incident to dilatation of the right side of the heart or other causes, ever has much agency in determining the occurrence of hemorrhage. Active congestion from causes other than simple hypertrophy of the heart may have this effect. Persons have been attacked when under mental excitement, as in public speaking or in a fit of anger. In two cases under my observation an apoplectic attack took place during sexual intercourse. Straining at stool, violent muscular exercise, the throes of labor, and drunkenness have in some cases appeared to produce an attack. All these are exciting causes. In the larger proportion of cases the hemorrhage takes place without any obvious exciting cause. In a considerable proportion of cases it occurs during sleep. I have known it to occur in a patient considerably reduced by prolonged menorrhagia, and to follow much loss of blood from hemorrhoids.

Formerly, much importance was attributed to a so-called *apoplectic constitution*, consisting of shortness of the neck, with considerable *embonpoint* and what is known as a full habit. An analysis of a considerable number of cases shows that no reliance is to be placed on these or any other external characters as denoting a predisposition to cerebral hemorrhage.

Diagnosis.—Apoplexy from cerebral hemorrhage may be confounded with several morbid conditions which involve coma more or less complete. There is little risk of confounding apoplexy and syncope except for a short time, for syncope is of brief duration. It is characterized by death-like pallor, coldness, catching of the breath, and great feebleness or extinction of the pulse—symptoms which do not belong to the clinical history of cerebral hemorrhage. The coma which sometimes follows an epileptic paroxysm bears a resemblance to apoplexy. Knowledge of the fact that the comatose state has been preceded by violent convulsions, with difficult and noisy respiration from laryngeal spasm, will at once settle the character of the attack. Without this knowledge the foam or blood on the lips and the wounded tongue show that epileptic convulsions have occurred. The doubt can only arise when the patient is a stranger found in an unconscious state. The physician generally is aware or is able to ascertain that the patient is subject to epileptic fits. A paroxysm of epilepsy may act as an exciting cause of apoplexy. Hysterical coma is another condition which may suggest the existence of apoplexy. This is to be discriminated by the characteristic phenomena of hysteria having preceded the comatose state; by convulsive movements in some cases, into which volition enters more or less; and by the absence of stertor, the mobility of the iris, and the restoration of consciousness on resorting to the cold douche. The coexistence generally, in uræmic coma, of epileptiform convulsions if dropsy do not coexist, suggests the probable existence of renal disease, and the urine is found to contain either albumen or casts, or both.

Profound alcoholic intoxication may be mistaken for apoplectic coma. The following are the differential points: The odor of spirits or of wine in matters vomited and in the breath of the patient; absence of hemiplegia; the pulse usually feeble or soft and increased in frequency, not diminished in frequency and hard as it is usually in apoplexy; the pupils dilated. The patient can generally be roused sufficiently to exhibit some of the manifestations of drunkenness in the manner of speaking. Pouring cold water upon the head from a considerable height is a ready mode of partially arousing the patient. It is important to make this discrimination. To call apoplexy drunkenness would be an unfortunate error, and the reverse would be annoying to the practitioner.

In deep narcotism due to opium-poisoning the patient by vigorous efforts can generally be roused for an instant. The respirations are notably diminished in frequency, but there is no stertor. The pupils are contracted. There is no hemiplegia.

Apoplectic coma caused by active hyperæmia (congestive apoplexy) and by meningeal hemorrhage is excluded by the coexistence of hemiplegia. Hemiplegia may generally be ascertained during the apoplectic state. The patient moves the extremities of one side, while those of the other side remain motionless. He may manifest sensibility only when the integument of one side is pinched or pricked. The face may be drawn to one side, especially if manifestations of pain can be produced. The paralyzed members, when raised and allowed to drop, fall as if inanimate. The absence of the cremaster reflex on the paralyzed side, and its presence on the other side, may assist in determining the hemiplegic side during coma. On the other hand, meningeal hemorrhage and cerebral hyperæmia do not, as a rule, give rise to hemiplegia. The existence or otherwise of hemiplegia is of course readily ascertained after the patient emerges from the apoplectic state. The exclusion of embolism as the cause is more difficult and is not always practicable. For the differential points the reader is referred to the diagnosis of embolic apoplexy (p. 655). Hemiplegia without apoplexy may be caused by cerebral affections other than hemorrhage, embolism, and thrombosis—namely, by tumors and abscess of the brain. These affections give rise to cerebral symptoms prior to the occurrence of hemiplegia, and not infrequently to paralysis limited to one or more of the cranial nerves. If the hemiplegia be not preceded by symptoms pointing to tumor or abscess, and if functional hemiplegia be excluded, the differential diagnosis lies between hemorrhage, embolism, and thrombosis. If the hemiplegia have occurred suddenly, thrombosis may be excluded. The points involved in discriminating embolic hemiplegia have been stated. The occurrence of spinal hemiplegia in rare instances is to be remembered in the diagnosis.

The importance of a careful examination of the head for the evidence of injury in cases of coma when nothing is known of the history may be here alluded to. A patient admitted into hospital in a comatose condition, without this precaution may be supposed to have apoplexy, when subsequently, perhaps at the autopsy, fracture of the skull with meningeal hemorrhage may be found.

PROGNOSIS.—An attack of apoplexy from cerebral hemorrhage may prove fatal within a very short period, sometimes within a few moments. When it proves suddenly fatal, the coma is at once profound and accompanied by great disturbance of respiration and loss of deglutition, death taking place by apnœa. A fatal termination frequently takes place after the lapse of from twelve to twenty-four hours. The coma in these cases continues, and becomes more profound; the respiration is more and more embarrassed; tracheal râles occur; deglutition is gradually lost; the urine is retained or flows away involuntarily; involuntary dejections occur; and the mode of dying is chiefly by apnœa. If no improvement take place within ten or twelve hours, the coma remaining or becoming complete, disturbance of the respiration and deglutition being more marked, there is little room for the expectation of amendment, and the case will probably end fatally within a short period. In other cases life is prolonged for several days, the patient not emerging from the apoplectic state, but more or less improvement, as regards consciousness, taking place in some cases. The mode of dying in these cases is by apnœa and asthenia combined.

On the other hand, the apoplectic attack may pass off in a few moments or a few hours, hemiplegia remaining. The paralysis at first is generally complete, and the sensibility on the affected side, as well as the power of motion, is often lost. The latter usually returns more or less speedily; and more gradually there is improvement as regards the power of motion. In most cases this improvement goes on progressively until a certain point is reached, and at this point, which varies much in different cases, the improvement stops. More or less of the paralysis remains permanently as a rule. It is impossible to judge beforehand of the degree of improvement which may be expected as regards the paralysis. The completeness of the paralysis at first is probably due to the shock or contusion of the parts of the brain in proximity to the extravasated blood. After recovery from this the amount of paralysis will depend on the extent of damage which the brain-substance has received.

The occurrence of cerebral hemorrhage involves liability to its recurrence. Of those who recover from a primary attack, a considerable proportion are again attacked after intervals of varying duration in different cases. As a rule, the hemorrhage in successive attacks is greater, rendering them more severe or dangerous. It is, however, to be borne in mind that recurrences do not always occur. Patients with hemiplegia resulting from a clot in the brain sometimes live for twenty years or longer, a second hemorrhage not taking place. In a recurrent attack the hemorrhage may be on the same side or in the other hemisphere, more frequently the former. Instances have occurred in which a hemorrhage occurred simultaneously in both hemispheres, causing double hemiplegia. The mental condition after the occurrence of hemorrhage has been already referred to.

TREATMENT.—To bleed or not to bleed is the first question which arises in the treatment of an attack of apoplexy from cerebral hemorrhage. The answer is neither affirmative nor negative as regards cases collectively, but it may be either according to the symptoms in individual cases.

If the apoplectic attack be accompanied by symptoms denoting determination of blood to the head and increased arterial tension—a flushed face and strong pulsation of the carotids—venesection is indicated, especially if the patient be robust or full-blooded and the action of the heart be abnormally strong. Under these circumstances it is fair to infer that the giving way of the cerebral artery was due in a measure to the hyperæmia or to the arterial tension as an accessory or exciting cause. Bleeding will not undo the mischief which has already taken place, but it may prevent an increase or a renewal of the hemorrhage. In a certain proportion of cases the symptoms are essentially different. The face may be pallid, the carotids do not beat strongly, and the pulse may be small and weak. Venesection under these circumstances is contraindicated. If the abstraction of blood be decided upon, it should be by venesection. It is important that the effect of the bleeding be produced promptly, and for this reason cups or leeches do not as well meet the indication. The amount of blood abstracted should be determined by the effect. A large bleeding is rarely indicated.

Generally, it is advisable to administer an active cathartic. Croton oil, from the facility with which it is administered, its efficiency and its quickness of operation, is to be preferred to any other cathartic. Three or four drops may be given at once and repeated in three or four hours if free purgation be not produced. Its operation may be promoted by large, stimulating enemas. An active cathartic should not be given if the pulse be notably small or feeble.

The patient should be placed in a cool, airy apartment; the head should be moderately raised; cold or evaporating lotions should be applied to the head, or, if the head be hot and flushed, the ice-cap; stimulating pediluvia may be employed if the extremities be cool; and everything constricting the neck and chest should be removed. Attention is to be directed to the bladder, and if required the use of the catheter is not to be delayed.

These are the measures to be pursued until it is decided whether the patient is to emerge from the apoplectic state. If after the lapse of several hours the symptoms denote a hopeless condition, the physician must content himself with measures which appear to contribute to the comfort of the patient, for he is, in fact, unconscious of suffering. Purgatives and other perturbating remedies under these circumstances will only tend to hasten the fatal termination. If, on the other hand, the attack pass off, the measures indicated are simply quietude and an unstimulating diet until symptoms denote inflammation around the clot. These do not call for active treatment. Cool applications to the head, a sinapism to the neck, saline laxatives, and perfect quietude are sufficient. The vital powers should not be impaired by active treatment. The recovery is effected by the disintegration and absorption of the clot, and this requires many weeks or months. The processes of disintegration and absorption are not promoted directly by any medicines. There is no ground for supposing that mercury, iodine, or other drugs have any direct influence over these processes. Blisters or other modes of counter-irritation are of no utility. Electricity applied to the brain is a doubtful measure, and is not without hazard of injury. As regards the clot, nothing can be done except by nourishing the patient well, avoiding stimulants, preventing as far as possible everything which disturbs body or mind, and meeting any symptomatic indications. The case is now one of hemiplegia dependent on the presence of the clot and the damage which it has occasioned.

A tendency to the formation of bed-sores is counteracted by observance of strict cleanliness and by not allowing continued pressure on susceptible parts.

Active treatment directed to the paralyzed muscles is contraindicated while the symptoms denote inflammation or irritation at the situation of the hemorrhage. Gentle rubbing of the paralyzed limbs and passive movements, however, may be resorted to with but little delay. More energetic measures are indicated after two or three months. The objects of treatment are the promotion of the circulation and nutrition in the paralyzed muscles, together with the increase, as far as possible, of their functional capacity. The means are massage or kneading, rubbing with stimulating liniments, passive movements, electricity, and volitional exercise. Kneading, rubbing, and passive movements should be systematically and perseveringly employed. If they do not occasion fatigue, they cannot be too much employed. It is to be borne in mind that paralyzed muscles do not of themselves resume at once their normal functional capacity, even after the conditions causative of the paralysis are removed; and having been for a considerable period paralyzed, they may remain so indefinitely if appropriate measures be not made for their restoration.

Electricity is a potential agent in promoting the objects stated, especially when the paralyzed muscles respond not at all or feebly to the will. It promotes circulation, nutrition, and is a substitute for the will as regards the functional exercise of the muscles. With respect to the choice between the two currents, the methods to be adopted, etc., the reader is referred to treatises on the practical use of electricity or to works devoted to nervous diseases. This agent is apt to be inefficiently employed. It may be injudiciously used, and it is potential for harm as well as good. When practicable, therefore, its employment should be under the direction of an experienced physician who has given special attention to electro-therapeutics.

When the paralysis is not complete, the most efficient measure for securing as much improvement as is compatible with the amount of damage which the brain has received is voluntary exercise of the paralyzed muscles. A patient who has been for some time a paralytic is in a condition not unlike that of the infant who has not learned to use the muscles. Persistent exercise in the one case, as in the other, is the means by which development and power are to be acquired. There is reason to believe that in not a few cases paralysis continues until it becomes fixed at a certain point, when persevering exercise in conjunction with other measures might have led to more or less improvement. It is often difficult to enforce this measure on account of its simplicity.

The liability to the recurrence of cerebral hemorrhage renders it important to observe all possible precautions by way of prophylaxis. Placing the system in the best possible condition by means of a well-regulated diet and regimen, and avoiding exciting causes, will afford all the security which can be obtained. It is not probable that any protection is afforded by reducing the powers of the system, and injury may thereby be produced. It is injudicious to adopt a diet which is insufficient for the wants of the system, or to resort to repeated bloodlettings, cathartics, or other lowering measures. In striving to avoid excesses and imprudences of all kinds care must be taken not to err in the opposite extreme. Mental occupation within certain limits is advisable.

A liability to hemorrhage if an attack have never occurred cannot be estimated with any degree of certainty. Apoplexy and paralysis are affections which many persons apprehend, and if certain cerebral symptoms be experienced, especially vertigo, the apprehension is often the source of much unhappiness. The suggestion by the physician that there is danger of these affections is an indiscretion which I have known to prove most calamitous. When apprehension is felt, the physician is warranted in giving assurances that vertigo and other cerebral symptoms are sufficiently common without being followed by apoplexy or paralysis, and that these affections are rarely preceded by obvious premonitions. Needless uneasiness may oftentimes be removed by these assurances.

The treatment of hemiplegia from cerebral hemorrhage is the same whether it occur with or without the occurrence of apoplexy.

In connection with the subject of cerebral hemorrhage the question arises, Can the particular situation of the extravasated blood be ascertained by means of the symptoms? This question arises in respect to the particular situation of softening from embolism and thrombosis, and also with reference to the localization of circumscribed inflammation or abscess of the brain, cerebral tumors, and atrophy. In order to avoid repetitions, the facts at present known concerning the localization of cerebral diseases are concisely stated under a separate heading.

Topical Diagnosis of Cerebral Diseases.

In the present article will be considered the diagnosis of cerebral diseases according to the situation of the lesion. The possibility of the topical diagnosis of cerebral diseases is based upon the fact that certain parts of the brain are endowed with special functions, the excitation or the abolition of which gives rise to characteristic symptoms. Our knowledge of the localization of these functions is due partly to physiological, but chiefly, so far as man is concerned, to clinical and pathological observations. This knowledge is still very imperfect. In the present article only such facts will be considered as are reasonably certain.

The diseases of the brain are conveniently classified as *focal* or *local* diseases, in which the lesion is confined to a circumscribed part of the brain, and *diffused* or *general* diseases, in which a large part of the brain is affected. Examples of focal cerebral diseases are — circumscribed hemorrhages, foci of softening either embolic or thrombic, localized encephalitis, abscess, and tumors. Examples of diffuse diseases of the brain are—anæmia and hyperæmia, diffuse encephalitis, and the conditions produced by concussion and compression of the brain. It is of course only the local diseases of the brain which admit of a topical diagnosis.

The symptoms which may be produced by local diseases of the brain are either *direct* or *indirect*. The direct symptoms are those due to irritation or to destruction of the part of the brain which is the seat of the lesion. The indirect symptoms are those referable to the influence exerted by the lesion upon neighboring or remote parts of the brain. Direct symptoms caused by destruction of brain-substance are permanent, except so far as some other part of the brain may be capable of assuming vicariously the functions of the destroyed part. Indirect symptoms are to a great extent variable and inconstant, and in the case of many local lesions they disappear after an indefinite period, leaving only direct symptoms. Although certain symptoms, such as coma, and in most instances headache, delirium, and vomiting, can be recognized as indirect symptoms, yet it is in general impossible from their nature alone to distinguish the indirect from the direct symptoms. This distinction, however, can often be made in the course of time by noting that certain symptoms disappear, and are therefore probably indirect, whereas other symptoms remain permanently and are considered to be direct. For instance, a fresh hemorrhage in that part of the internal capsule occupied by the motor tracts may give rise, in addition to hemiplegia, to anæsthesia, aphasia, and strabismus, the last three symptoms being indirect, as they are referable not, like the hemiplegia, to destruction of the part named, but to an influence exerted upon other parts by pressure, inflammatory œdema, etc. After a time anæsthesia and the other secondary or indirect symptoms disappear, and the hemiplegia remains as the direct symptom of the lesion. It is evident that the fewer the indirect, and the more pronounced the direct symptoms, the greater the probability of making an accurate diagnosis of the seat of the lesion. Hence it follows that a topical diagnosis is often impossible in the early stage of cerebral hemorrhage or softening, but can frequently be made at a later stage, when the indirect symptoms have disappeared.

For various reasons mistakes are especially liable to be made in the topical diagnosis of tumors. Not only do tumors often grow in parts of the brain the function of which is very imperfectly understood, but they give rise to many diffuse and indirect symptoms, and by their slow development they frequently displace nerve-elements without destroying them.

Hughlings-Jackson has graphically designated as *discharging* lesions those which irritate nerve-elements, and as *destroying* lesions those which annul their function. A discharging lesion in a motor tract produces spasm—a destroying lesion, paralysis.

After these few remarks by way of introduction, the lesions situated in the different parts of the brain will be briefly considered with reference to their topical diagnosis.

1. *Cerebellum.*—Hemorrhage in the cerebellum occurs much less frequently than in the cerebrum. The proportion is variously estimated as from 1 to 12 to 1 to 35. Embolism of the cerebellar arteries is even more unusual. Other local diseases of the cerebellum are abscesses, encephalitis, tumors, and atrophy.

Lesions, often extensive, of the cerebellum may remain entirely latent during life. This is believed to be the case only when the lesion is localized in one cerebellar hemisphere, and when its influence does not extend beyond the seat of disease. In a remarkable case reported by Ebstein,[1] nearly the whole left hemisphere of the cerebellum was occupied by an osteoma which did not encroach upon surrounding parts. The patient died of a pulmonary affection, having manifested no symptom referable to the nervous system. Characteristic, although not pathognomonic of lesions of the cerebellum, are disturbances of co-ordination designated as cerebellar ataxia and dizziness. These symptoms are believed to be the result of an affection of the vermiform process or middle lobe of the cerebellum, either directly when it is the seat of disease or indirectly by an encroaching lesion. The gait in cerebellar ataxia, which affects chiefly the lower extremities, resembles that of a drunken man, and not that of a patient affected with locomotor ataxia or posterior spinal sclerosis. The symptoms in the great class of cerebellar lesions, such as fresh hemorrhages and tumors which act upon adjoining parts, particularly upon the pons and medulla oblongata, are various, but they can sometimes be utilized in making the diagnosis. Of symptoms produced by cerebellar disease, but not directly referable to disturbances of function of the cerebellum itself, may be mentioned vomiting, amblyopia, and amaurosis; headache, most frequently in the occipital region; hemiplegia, either corresponding to the side of the lesion or to the opposite side; less often epileptiform convulsions and spasm; and sometimes irregular and infrequent action of the heart.

2. *Crura Cerebelli.*—No characteristic symptoms of lesions of the superior or of the inferior peduncles of the cerebellum have as yet been discovered. On the other hand, lesions of the middle peduncles, the crura ad pontem, have repeatedly been observed to be attended by so-called forced or compelled movements. These peculiar movements appear, according to Nothnagel, when the lesion acts as an irritant and does not completely sever the connection of the affected crus with the cerebellum. Recognized forms of these compelled movements are rotary movements of the body around its long axis; the assumption of a compelled position upon one side, to which the patient instantly returns when moved; the tendency to fall to one side, accompanied with dizziness; and the deviation of the head and of the eyes, most characteristic when one eye is turned upward and the other downward. These movements may occur during coma, but they are more characteristic of disease of the crura cerebelli when present in the waking state. With the exception of the rotary or "circus" movements and the peculiar deviation of the eyes, similar compelled movements have been observed in diseases

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[1] *Virchow's Archiv*, Bd. 49, p. 145.

situated in other parts of the brain. The movements may be toward the side of the lesion or toward the opposite side.

3. *Pons Varolii.*—Hemorrhages occur in the pons with about the same frequency as in the cerebellum. Emboli rarely lodge in the basilar artery, because its size exceeds that of either vertebral artery, and because the latter is unfavorably placed for the reception of an embolus. On the other hand, thrombosis of the basilar artery in consequence of atheromatous degeneration, followed by softening in the pons, has been repeatedly observed. It is important to keep distinct from each other tumors of the pons and tumors exterior to the pons and situated at the base of the brain, although they may have many symptoms in common.

Fresh hemorrhages in the pons can be diagnosticated almost with certainty when *alternating* or *crossed paralysis* (that is, facial paralysis on the side of the lesion and paralysis of the extremities on the opposite side) can be made out; but in many cases, perhaps the majority, of fresh hemorrhage in the pons there are no characteristic symptoms present and the topical diagnosis is impossible. Often the symptoms are those of an ordinary apoplexy, such as follows hemorrhage in the internal capsule. When it is considered that through the pons pass the bundles of motor and of sensory fibres which connect the cerebrum with the spinal cord, that here are situated the roots of origin of the trigeminus, abducens, facial, and acoustic nerves, and taking into view the close relation of the pons to the medulla oblongata, it cannot occasion surprise to find that the symptoms of stationary lesions, such as of old hemorrhages and foci of softening, and particularly of slowly-progressive tumors, may present a variety and complexity somewhat diagnostic of affections of this part. It would be beyond the limits and scope of the present summary to enumerate the possible, or even many of the frequent, combinations of symptoms which may attend diseases of the pons. The most characteristic, and when occurring suddenly probably a pathognomonic, symptom is crossed paralysis (*paralysie alterne*). When this variety of paralysis is due to a stationary, non-encroaching lesion, this lesion will almost always be found in the lower half of the pons. In crossed paralysis, while the extremities are always paralyzed upon the side opposite to the lesion, there is paralysis of the facial, abducens, or hypoglossus nerves upon the side of the lesion. The facial paralysis of crossed hemiplegia resembles peripheral facial palsy; that is, all of the muscles supplied by the facial nerve (including the occipito-frontalis, corrugator supercilii, and orbicularis palpebrarum) are involved and the reaction of degeneration appears. Crossed hemianæsthesia may likewise occur, but less frequently than crossed hemiplegia.[1] It must not be inferred that the facial paralysis is necessarily on the side of the lesion in affections of the pons. It is sometimes on the same side as the body paralysis, in which case the hemiplegia is in no way characteristic of disease of the pons. In a remarkable case of tumor of the posterior portion of the pons, reported by Wernicke,[2] there existed paralysis only of cranial nerves, and not at all of the extremities. When crossed paralysis fails, it is from the grouping of the symptoms and by a careful analysis of individual symptoms that the diagnosis of a pons lesion is to be made, rather than by the presence of any one characteristic symptom. Bilateral paralysis is rare—in fact, much less frequently met with than was formerly assumed or would seem probable on theoretical grounds, if we except those extensive hemorrhages which lead to death during the stage of apoplectic coma.

4. *Medulla Oblongata.*—Hemorrhage into the medulla oblongata is not fre-

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[1] The manifold variations under which crossed paralyses of motion and of sensation may appear have been tabulated by Sigerson, *Dublin Med. Journ.*, Feb., 1878.

[2] *Arch. f. Psych.*, Bd. 7, p. 513.

quent. It leads almost invariably to death, either instantaneously or within a few hours, in consequence of injury to the centres of respiration and of circulation. Tumors occur in this situation, but they are rare, and their symptomatology is very imperfectly understood. The most interesting and carefully studied of the local diseases of the medulla oblongata is bulbar paralysis, the acute form of which is generally due to ischæmic softening or to inflammation, rarely to hemorrhage. Both the acute and the chronic forms of bulbar paralysis will be subsequently considered.

The nerves which originate in the medulla oblongata are the hypoglossal, the vagus, the spinal accessory, and the glosso-pharyngeal. The symptoms which are most characteristic of lesions of the medulla oblongata are difficulty of articulation, called dysarthria and anarthria, and difficulty in swallowing. Disturbances in the rhythm of respiration and of circulation are frequent. Lesions of the medulla oblongata are the only central lesions which can cause aphonia. Diabetes mellitus and diabetes insipidus have been occasionally observed to attend lesions (tumors) affecting the floor of the fourth ventricle. Lesions of the medulla can of course cause extensive paralysis of motion and of sensation, which may be either unilateral or bilateral according to the situation of the lesion, and which may exist without the coincidence of characteristic symptoms.

5. *Pedunculus Cerebri.*—Lesions confined to one of the peduncles are rare. Nevertheless, localized hemorrhages, foci of softening, and tumors, particularly tuberculous tumors, have been observed in this situation. It is, as a rule, only when the affections are situated in the basal portion of the peduncle near the pons that their effects are limited to this structure. The peduncles are composed essentially of centripetal and of centrifugal nerve-fibres connecting the cerebrum with the spinal cord. The motor fibres, constituting the so-called pyramidal tracts, occupy the middle third of the pes pedunculi; and the sensory fibres are situated in the tegmentum. The intimate relation between the inner border of the crus and the oculo-motor nerve is to be borne in mind. Characteristic of localized lesions of the crus cerebri are paralysis of motion and often of sensation of the side of the body opposite to the lesion, and paralysis of the third or oculo-motor nerve on the same side as the lesion; in other words, a variety of crossed paralysis. The symptoms of paralysis of the third nerve are ptosis, external strabismus, and dilatation of the pupil, often combined with double vision. It is only when the ocular paralysis and the crossed facial or body paralysis develop at the same time that it is safe to diagnosticate an affection of the crus. Lesions of the crus may produce simply hemiplegia or hemianæsthesia without ocular paralysis, in which case the topical diagnosis would be impossible.

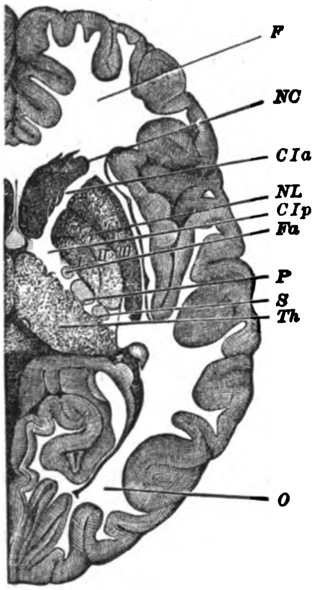
6. *Corpora Quadrigemina.*—Of the anatomical facts bearing upon the functions of these bodies, the most important is the termination of a part of the fibres of the optic tracts in the anterior pair or the nates. Furthermore, is to be considered the close relation between the terminations of the third and fourth cranial nerves and the corpora quadrigemina. Very little is known concerning symptoms referable to disease of the corpora quadrigemina. Lesions of the anterior pair lead to diminution of vision or to blindness. If the lesion be confined to one of the anterior pair, it is probable that hemianopsia is produced. Paralysis of the oculo-motorius, reflex immobility of the pupil, nystagmus, and disturbances of co-ordination resembling cerebellar ataxia have also been observed in lesions, particularly of the posterior pair and of the superior cerebellar peduncles. None of these symptoms, however, is characteristic of disease of the corpora quadrigemina, which, with our present knowledge, does not admit of positive diagnosis.

7. *Thalamus Opticus.*—Although the optic thalamus is very often involved

in hemorrhage, softening, and other affections of the internal capsule and the basal ganglia, it is rare that a lesion is confined exclusively to this body. Contrary to former views, it is now believed that lesions situated in the optic thalamus, their influence not extending beyond the seat of disease, do not produce paralysis either of motion or of sensation. The posterior part of the optic thalamus—namely, the pulvinar and the external geniculate body—receive fibres derived from the optic nerves, so that lesions of this part produce loss of vision, probably in the form of hemianopsia — a disturbance which, however, is not peculiar to lesions in this situation. Post-hemiplegic hemichorea has been repeatedly observed with lesions of the optic thalamus, but whether as a direct or an indirect symptom is uncertain. No symptoms which can render certain the topical diagnosis are as yet known to attend localized lesions of the optic thalamus.

8. *Corpus Striatum* (*nucleus caudatus, nucleus lenticularis, and capsula interna*).—This region is the most frequent seat of hemorrhage and of softening. It is important to be familiar with the anatomical relations of the parts here situated. The anatomy of this region is shown in Fig. 1, which represents a horizontal section through the right cerebral hemisphere. The corpus striatum is composed of the caudate nucleus (*N C* in Fig. 1) or intraventricular nucleus, the lenticular nucleus (*N L*) or extraventricular nucleus, and the anterior division of the internal capsule[1] (*C I a*). The caudate and the lenticular nuclei are masses of gray matter. The internal capsule is composed of white matter. The internal capsule is made up of an anterior division, situated between the caudate nucleus and the lenticular nucleus, and a posterior division, between the optic thalamus and the lenticular nucleus. The two divisions meet at a bend called the knee of the internal capsule. The destination of the fibres composing the anterior division of the internal capsule is not thoroughly understood. In the posterior division are situated the motor and the sensory tracts of nerve-fibres supplying the opposite half of the body. The motor tract forms a compact mass of nerve-fibres called the pyramidal tract (*P*), situated in the middle third of the posterior division of the internal capsule.[2] The fibres belonging to the facial nerve are probably situated in front of the pyramidal tract, nearer the knee of the capsule. The sensory tract occupies the posterior third of the posterior division of the internal capsule. The sensory tract includes both the nerves of ordinary sensation and the nerves of special sense.

FIG. 1.



Horizontal Section through the Right Cerebral Hemisphere (from Strümpell): *NC*, nucleus caudatus; *NL*, nucleus lenticularis; *Th*, thalamus opticus; *C I a*, anterior division of the capsula interna; *C I p*, posterior division of the capsula interna; *Fa*, fibres belonging to the facial nerve; *P*, pyramidal tract (motor); *S*, sensory tract; *O*, occipital lobe; *F*, frontal lobe.

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[1] Some authors include only the caudate nucleus in the corpus striatum.

[2] The exact situation of the pyramidal tracts in the internal capsule varies somewhat with the level of the section, these tracts being nearer the knee in the superior part and nearer the posterior extremity in the inferior part of the internal capsule.

The further course of the pyramidal tract is as follows: The tract is composed of motor fibres which originate in the motor cortical area—namely, the central convolutions and the paracentral lobule. (See Figs. 2 and 3, pp. 678, 679.) These fibres descend through the centrum ovale in the corona radiata, and are collected together into a compact mass in the middle third of the posterior division of the internal capsule. From the internal capsule the pyramidal tract is continued into the middle third of the pes pedunculi,[1] thence into the anterior half of the pons, where the fibres are scattered among the longitudinal bundles of the pons, and thence into the medulla oblongata, where they form the anterior pyramids. At the crossing of the pyramids a part of the pyramidal fibres usually remains uncrossed, and descends in the anterior columns of the spinal cord, while the larger part crosses and descends in the lateral columns of the cord.[2] (See Fig. 5, p. 729.) Here the pyramidal tracts terminate in the ganglion-cells of the anterior horns, whence the anterior nerve-roots originate. The pyramidal tracts, therefore, transmit voluntary motor impulses directly from the motor cortical area to the motor ganglion-cells in the anterior horns of the spinal cord. In the pyramidal tracts of the brain are also included fibres belonging to the motor cranial nerves.

Destruction of the pyramidal tract of one half of the brain causes hemiplegia of the other half of the body. It is evident that the lesion must be very large to destroy all of the pyramidal fibres in the centrum ovale, whereas a small lesion in the posterior division of the internal capsule will cut across the entire tract. For reasons which have been previously stated, the internal capsule and the adjacent ganglia are the most frequent seats of hemorrhage and of softening. Hence the symptoms which attend these lesions in this situation are usually taken as the basis of the clinical history of cerebral hemorrhage and of cerebral embolism.

When the lesion involves the anterior two-thirds of the posterior division of the internal capsule, there is ordinary hemiplegia; that is, paralysis of motion of the opposite extremities and paralysis of the lower branches of the opposite facial nerve. The hypoglossal nerve is paretic. Secondary contracture of the paralyzed muscles in most cases ensues when life is sufficiently prolonged. The muscles undergo only slight atrophy from disuse, and preserve their normal electrical reactions.

Lesions involving the posterior third of the posterior division of the internal capsule are much less common. They cause hemianæsthesia or paralysis of sensation of the opposite half of the body. This hemianæsthesia is often complete, including not only ordinary sensation, but the special senses of sight, hearing, smell, and taste. The special senses may be only partially or not at all involved. The affection of sight is probably in the form of hemianopsia. Post-hemiplegic hemichorea (which was first described by Weir Mitchell) and athetosis have been observed especially with lesions in this situation.

No symptoms have been referred directly to lesions confined to the anterior division of the internal capsule, to the caudate nucleus, or to the lenticular nucleus. Lesions of these parts, however, are almost invariably accompanied by hemiplegia, but this is referable to involvement by pressure or otherwise of the neighboring pyramidal tracts of the posterior division of the internal capsule. If the pyramidal tracts be not directly involved, the hemiplegia is often temporary, disappearing when the effects of pressure or of collateral œdema have been removed. The hemiplegia caused by destruction of the pyramidal tracts is permanent. The partial facial paralysis, however, seems to be an exception to this rule, as it is probable that one hemisphere may innervate the facial muscles of both sides.

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[1] Here, too, the situation varies somewhat with the height of the section, the tracts occupying the third quarter (reckoned from within outward) in the upper or anterior part of the peduncle.

[2] Sometimes all of the pyramidal fibres cross to the opposite lateral column. It is believed that very rarely the pyramidal fibres do not decussate at the crossing of the pyramids, but descend in that half of the spinal cord which corresponds to the hemisphere in which they originate. In the latter case it has been claimed that a lesion in the brain would produce hemiplegia on the same side as the lesion.

9. *Centrum Ovale.*—We know of no symptoms diagnostic of lesions situated in the centrum ovale of the cerebral hemispheres. Lesions involving the fibres of the corona radiata, which connect centres in the cortex with peripheral parts, cause the same symptoms as lesions of the cortical centres themselves. Hence lesions affecting the fibres descending from the motor cortical area, if extensive, cause hemiplegia; if of small extent, monoplegia of the opposite side. Lesions of fibres of the corona radiata coming from the visual area of the occipital lobe cause hemiopia. Lesions of fibres from the third frontal convolution or from the first temporal convolution of the left side produce aphasia. Lesions in the frontal lobe, and less frequently in the white substance of the other cerebral lobes, may give rise to no appreciable symptoms.

10. *Cortex Cerebri.*—Since the publication by Fritsch and Hitzig, in 1870, of their discovery of motor-centres in the cortex of the brain, much attention has been directed, and with fruitful result, to the clinical and pathological study of localized lesions of the cerebral cortex.

A. *Motor Area of the Cortex.*—In confirmation of physiological experiment, pathological observations have demonstrated that lesions of a certain region of the cerebral cortex are followed by motor disturbances. This region, designated as the motor cortical area, embraces the anterior central and the posterior central convolutions (also called respectively the ascending frontal and the ascending parietal convolutions) and the paracentral lobule. The results

FIG. 2.

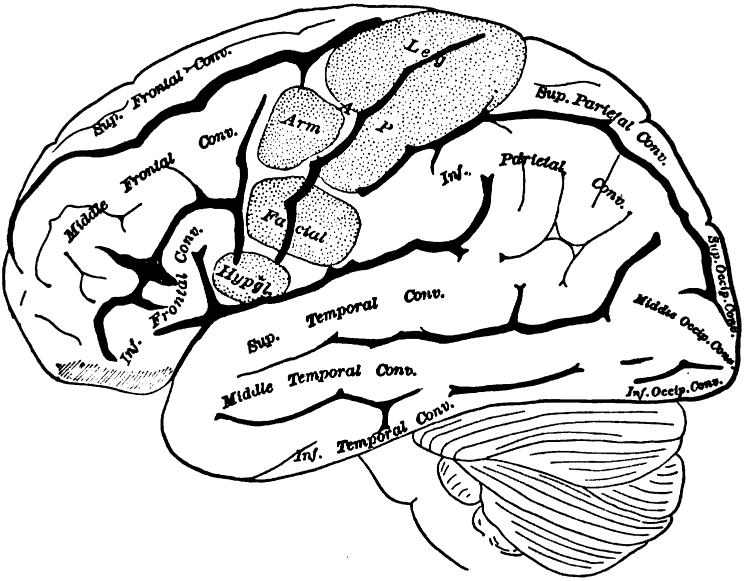

Diagram showing Position of Motor-centres in the Cortex (after Eichhorst).

of experiment, as well as the symptoms following small circumscribed lesions in the motor area, justify the recognition of separate motor centres in this part, the locations of which are represented in the accompanying diagrams (Figs. 2 and 3).

The centre for the movements of the lower facial muscles (those supplied by the buccal, oral, and nasal branches of the facial nerve) of the opposite side is situated in the lower third of the central convolutions. Near this, and probably somewhat lower, is the centre for the movements of the tongue (*Hypgl*, in Fig. 2). The centre for the movements of the opposite arm occupies the middle third of the anterior central convolution. The centre for the movements of the opposite lower extremity is situated in the upper part of the central convolutions, especially in the paracentral lobule. (See Figs. 2 and 3.)

FIG. 3.

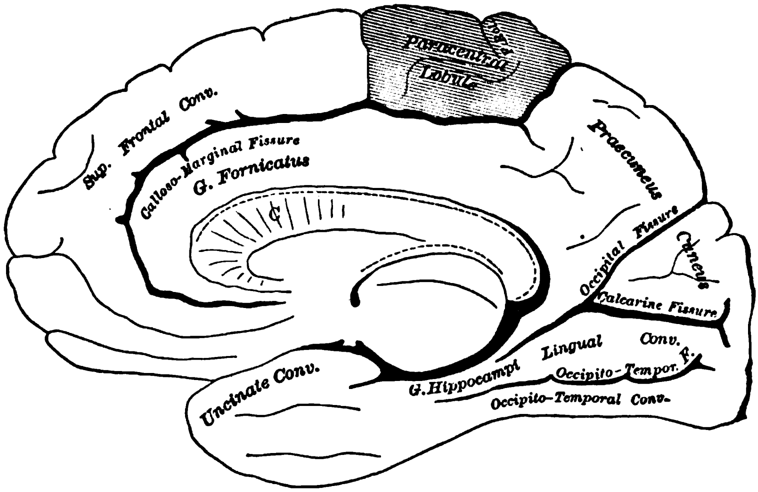

Diagram of the Median Surface of the Cerebral Hemisphere, showing the Paracentral Lobule (*Lbl. Paracentrl.*) (modified from Ecker). Abbreviations: *G*, gyrus; *F*, fissure; *C*, corpus callosum; *Conv*, convolution; *F. Rol*, fissure of Rolando; *Sup*, superior.

The motor cortical area receives its blood from the middle cerebral artery—a fact which explains the greater frequency of embolic and of thrombic softening in this district than in other parts of the cortex.

Lesions involving the entire motor area of the cortex are attended by the symptoms of an ordinary cerebral hemiplegia, and then, if the hemiplegia develop at once, it cannot usually be distinguished from lesions of the internal capsule or of the pyramidal tract in other situations. Such lesions in the cortex must be very extensive, and they are rare.

Less extensive lesions in the motor cortical area cause paralysis of only certain muscles of the opposite side. The term monoplegia is used to designate these localized paralyses. The following forms of monoplegia have been observed to attend localized cortical lesions: 1, oculo-motor monoplegia (isolated ptosis); 2, facial monoplegia, sometimes combined with paralysis of the hypoglossal nerve; 3, brachial monoplegia, or paralysis of the opposite arm; 4, crural monoplegia, or paralysis of the opposite leg; 5, brachio-facial monoplegia, or paralysis of the arm and face. It is evident, from the situation of the motor-centres, that while facial monoplegia and brachial monoplegia, and also brachial monoplegia and crural monoplegia, may be and often are combined, paralysis of the face and of the leg without involvement of the arm can scarcely be produced by a single cortical lesion, and in fact has never been observed. In progressive cortical lesions a paralysis at first confined to one arm or to the face may advance to total hemiplegia, the cortical localization being then characterized by the development of the hemiplegia out of successive monoplegias. Secondary contractures make their appearance almost invariably in the paralyzed muscles, but are of course not distinctive of cortical affections. As in other forms of cerebral paralysis, the electro-muscular reactions are normal or only quantitatively affected—a circumstance which distinguishes cortical monoplegias from most of those produced by lesions of the spinal cord and of peripheral nerves.

Of great value in the diagnosis of lesions of the motor cortical area are spasms affecting isolated groups of muscles (mono-spasms), such as spasms confined to the muscles of one side of the face or to the muscles of one of the opposite extremities. The spasms are both tonic and clonic, and they are sometimes designated as partial epilepsy of cortical origin. They may occur without paralysis, they may develop at the same time with paralysis, they may precede the paralysis, or they may attack muscles which have been paralyzed for a variable length of time. The latter two forms of development are especially characteristic of cortical lesions. Partial epilepsy of cortical origin is sometimes accompanied or followed by general epileptic convulsions, in which case the spasm begins regularly in the same group of muscles. In the most characteristic form of the disease consciousness is not lost at the onset of the paroxysm. The patient observes the progress of the convulsions from one group of muscles to another, and finally loses consciousness when the convulsions become general. When this variety of epilepsy is preceded by monoplegic paralysis, it may be considered as almost pathognomonic of a cortical lesion.

In most cases of cortical paralysis careful examination will show some sensory disturbances, but our knowledge of these is imperfect. It is believed that the nerves of ordinary and of muscular sensation terminate in the cortex of the motor area and in the cortex of the parietal lobe, but, thus far, it has not been possible to definitely fix centres for sensation.

B. *Cortical Centres for Speech*—These exist in the left inferior frontal (Broca's) convolution in its posterior part (or pars opercularis), and in the left superior temporal convolution. It is probable, although not definitely proven, that the left island of Reil also contains centres for speech. When the individual is left-handed or when the speech-centres of the left hemisphere are congenitally defective, the centres for speech may occupy the corresponding parts of the right hemisphere.

Ataxic or motor aphasia is due to difficulty in the co-ordination of the muscles used in speaking. It is caused by lesions of the left inferior frontal convolution. Amnesic aphasia, or loss of the memory of words, is probably the result of a lesion of the left superior temporal convolution. The disturbance of speech called by Wernicke sensory aphasia, and by Kussmaul word-deafness and word-blindness, has been observed also to follow lesions of the first temporal convolution of the left side. Sensory aphasia is characterized by inability to understand spoken or written words. On superficial observation the patients are thought to be deaf. The various forms of aphasia are often combined with each other.

C. *Visual Area of the Cortex.*—It has been demonstrated that the occipital convolutions contain centres for vision. Although not absolutely proven, the view is widely accepted, that the fibres of the optic nerve which supply the outer or temporal half of the retina do not cross in the optic chiasm, but terminate in the occipital lobe of the same side, while the fibres which supply the inner or nasal half of the retina cross in the optic chiasm and terminate in the occipital lobe of the opposite side. The accompanying diagram (Fig. 4) illustrates this view of the semi-decussation of the optic nerves.

Thus, the right occipital lobe contains the visual centres for the temporal half of the right retina and the nasal half of the left retina. Destruction of these centres in the right occipital lobe would therefore cause blindness of the temporal half of the right retina and of the nasal half of the left retina. In this case objects occupying the left half of the visual field would not be perceived. With an affection of the left occipital lobe of course the relations would be reversed. This form of disturbed vision is called *hemianopsia* or *hemiopia* (strictly, lateral homonymous hemianopsia). As is indicated in the diagram, the hemianopsia is limited by a vertical line passing through the point of fixation (macula lutea), and not by a line dividing the retina into two equal parts. It is thus evident that the defect of vision in hemianopsia is greater in the retina opposite to the lesion.[1] Hemianopsia is likely to be overlooked unless a special examination of the vision be made.

FIG. 4.

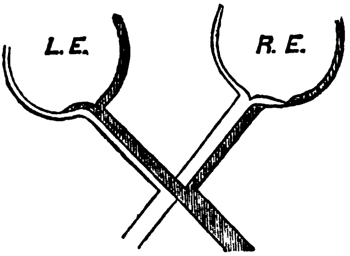

Diagram showing Semi-decussation of the Optic Nerves (after Landois).

Hemianopsia may be caused also by lesions of one optic tract, of the pulvinar and corpus geniculatum externum of the optic thalamus, of the posterior part of the internal capsule, of the medullary substance of the occipital lobe, and perhaps also by lesions of one of the anterior pair of the corpora quadrigemina.

Hemianopsia is the only symptom which has been directly referred to lesions of the occipital lobe. In many instances affections of the occipital lobe have given rise to no symptoms which attracted attention.

D. *Other Parts of the Cortex.*—Some physiological experiments render probable the existence of centres for hearing in the temporal lobe, but auditory disturbances other than the peculiar form called sensory aphasia are not positively known to result from cortical lesions.

Diseases of the frontal lobes are often latent. No distinctive symptoms have been assigned to lesions of the parietal lobes.

Meningeal Hemorrhage.

One of the most important varieties of meningeal hemorrhage is hæmatoma of the dura mater, due to pachymeningitis hæmorrhagica, under which head it will be subsequently considered.

The extravasated blood in meningeal hemorrhage may be seated as follows: 1, between the dura mater and the skull; 2, in the so-called arachnoid cavity; and 3, in the subarachnoid space or the meshes of the pia mater. When seated between the dura and the skull the most frequent cause is traumatism. In the majority of cases of traumatic meningeal hemorrhage, but not in all, there is fracture of the skull. The hemorrhage resulting from traumatism may be in any of the three situations mentioned, as well as in the substance of the brain. It may be most abundant at the base or on the convexity. The study of this important variety of meningeal hemorrhage belongs to the domain of surgery. Another cause of meningeal hemorrhage is the rupture of aneurisms of the arteries of the base, most frequently of the basilar or the middle cerebral. The lodgment of calcareous emboli in the cerebral arteries may lead to aneurism and hemorrhage. The occasional occurrence of meningeal hemorrhage in consequence of thrombosis of the sinuses, and in consequence of an intracerebral hemorrhage bursting through the peripheral brain-substance, has already been mentioned. In a case given in a former edition of this work the hemorrhage was attributable to wounding of a vessel by needle-shaped spiculæ of bone in the dura mater, without any injury of the skull. Hemorrhages, usually in the form of ecchymoses, are often observed in the meninges in infectious diseases. Larger hemorrhages occur in purpura and other diseases with a hemorrhagic diathesis. Meningeal hemorrhage has been known to follow a paroxysm in whooping cough, to occur during Bright's disease, and rarely under other conditions when no especial cause could be assigned. Meningeal hemorrhage is common in new-born infants, in whom it is sometimes, but not always, attributable to mechanical causes attending labor.

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[1] Hemianopsia is designated as right-sided or left-sided with reference to the defect in the visual field. Hence with lesions of the right occipital lobe, left hemianopsia occurs.

Large meningeal hemorrhages are always fatal; but small hemorrhages may be absorbed, leaving only a pigmented spot behind.

Meningeal hemorrhage, if the extravasation be considerable and rapid, gives rise to sudden coma or apoplexy, which is very rarely accompanied by hemiplegia. It may destroy life speedily. In a case reported in a previous edition of this work death took place two hours after the apoplectic seizure. Cases which prove fatal in the condition of coma cannot be differentiated by means of the symptomatology from cases of cerebral embolism or cerebral hemorrhage. The diagnosis is rendered probable by a traumatic causation. From congestive apoplexy it is distinguished by the absence of the symptoms which denote determination of blood to the head.

In the case referred to, in which the hemorrhage was caused by spiculæ of bone, epileptiform convulsions occurred. Symptoms of irritation, such as convulsions, are more frequent in meningeal than in cerebral hemorrhage. If the hemorrhage be not large it may give rise to no symptoms pointing to a grave affection. In the case of Daniel Webster the autopsy showed that slight meningeal hemorrhage had occurred some time prior to his fatal illness. The hemorrhage was connected with an injury which had not incapacitated him from subsequent intellectual efforts in which was manifested no diminution of his mental powers.

It follows, from what has just been stated in regard to the symptomatology and diagnosis, that the treatment is to be governed by symptomatic indications.

Insolation, or Sunstroke.

The terms *insolation* and *sunstroke* are applied to an affection—or perhaps more properly different affections—occasioned not exclusively by exposure to the sun's rays, as the terms signify, but by the action of great heat combined generally with other causative circumstances. The terms denote a sudden attack in extremely hot weather after exposure to the direct rays of the sun or to great heat, the attack characterized by loss of consciousness, followed frequently by stertor and convulsions, and death taking place, in a considerable proportion of cases, within the space of a few minutes or a few hours. Now, in a certain proportion of these cases the morbid condition, as judged by the symptoms, is expressed by the term exhaustion. The vital powers are prostrated, the forces carrying on the circulation give way, and death takes place by syncope. In another class of cases the affection apparently is similar to congestive apoplexy. In still another class of cases the symptoms show a combination of apoplexy and exhaustion. The cases last referred to probably constitute the majority of the cases of so-called insolation or sunstroke. In other cases the condition is referable to the abnormally high temperature of the blood. Other names than insolation and sunstroke have been proposed—namely, solar asphyxia, heat-apoplexy, thermal or heat fever, etc.

Morbid appearances found after death are as follows: The blood is dark and imperfectly coagulated, often fluid, and collected in the large veins. The heart is usually firmly contracted, and the left ventricle is empty, the right containing a small amount of dark fluid or partly clotted blood. Ecchymoses, especially subserous hemorrhages, are, as a rule, present. Extravasations of blood into the sympathetic ganglia of the neck, about the vagus in the neck, and in the connective tissue are sometimes met with. The lungs are deeply congested; sometimes they contain hemorrhages, and they are often œdematous. A moderate amount of serous transudation into the pleural and pericardial sacs and into the ventricles of the brain is not uncommon. The spleen is sometimes swollen and soft. Parenchymatous degeneration or cloudy swelling of the kidney, liver, spleen, and heart-muscle has been described as a lesion of insolation. The larger veins of the pia and dura mater are usually distended; and injection of the smaller vessels of the membranes and of the cerebrum is occasionally but not constantly found. The consistence of the brain and cord is sometimes notably soft.

The premonitions are slight or wanting. Dr. Swift, in a report based on the observation of 60 cases in the New York Hospital, gives the following account of the attack: "The patients are suddenly seized, while in the performance of their labors, with pain in the head and a sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially of the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appear of uniform color. In a great majority of cases this was, so far as could be ascertained, blue or purple. In one instance everything appeared red, in another green, and in another white."[1] It has been stated that irritability of the bladder precedes the attack. Pain in the head, although generally present, does not always precede unconsciousness. It was wanting in the case of a medical friend of the author who had a severe attack accompanied by violent convulsions.

The attack may consist of only a transient insensibility; but in severe cases the patient passes quickly into a comatose state. It is in this state that cases come under medical observation. The pupils are in some cases dilated, in some contracted, and sometimes the size is normal, but the respondency to light is lessened or lost. There may be contraction and dilatation at different periods in the same case. The pulse may be at first infrequent and full, but it often becomes frequent and feeble toward the fatal termination. I have observed death to take place purely by apnœa, the pulse having considerable force until after the cessation of breathing. The respirations in some cases are stertorous, and in other cases suspirious and accompanied by moaning. They are increased in frequency in the majority of cases. In most cases the temperature of the body is notably raised. The maximum rise in cases at Bellevue Hospital, in July, 1868, was 109½° F., and the lowest observed was 99½°. In a case observed in July, 1872, the temperature in the axilla was 110¼°. If the thermometer indicate a rise to 107° or more, a fatal termination is to be expected. In the case, however, just referred to, in which the temperature was 110½°, the patient recovered. Convulsions and rigidity of the muscles are of frequent occurrence. Vomiting frequently occurs, and toward the close of life there are involuntary evacuations from the bowels. The prognosis is rendered extremely unfavorable by the occurrence of vomiting and purging. The duration varies from a few minutes to several hours. The average duration in the fatal cases observed by Swift was four hours. If the patient emerge from the comatose state, convalescence is usually speedy. Of 20 cases observed by Pepper, in 3 insanity followed. This sequel did not once occur in more than 100 cases received at the New York Hospital. Temporary delirium succeeding the attack is occasionally observed. Paralysis is very rarely a concomitant or a sequel. I have, however, known of the occurrence of incomplete transient hemiplegia in one case after recovery from the attack, and in one case before recovery. In the latter case it lasted but a few hours.

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[1] "Observations on Exhaustion from the Effects of Heat," by H. S. Swift, Resident Physician of the New York Hospital, *N. Y. Journal of Medicine*, July, 1854.

As already stated, the pathological character of the affection in all cases of insolation is not uniform. Instances of sudden death from exhaustion or syncope produced by heat and over-exertion are reckoned among cases in which the morbid condition is quite different. In a certain proportion of cases the appearances after death, the character of the attack, and all the symptoms denote congestive apoplexy. In other cases, which probably constitute the majority, the pathological character is mixed; an apoplectic condition being combined with exhaustion. It is highly important to take cognizance of these differences as regards pathological character in treating the different cases which are grouped together under the name of insolation. The notably increased temperature of the body probably stands in a causative relation to certain of the phenomena. Regarded from this point of view, insolation is in a marked degree a febrile disease. That toxical changes in the blood play an important part in the pathology is probable. Vallin of Paris has reported a series of observations upon dogs and rabbits destroyed by insolation. These animals, placed upon the back, fastened in this position, and exposed to the direct rays of the sun in the months of July and August in the climate of Paris, presented phenomena analogous to insolation in man, and they died generally after an exposure of from half an hour to two hours. Examinations after death showed hyperæmia of the brain and lungs, the cavities of the heart, especially the left ventricular cavity, being empty.. These experiments have been repeated by Prof. Horatio C. Wood. Vallin is led to attribute these conditions and death to coagulation of the liquid of the muscular tissue (*suc musculaire*, myosin) of the heart and diaphragm. He does not, however, apply this explanation to all the cases embraced under the name of insolation. In some cases he attributes the death to toxical changes in the blood, the action of the heart being arrested by a morbid agency operating through the pneumogastric nerve. In the latter cases the heart-cavities may be filled with blood.[1] The observations of Prof. Horatio C. Wood lead him to the conclusion that the contraction of the ventricles of the heart found after death, especially when autopsies are made within a short time after death, is in most cases of post-mortem, not of ante-mortem, occurrence.[2]

Excessive heat is the chief agency in causing insolation, but other circumstances co-operate in the causation. Muscular exertion, unduly great or prolonged, is a powerful auxiliary cause. Persons are generally attacked when engaged in labor, but there are exceptions to this rule. Dr. Swift states that of the cases which he observed a large proportion were attacked shortly after dinner. The concentration of innervation upon the processes of digestion, the distension of the stomach with food and liquids, the latter being frequently taken in large quantity, and perhaps, in some cases, the disengagement of gas from chemical changes arising from indigestion, will account for the fact stated by Swift. In most cases persons are attacked when exposed to the direct rays of the sun, but there are exceptions to this rule. Swift states that eleven patients were attacked on the same morning in the laundry of one of the largest hotels in this city, and several cases were brought to the hospital from a sugar-refinery. Soldiers are not infrequently attacked after they have retired to their tents.

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[1] Vide *Archives de Médecine*, Feb., 1870.

[2] *On Thermic Fever, or Sunstroke*, Boylston Prize Essay, 1872.

Cases are more frequent by far in tropical than in cold or temperate latitudes, and in the latter they occur during the heated term of summer, when the weather is unusually and continuously hot. Atmospheric influences other than heat have been supposed to be involved in the causation—namely, an unusual accumulation of electricity, dryness and rarefaction of the air, but facts showing the importance of these influences are wanting. The number of cases in the same place in different years varies greatly. According to Dickson, more persons died in Charleston, S. C., from sunstroke in 1824 than in any twelve other years; and in the city of New York in the summer of 1853 there were 260 cases reported, whereas in no previous year had there been more than 36 cases.[1]

The attack generally takes place at periods when the heat of the day is at its maximum. Of the 60 cases observed by Swift, 40 occurred between 11 A. M. and 4 P. M., 17 between 4 and 9 P. M., and 3 between 8 and 11 A. M.

The DIAGNOSIS is, in general, made without difficulty. The circumstances connected with the attack and the symptomatic phenomena are sufficiently distinctive. Apoplexy with extravasation of blood is to be distinguished by the existence of hemiplegia. Persons deeply intoxicated are not infrequently brought into hospitals as cases of sunstroke, but the characters of alcoholic coma suffice for its discrimination.

Exclusive of mild cases in which there is transient stupor or insensibility, the danger in cases of insolation is always very great. The death-rate ranges from 40 to 50 per cent. If, with deep coma, the breathing be stertorous, sighing, or moaning, the prognosis is extremely unfavorable. Great frequency and feebleness of the pulse, relaxation of the sphincters, tracheal râles, and complete immobility are forerunners of a fatal termination. Convulsions are extremely unfavorable, yet I have known recovery to take place after violent convulsions. In accordance with the difference in pathological character in different cases, the mode of dying is sometimes by rapid asthenia or syncope. This mode of dying is exemplified among the cases in which the death is notably sudden, taking place in the space of a few moments after the attack. The mode of dying in other cases is by apnœa, but in the larger proportion of cases by apnœa and asthenia combined.

The TREATMENT is to be adapted to the pathological character of the affection, as represented by the symptoms, in individual cases. Pursuing this course, therapeutical measures, so far from being the same, will be diametrically opposite in different cases. In cases of nervous exhaustion, the danger being of death by rapid asthenia or syncope, complete rest is of the first importance. The removal of patients to their homes or to hospitals in this condition may contribute in no small measure to a fatal result. Stimulants are to be administered by the mouth very cautiously in order not to excite vomiting. They may be given with less risk by the rectum. Alcoholic stimulants may be administered by enema or by hypodermic injection. The carbonate of ammonia is an effective heart-stimulant administered hypodermically. The oil of turpentine given per enema is recommended. All restraints of dress are to be removed. The patient should be kept in as cool and pure an atmosphere as possible. If the surface be hot and dry, sponging the body with spirit and water should be employed. These should constitute measures employed during the attack. Cathartics, emetics, bloodletting, and all depressing agencies are pernicious. The symptoms representing the condition which calls for the treatment just stated are—frequency and feebleness of the pulse, weakness of the heart-sounds, with absence of stertor and the embarrassment of breathing indicative of cerebral compression.

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[1] *Elements of Medicine*, 2d ed., 1859.

In well-marked apoplectic cases I believe bloodletting to be the measure especially indicated. The treatment called for is the same as in congestive apoplexy. The life of the patient may depend on the prompt employment of bloodletting. The bowels should be freely opened with croton oil. Cold should be applied to the head, either by means of the ice-cap or the douche. The head should be elevated, and everything constricting the chest or neck should be removed. Revulsive applications should be made to the extremities. The symptoms representing the condition calling for these measures are—fulness of the pulse, slowness of the respiration, with perhaps stertor, heat of the surface, congestion of the face, and throbbing of the carotids and of the temporal arteries.

The question as to the propriety of bloodletting in cases of insolation has given rise to much discussion and difference of opinion. The doctrine which I desire to inculcate is, that to abstract blood is vastly important in some and is destructive in other cases. Never to employ bloodletting or to employ it in all cases would be alike injudicious. It is, of course, for the judgment of the practitioner to discriminate between the cases in which bloodletting is called for and the cases in which it will do harm. I have noted several cases which apparently exemplify the importance of bloodletting.

In the majority of the cases of insolation symptoms denoting congestive apoplexy are combined in variable proportions with those denoting exhaustion. The importance of bloodletting in these cases is to be measured by the predominance of the apoplectic phenomena, and it is contraindicated if predominance of exhaustion be denoted by the frequency or the feebleness of the pulse. The success of the treatment will depend on the judgment of the practitioner in deciding whether bloodletting be called for or otherwise, and if it be indicated, in determining the amount of blood to be taken. Discrimination is also important in prescribing croton oil. If the danger be from exhaustion, an active purgative is not indicated. Cold to the head is important in proportion as the symptoms of cerebral congestion predominate. On the other hand, in the cases in which the circulation is notably feeble stimulant remedies by the mouth or rectum are called for. In all cases quietude is important. In the cases in which convulsions occur these may be excited by movements of the body. The most efficient measure having reference to convulsions is the inhalation of chloroform.

For several years considerable reliance in Bellevue Hospital has been placed on sponging the body freely with cold water in addition to ice to the head. In 1872 the application of cold water to the surface was carried out, under my direction, more efficiently than before, in the following manner: The patient was enveloped in a sheet wet with cold water, and placed on blankets covered with india-rubber cloth. With a sprinkling-pot the cold water was applied over the whole body at intervals of a few moments. This was kept up for a period ranging from half an hour to several hours, the continuance being determined by the effect on the temperature, pulse, and other symptoms. The temperature of the body was reduced more or less, and sometimes several degrees. The measure was repeated as often as the temperature again rose. If the pulse were feeble, stimulants were given more or less freely during the application of the water. The good effect of this plan of treatment has been marked in some cases. A patient admitted at 2 P. M., unconscious, pupils contracted, pulse 150, breathing stertorous, the axillary temperature 110½°, and the body cyanosed, was at once placed in the wet sheet and sprinkled. At half-past 4 P. M. the temperature in the axilla had fallen to 104¾°, the frequency of the pulse and the cyanosis persisting. He was taken out of the sheet, dry cups were applied to the chest, and at 5 o'clock the wet sheet was again applied, and continued until fifteen minutes after 7 P. M. The temperature then was 101⅛° and the pulse 120. At 9.45 P. M. the temperature fell to 100⅘°. At midnight he became conscious. Progressive improvement followed, ending in recovery. The only additional measures employed up to the restoration of consciousness were an enema containing castor oil with a few drops of croton oil, and the hypodermic injection of ten minims of the tincture of digitalis, the latter having reference to the frequency and feebleness of the action of the heart.

The employment of cold water in the manner just described was resorted to only in the cases in which there was a considerable increase of axillary temperature—104° and upward. When the temperature was less, sponging the body with cold water was employed. Observations since 1872 have confirmed the efficacy of this plan of treatment in cases characterized by notable increase of temperature. The same method of applying cold has also been found efficient in other affections in which antipyretic treatment is indicated.

Dry cups to the chest relieve symptoms dependent on pulmonary congestion—namely, embarrassed breathing and cyanosis. Applied over the spine, they are useful in relieving convulsions and muscular rigidity.

In some cases treated in Bellevue Hospital the Calabar bean has been given in doses of one grain by hypodermic injection, and repeated two or three times after intervals of from half an hour to an hour. This remedy was tried with reference to muscular rigidity and convulsive movements. It seemed to be useful, but the observations are too few to warrant any positive conclusions. Given in the dose just stated, it produced no marked effect upon the pupils.[1]

Hyperæmia and Anæmia of the Spinal Cord and its Meninges.

As regards hyperæmia and anæmia, the spinal cord and its meninges are to be considered together. These pathological conditions are probably never limited to either the meninges or the cord separately. In treating of spinal paralysis occurring without symptoms which denote either inflammation or lesions of structure—that is, functional paralysis—the question will arise as to the existence of one or the other of these conditions. Exclusive of this connection, it is difficult, with our present knowledge, to form an opinion as to the importance of these conditions. It is customary to refer certain symptoms to hyperæmia or anæmia of the cord on conjectural grounds. Our present knowledge does not lead us far beyond conjecture as regards the frequency of these affections, their causation, and their symptomatic phenomena. Both conditions are probably not infrequent. They are attributable to various causes, and it is not irrational to refer to these certain symptoms.

Hyperæmia here, as in other situations, may be active or passive. (Vide p. 26.) Active hyperæmia may be caused by prolonged muscular exercise and sexual abuse or excess. Collateral fluxion is supposed to be causative in certain cases of suppression of the menses, the cessation of bleeding from hemorrhoids, and prolonged exposure to cold. Passive hyperæmia is incident to stasis of the systemic venous or the portal system.

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[1] For a report of 55 cases treated in Bellevue Hospital, July and August, 1872, prepared, at my request, by Dr. Katzenbach, at that time house-physician, vide *New York Journal of Medicine*, Jan., 1873.

The SYMPTOMS referred to active hyperæmia are—lumbar pain and pain in the limbs, increased cutaneous sensibility, formications, muscular twitchings, and paresis of the muscles. On the other hand, numbness of the extremities and anæsthesia point to passive hyperæmia.

The DIAGNOSIS requires absence of fever and of the local symptoms which denote either spinal meningitis or myelitis. The existence of causative agencies is to be taken into account.

The condition is without danger, except that it may be a factor in the causation of hemorrhage.

Cupping over the spine, using dry or hot cups according to the local symptoms and the general condition of the patient, stimulating foot-baths, the application of cold to the back, and cathartics, are the therapeutical measures indicated. Ergot and belladonna are considered to be useful by causing contraction of the arteries. The patient should lie as much as possible on the side or face, and not on the back.

Spinal anæmia (ischæmia) is an effect of aortic obstruction above the point at which the lumbar arteries are given off (Stenson's experiment). Temporary paraplegia is produced by compression of the aorta above that point. A few cases of paraplegia due to anæmia of the lumbar part of the spinal cord in consequence of embolism or thrombosis of the abdominal aorta have been recorded. Spasm of the intraspinal arteries has been supposed to be a cause of anæmia of the cord, continuing long enough to lead to impairment of nutrition and giving rise to paraplegia. Doubtless the cord, like the brain and other organs, suffers functionally whenever the blood is notably impoverished. This local anæmic condition, which is incident to general anæmia, probably has a causative connection with neuralgia affecting the intercostal and other nerves, together with other effects pertaining to the so-called "spinal irritation." Exclusive of a conjectured causative connection with functional or so-called reflex paralysis, and of the participation of the spinal cord in the effects of anæmia as a blood lesion, the diagnosis of spinal anæmia is not warrantable in the present state of knowlege.

Spinal Hemorrhage.

Hemorrhage within the spinal canal, not dependent on injuries of the spine, is an exceedingly rare event. Its infrequency is in striking contrast to intracranial hemorrhage. Spinal hemorrhage may be meningeal (hæmatorrhachis), or it may be in the substance of the cord (hæmato-myelus). In cases of meningeal hemorrhage the extravasation may be between the periosteum and the dura mater, between the latter and the arachnoid membrane (in the so-called arachnoid cavity), or in the meshes of the pia mater. In cases of intracranial meningeal hemorrhage the blood may flow down into the spinal canal. Hemorrhage into the substance of the cord may be either punctate or in the form of a larger focus. It is most frequently situated in the gray matter, in which it may extend for some distance in a longitudinal direction. The same changes occur in the extravasated blood as in cerebral hemorrhage. Both medullary and meningeal hemorrhage may result from traumatism, such as fractures and dislocations of the vertebræ, blows upon the back, etc. Most of the cases formerly considered as spontaneous spinal apoplexies are cases of hemorrhage resulting from a previous myelitis, the vessels sharing in the softening process (hæmato-myelitis); but there are very rare cases which can hardly be interpreted otherwise than as primary hemorrhage into the cord. Aneurisms of the aorta, after having occasioned erosion of the vertebræ, sometimes burst into the spinal canal.

Meningeal spinal hemorrhage causes paralysis, as a rule, so suddenly that the patient falls. Consciousness is retained. Severe pain may be felt at or near the site of the hemorrhage. Pain is also felt in the extremities. The cutaneous sensibility becomes impaired, and abnormal sensations are referred to the skin. The paralysis and anæsthesia are rarely complete. If the hemorrhage take place in the lumbar region, the lower limbs are paralyzed, together with the bladder and rectum. If the hemorrhage be in the upper part of the spinal canal, in addition to paralysis of the lower extremities paralysis and anæsthesia are present in the upper limbs, and these symptoms may be accompanied by difficulty in breathing and swallowing. Cramps, spasms, and tremor often occur in the upper or lower extremities according to the situation at which the hemorrhage takes place.

The **SYMPTOMS** are due to excitation of the spinal cord and the paralyzing effect of pressure. According to the rapidity of the hemorrhage, they occur abruptly or are developed more or less slowly. Their degree is determined by the amount and situation of the hemorrhage.

If the symptoms follow an injury of the spinal column, violent muscular exertions, or severe convulsive movements, these etiological relations render the diagnosis probable. The diagnosis is substantiated by the exclusion of spinal meningitis and myelitis. These diseases are excluded by the sudden or rapid development of the symptoms and by the absence of fever. Secondary fever may be developed after the lapse of several days.

If the hemorrhage take place at the upper part of the spinal canal, and if the amount be considerable, it may cause speedy death by disturbance of respiration (apnœa). A large extravasation in a lower situation, giving rise to complete paraplegia, with cystitis and bed-sores, is often fatal after a duration more or less protracted. Recovery may be expected, except in cases of injury of the spinal cord by fracture or dislocation of vertebræ. The removal of the coagulated blood by absorption is, however, a slow process, and recovery takes place after a considerable period, the improvement being very gradual.

The **TREATMENT** at the time of the attack has for its object the arrest of the hemorrhage. Absolute rest and cold applied to the spine are the most important of the measures for this object. Bloodletting is to be employed under the conditions which render this measure advisable in other connections.

The subsequent treatment has reference to the relief of symptoms, the absorption of the extravasated blood, the prevention of muscular atrophy, and the restoration of the functional activity of the paralyzed muscles. Iodine, mercury, and electricity are recommended as sorbefacients, but their usefulness in this way is open to scepticism. The treatment to be addressed to the muscles is the same as in cases of paralysis caused by other spinal affections.

Hemorrhage into the substance of the cord is preceded by symptoms denoting myelitis in a certain proportion of cases. These are wanting if the hemorrhage be occasioned by injury of the spine or violent muscular exertions. In either case there is sudden paralysis without loss of consciousness, accompanied by a localized pain in the spine. The paralysis will involve only the lower extremities, or in addition the upper extremities and muscles of the trunk, according to the seat of the extravasation. The pain is temporary, and the symptoms of excitation—spasms, cramps, etc.—which characterize meningeal hemorrhage are wanting. Sensory paralysis below the seat of the hemorrhage is more or less marked, and it is often complete, as regards the sensibility to both tact and pain. The paralysis involves the bladder and rectum. Atrophy of the paralyzed muscles takes place rapidly. Bed-sores are produced speedily. Death may take place quickly by apnœa if the hemorrhage be into the upper portion of the cord. If the seat of the hemorrhage be in the lumbar region, the termination is generally fatal, after a shorter or longer duration, in consequence of sloughing of soft parts exposed to pressure, cystitis, etc.

A small clot in a lateral half of the upper part of the cord gives rise to spinal hemiplegia characterized by motor paralysis on the side of the lesion and sensory paralysis on the opposite side. This is very rare.

The points involved in the differentiation from meningeal hemorrhage have been mentioned. Myelitis with softening gives rise to paralysis more or less slowly. This affection is excluded if the paralysis occur suddenly without having been preceded by any symptoms of spinal disease. In hæmato-myelitis, however, the paralysis may develop with great rapidity; but myelitis generally precedes the hemorrhage if the latter be not produced traumatically. Sudden paralysis preceded by symptoms of myelitis renders hemorrhage probable. The affection generally known as the acute spinal paralysis of children (sometimes occurring in adults) is excluded by the absence of fever.

The objects of the TREATMENT are essentially the same as in cases of meningeal spinal hemorrhage.

CHAPTER II.

INFLAMMATORY DISEASES OF THE MENINGES OF THE BRAIN AND OF THE SPINAL CORD.—HYDROCEPHALUS.—HYDRORRHACHIS.

Pachymeningitis and Hæmatoma of the Dura Mater.—Simple Acute Cerebral Meningitis: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Chronic Cerebral Meningitis.—Tuberculous Meningitis.—Acute Spinal Meningitis.—Chronic Spinal Meningitis.—Cervical Hypertrophic Meningitis.—Cerebro-spinal Meningitis: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Hydrocephalus.—Hydrorrhachis.—Syringo-myelus.

THE dura mater, which is composed of fibrous tissue, is the most external of the three membranes enveloping the brain. It consists of an external lamella, which serves as the internal periosteum of the cranium, and an internal lamella, the inner surface of which is lined with flat, polygonal endothelial cells. Most of the recent writers, discarding a parietal layer of the arachnoid, confine the name arachnoid membrane to that which was formerly described as its visceral layer. This is a thin connective-tissue membrane whose outer surface is smooth and covered with endothelium. The pia mater is the vascular membrane of the brain, the convolutions of which it follows and to which it closely adheres. It is connected with the arachnoid by interlacing bundles of connective tissue. The space between the arachnoid and the pia mater is known as the subarachnoid space, or sometimes as the meshes of the pia mater. In it is contained the cerebro-spinal fluid. The space between the dura mater and the arachnoid, which is usually found empty at autopsies, is called the subdural space, or, according to the former nomenclature, the arachnoid cavity. The subdural and the subarachnoid cavities are to be regarded as lymph-spaces.

By the term meningitis is usually understood inflammation of the pia mater or of the pia mater and arachnoid, it being impracticable to distinguish the inflammation of one of these membranes from that of the other. In fact, both are generally involved in meningitis. Sometimes inflammation of the pia mater is denominated *leptomeningitis*, in distinction from *pachymeningitis*, which is inflammation of the dura mater.

Pachymeningitis—Hæmatoma of the Dura Mater.

Pachymeningitis is divided into *pachymeningitis externa* and *pachymeningitis interna*.

External pachymeningitis may be either acute or chronic. Acute pachymeningitis is always suppurative, and is chiefly of surgical interest. It follows injury and disease of the cranial bones. Purulent inflammation of the dura mater follows suppurative disease of the internal or middle ear (otitis interna or media), with consecutive caries of the petrous portion of the temporal bone. It is often combined with purulent thrombosis of the lateral or other sinuses of the dura mater. The purulent inflammation may extend to the pia mater and to the substance of the brain.

By chronic external pachymeningitis is understood the fibrous thickening of the dura mater, often combined with the formation of osteophytes, which occurs especially in old age. The thickened dura becomes adherent to the skull. This form of pachymeningitis is analogous to the ossifying periostitis of the long bones.

Internal hemorrhagic pachymeningitis is of greater medical interest. This affection is characterized by the formation of an adventitious membrane upon the inner surface of the dura mater, usually in circumscribed patches over the convexity, and either limited to one side or extending to both sides of the median line. At first the membrane is extremely thin, delicate, and hardly to be recognized, except as a reddish, yellow, or brown stain. If one of these membranes be stripped off and examined under the microscope, it is found to consist chiefly of capillary blood-vessels with wide lumina, often provided with varicosities and with thin embryonic walls. Between the vessels are a few round, stellate, or fusiform cells, but there is very little fibrillated connective tissue. A considerable number of extravasated red blood-corpuscles, which have escaped either by diapedesis or by rupture of the vessel-walls, and a greater or less quantity of blood-pigment in the form of reddish-brown granules or of ruby-red crystals of hæmatoidin, will also be found in these membranes. The newly-formed blood-vessels are in organic connection with the pre-existing vessels of the dura mater. In more advanced cases the new membrane is thicker and composed of several layers, of which the deeper—that is, those nearest the dura mater—resemble ordinary fibrillated connective tissue. In some cases extensive extravasations of blood occur from rupture of some of the thin-walled, newly-formed vessels, and then give rise to the symptoms of meningeal apoplexy. To the bloody tumors thus formed upon the inner surface of the dura mater the name hæmatoma is applied. Hemorrhages confined to the subdural space, and not of traumatic origin, in the great majority of cases are due to hemorrhagic pachymeningitis. This fact was first established by Virchow. Some recent writers advocate the view which prevailed before Virchow's investigations—namely, that the first stage in hemorrhagic pachymeningitis is an extravasation of blood upon the inner surface of the dura mater, and that the development of blood-vessels and of new connective tissue is secondary. It is, however, admitted that when the extravasation is abundant (hæmatoma, properly speaking), an organized membrane has in the majority of cases already been formed. The more usually accepted view is that in the majority of cases the development of new blood-vessels upon the inner surface of the dura mater antedates the extravasation.

The symptomatic phenomena embrace those proceeding from the chronic inflammation of the dura mater and those caused by pressure upon the surface of the brain. Cerebral symptoms denoting inflammation are—cephalalgia, which is often notably intense, vertigo, tinnitus aurium, vigilance, and disturbed sleep. Other symptoms are sensitiveness to light and, in a marked degree, contraction of the pupils. The hemorrhage may occur either rapidly or slowly. If rapid and considerable, it causes an apoplectic attack, which may prove speedily fatal, but in most cases the patient recovers. Successive apoplectic attacks occur after variable intervals. If the hemorrhage take place slowly, it causes somnolence which continues for twenty-four or thirty hours, the mind on awaking being more or less disordered. Successive periods of somnolence mark repeated, slow, and small or moderate hemorrhages. Contraction of the pupils persists, and if the hæmatoma be limited to one hemisphere, the contraction is greater on that side. At a later period, however, the contraction of the pupils may cease. The pulse is slow and irregular, becoming frequent in fatal cases before death. Paralysis of the facial muscles and hemiplegia may occur, limited to one side, or occurring first on one and afterward on the other side. Muscular contractions, twitchings, and convulsive movements more or less marked are liable to occur on one side or on both sides. The convulsions sometimes have an epileptiform character. Strabismus and ptosis are wanting. The mental faculties are impaired in a greater or less degree.

The clinical history has so much diversity in respect to the events which occur, the order of their occurrence, their duration, etc. that there are few reliable typical characteristics. As regards its course, the affection may be either acute or chronic. Moreover, its phenomena are often obscured by those of various other diseases with which it is often associated.

If death take place in a primary attack of apoplectic coma, a diagnostic guess may be based on the antecedent cerebral symptoms and certain etiological points. In the cases which have not this termination the diagnostic points pertaining to the clinical history are the occurrence of successive attacks of coma or sopor, lasting for many hours or even for days, and the occurrence of more or less of the symptoms which have been mentioned, these occurring in a way not significant of other cerebral affections. Diseases to be excluded are—simple cerebral meningitis, tuberculous meningitis, cerebral hemorrhage, embolism or thrombosis, and general cerebral paralysis. There are cases in which the affection gives rise to no morbid symptoms. The morbid condition is sometimes found after death when it had not been suspected during life.

Points relating to the etiology are of much account in the diagnosis. The affection rarely occurs at less than forty years of age, and of the different decades the largest proportion of cases is between seventy and eighty. It is rare in early life, but the percentage of cases is larger prior to ten than from ten to twenty years of age.[1] Men are affected much oftener than women. Persons in health are never affected, if traumatism be excepted. Of the various affections in connection with which it occurs intercurrently, those caused by alcoholism take precedence. Clinical observations show that it is a disease to which drunkards are especially liable, whatever may be the pathological explanation. It is incident to different chronic lesions of the brain, particularly to those belonging to general cerebral paralysis. In a considerable proportion of cases it is referable to injuries from blows, falls, etc. It occurs sometimes in diseases attended by a hemorrhagic diathesis, such as scurvy, purpura, leucocythæmia, and pernicious anæmia. Here doubtless the hemorrhage is primary.

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[1] Vide Huguenin, in *Ziemssen's Cyc.*, Am. ed. vol. xii.

The course of the disease varies from a day to a year or longer. Huguenin gives the following as the results of an analysis of all the carefully recorded cases which he could collect: The duration was from one day to five days in 34 per cent.;[1] from five to thirty days in 40 per cent.; from one month to six months in 18 per cent.; from six months to one year in 4 per cent.; and more than a year in 4 per cent.

The prognosis will depend on the extent of the hemorrhage if hæmatoma form. Patients have recovered who had presented symptoms supposed to be a sufficient warrant for the diagnosis. Coexisting diseases often contribute to a fatal ending.

Recovery is not to be expected as a result of any special medication. Impending danger from hemorrhage may be perhaps averted and death postponed by therapeutical measures. Measures are to be addressed to the inflammatory condition in proportion to the symptoms. Generally, cold applications to the head, counter-irritation to the neck, cathartics, and stimulating foot-baths suffice. Venesection or topical bleeding is rarely indicated. With reference to the limitation of hemorrhage, the bromides and ergot may be prescribed. It is doubtful if the iodide of potassium or mercury promotes absorption of the effused blood. Palliative remedies, according to symptomatic indications, and the analeptica, form part of the treatment.

Simple Acute Cerebral Meningitis.

The name acute cerebral meningitis denotes, conventionally, inflammation of the meninges of the brain exclusive of the dura mater; that is, inflammation affecting the arachnoid membrane and the pia mater. It is also called *leptomeningitis*. To distinguish it from tuberculous meningitis it is designated as simple meningitis. It is sometimes called acute meningitis of the convexity in consequence of its localization by preference upon this part.

ANATOMICAL CHARACTERS.—Inflammation here, as in other situations, is preceded and usually accompanied by hyperæmia. The inflammatory products are serum, fibrin, and pus in varying amount and in varying proportions. These products of inflammation are chiefly in the meshes of the pia mater (subarachnoid space), but in severe cases they may be present, to a certain extent, also upon the outer surface of the arachnoid (subdural space), and may extend into the cortical substance of the brain. The inflammation affects usually in the highest degree the pia mater covering the convexity of the hemispheres, but it may extend to the meninges of the base, or in rare instances be chiefly confined to the base. When death takes place soon after the onset of the inflammation, the exudation is found to consist chiefly of serum rendered somewhat turbid by the admixture of pus-cells. When death occurs later, as it does in the majority of cases, the exudation is more distinctly purulent. The greenish-yellow, opaque streaks of pus are found in greatest abundance in the furrows between the convolutions, especially in the track of the large veins of the pia mater. In severe cases of some duration a greenish-yellow layer may be found spread nearly uniformly over the convex surface of the brain. The exudation, examined microscopically, is found to consist of serum, pus-cells, fibrillated fibrin (sometimes in great, sometimes in small amount), together with a certain number of red blood-corpuscles. The amount of serum varies considerably in different cases. The pus-cells are doubtless emigrated white blood-corpuscles. Pus-cells are also found, sometimes in sufficient number to be visible to the naked eye, in the cortex cerebri, especially around the blood-vessels. Punctate hemorrhages are sometimes found both in the pia mater and in the cortex. The ventricles may contain a moderate quantity of serum, which is usually turbid, or they may be empty. It is to be borne in mind in examinations after death that increase in the amount of serum in the subarachnoid space is not evidence of inflammation; nor is mere opacity of the arachnoid to be mistaken for acute inflammation.

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[1] Of course, in these short durations the affection is not a hemorrhagic pachymeningitis, which is necessarily chronic, but it must be a simple hemorrhage on the inner surface of the dura mater, it being Huguenin's view that this hemorrhage precedes the pachymeningitis.

CLINICAL HISTORY.—Simple acute meningitis may be abrupt or gradual in its development. The premonitions are the symptoms of determination of blood or active hyperæmia. There is a marked variation in symptoms at different periods of the disease—namely, prior to and after a certain amount of sero-fibrinous exudation. The course is divisible into two stages, the division being based on the amount of the products of inflammation. The first stage extends to the period when, owing to the pressure of these products, a notable change in the symptoms occurs.

The first stage is called the stage of excitement. It is characterized by pain, usually intense, referred to the entire head, sometimes greatest in the anterior and sometimes in the posterior portion. The pain is the same as in active cerebral hyperæmia. Delirium frequently occurs, and the delirium in this stage is generally active or maniacal, or in some cases hilarious. Owing to the prominence of the delirium, patients are sometimes carried to insane institutions, the affection being mistaken for acute functional mania. Convulsions may occur, especially in young subjects. The senses of sight and hearing are abnormally acute. Light and sounds occasion distress and increase the cerebral excitement. There is photophobia, and the eyebrows are corrugated to shield the eyes from the light. The pupils are generally contracted. The face is flushed. The carotids and temporal arteries pulsate strongly. The head is hot. Vomiting generally occurs in this stage, and is often prominent as a symptom. The bowels are constipated. The urine is diminished in quantity, and is sometimes albuminous. The abdominal walls are depressed. Fever is more or less intense; the pulse is accelerated, strong, and full; and the axillary temperature may rise to or above 104° F. Exacerbations occur in which the delirium and cephalalgia are notably increased. The duration of this stage is short. It may continue but a few hours, and it rarely extends beyond two or three days.

The second stage is called the stage of oppression. The symptoms distinctive of this stage are those denoting pressure by the inflammatory products. The pain in the head is diminished. The morbid sensitiveness to light and sounds is lessened. The pupils may be dilated or contracted, and their respondency to light is diminished or lost. Strabismus and loss of vision may occur. Drowsiness succeeds the state of mental excitation. There is rigidity of the muscles of the back of the neck. The pulse is diminished in frequency, and is often irregular in force and rhythm. It becomes rapid and thready a few hours before death. The respirations are disordered in rhythm or suspirious. The Cheyne-Stokes respiration may be present. Paralysis may occur, limited to the face or one extremity or extending over one side of the body. If the progress of the disease be unfavorable, coma is at length induced, and continues until death. Convulsions may occur in the second as well as the first stage.

CAUSATION.—Although acute simple cerebral meningitis often presents itself clinically as a primary or idiopathic affection, the evidence of post-mortem examination shows that it can very rarely be considered as a primary disease. The majority of the cases which are not secondary and which occur sporadically are instances of epidemic cerebro-spinal meningitis. In these cases an examination of the membranes of the spinal cord will usually show that these are inflamed. It is in accordance with modern views to suppose that in all cases of purulent meningitis a special infective agent has gained access to the inflamed membrane. This agent is causative of most of the cases in which the disease is primary. Very frequently simple cerebral meningitis is secondary to some other disease. Secondary meningitis may be caused by infection from some disease of a part contiguous to the cerebral membranes, or from some remote disease, whence the agent of infection is transported to the brain by the blood or the lymph-current, or from some blood disease.

Acute cerebral meningitis may be referable to injuries of the head involving wounds of the soft parts or fracture of the skull. In not a few instances it is secondary to inflammation of the middle ear, with or without caries of the petrous portion of the temporal bone. In these instances an acute circumscribed pachymeningitis usually, but not always, precedes the affection of the pia mater. Caries of the cranium in any situation may give rise to meningitis, either circumscribed or more or less diffused. Purulent inflammations in the upper part of the nasal cavities and in the orbits may be followed by meningitis. Abscesses of the brain which reach the surface lead to purulent meningitis.

Various infectious diseases form an important group of causes. A certain proportion of cases of acute lobar pneumonia, especially in drunkards, are complicated with acute cerebral meningitis. To this group of causes belong also acute ulcerative endocarditis, pyæmia, septicæmia, erysipelas, the eruptive fevers, typhoid fever, typhus fever, Bright's disease, purulent pleurisy, and acute articular rheumatism. It is uncertain whether in these cases the meningitis be caused by the special virus of the disease or be due to some secondary infection. In the meningitis complicating lobar pneumonia the same organisms have been found in the exudation in the meninges as in the exudation in the lungs, so that here, at least, it is probable that the special cause of the pneumonia is also the cause of the meningitis. The weight of evidence is in favor of the view that acute cerebral meningitis may be caused by a variety of micro-organisms, including those of epidemic cerebro-spinal meningitis, the bacteria of suppuration, and the specific germs of many of the acute infectious diseases. The cerebral meninges are rendered more susceptible to the causes of acute purulent inflammation by the pre-existence of chronic meningitis, such as is often met with in drunkards, syphilitic patients, insane persons, and idiots.

Acute cerebral meningitis may occur at any age. It is more common in males than in females.

DIAGNOSIS.—Acute meningitis may be confounded with active cerebral congestion. If the inflammation go off by delitescence—that is, without passing into the second stage—it may be impossible to say after recovery whether the affection were meningitis or merely active congestion. In other words, more or less of the symptoms belonging to the second stage are required for a positive diagnosis.

Cases of typhoid fever characterized by early and active delirium have heretofore not infrequently been considered as cases of acute meningitis. The points involved in this differential diagnosis are the following: The access or slow development of the fever; the distinctive events belonging to it—namely, diarrhœa, meteorism or tympanites, tenderness in the iliac regions, together with absence of morbid acuteness of the senses, and continuance of the delirium without the supervention of the phenomena of the second stage of meningitis; also the eruption and the characteristic rise of temperature during the first week. These points show the disease to be a fever, and not meningitis. In the somnolence of typhoid or typhus fever patients manifest no opposition to being aroused, to having their position changed, etc.; whereas in meningitis the patient is irritable when disturbed, and resists, perhaps with violence, efforts to give nourishment, to examine the pupils by raising the eyelids, or to interrupt in any way the somnolent condition.

Functional mania may be erroneously considered as denoting acute meningitis. The discrimination is to be based on the absence of morbid acuteness of the senses, together with the absence of febrile movement, and the continuance of the mania without the symptoms of compression.

Other diseases are more likely to be considered as acute meningitis than this is to be mistaken for other affections. In view of the infrequency of primary cerebral meningitis the ability of a practitioner to distinguish it from other affections may be suspected if he believe that he has met with many cases of it.

PROGNOSIS.—Acute meningitis involves much danger. It may destroy life quickly. Cases have terminated fatally in thirty-six hours. As a rule, the duration in fatal cases does not extend beyond eight or nine days. The symptoms denoting an unfavorable prognosis are—profound coma, notable disorder of respiration, difficulty of deglutition, general immobility and anæsthesia, feebleness of the pulse, etc. Strabismus, hemiplegia, and convulsions may be present in cases which recover. A few well-marked cases, presenting these symptoms, which have fallen under my observation have ended in recovery. Data are wanting for determining the ratio of cases which end fatally, but it is certain that the fatal cases greatly preponderate. The association with acute articular rheumatism is considered as rendering the prognosis extremely unfavorable. I have, however, observed a well-marked case, the symptoms denoting spinal as well as cerebral meningitis, which ended in recovery.

In cases which prove rapidly fatal death may be due chiefly to apnœa, but if life be prolonged for several days the mode of dying is by apnœa and asthenia combined.

TREATMENT.—The treatment must have reference to the stage of the disease. The objects in the first stage are to lessen the determination of blood to the head and to limit the amount of exudation. If the heart act with abnormal power, bloodletting in this stage is to be employed. As a measure of depletion and revulsion active purgation is indicated. Cold is to be applied to the head, either by means of the ice-cap, the cold douche, or constant ablution. Cutting the hair close to the head renders these applications more efficient.

Bloodletting in this disease is indicated in view of the promptness of its operation and the short duration frequently of the first stage. Bearing in mind that the chief source of danger is from the products of inflammation, it is important that the measures employed with the hope of limiting these should be resorted to more promptly and vigorously than in other acute inflammations—for example, pneumonitis—in which the products of inflammation generally do not give rise to serious results. Moreover, there is not the same necessity for guarding the vital forces as in other acute inflammations; for in this disease there is danger that life may be quickly destroyed by apnœa caused by the pressure of effused serum and lymph, whereas in most other acute inflammations the danger is that they will destroy life by asthenia. It is not, however, to be forgotten that asthenia enters into the mode of dying in cases of meningitis which do not pursue a rapid career, and hence blood-letting and other measures of depletion may be pushed too far. These measures are indicated only in the first stage of the disease. Perfect quietude, exclusion of light, elevation of the head, and the avoidance of everything which may tend to increase cerebral excitement by acting on the senses or the mind form an important part of the treatment of this stage. The bromides are useful for tranquillizing intense cerebral excitation. If these do not prove effective, codeia may be given or a salt of morphia, the latter administered hypodermically. Sinapisms to the neck and stimulating pediluvia have a certain measure of utility. Vomiting calls for palliative measures. The diet in this stage should be restricted to the blandest articles taken cold.

In the second stage the great object of treatment is to promote absorption of the morbid products, serum and lymph. Depletion is no longer admissible. Mercurialization is advised for this object, and although the efficiency of mercury as a sorbefacient has doubtless been much exaggerated, we are not warranted in saying that it has no effect in that way. If it exert never so small an effect, it is indicated in a disease like this, in which the object to be effected is of so much importance. Iodine is another remedy supposed to be useful as a sorbefacient. I have witnessed, as I think, the good effect of the iodide of potassium in several cases. Patients under my observation have recovered under the use of this remedy, mercury not having been employed. It may be given in doses of from five to ten grains four or five times daily, increasing the doses, if improvement do not take place, up to the limit of tolerance. A blister may be applied over the nucha. The propriety of blistering the scalp is questionable.

Another object of treatment in the second stage, if death do not take place by apnœa, is to support the powers of the system. Measures for this object are indicated in proportion as the tendency to death is by asthenia.

Attention to the bladder is important in the second stage.

Chronic Cerebral Meningitis.

Simple chronic cerebral meningitis—that is, meningitis not incident to a tuberculous or any prior cerebral affection—is infrequent, although it is less rare than the acute form of the disease. The statement has reference to periods of life after infancy, but it holds good also with respect to the latter period. In a great majority of cases it is a subacute affection *ab initio*.

The ANATOMICAL CHARACTERS are thickening and opacity of the pia mater and arachnoid caused by a new formation of connective tissue. The inflammation is usually circumscribed. The thickened and opaque membranes are abnormally adherent to the brain, and frequently adhesions of fibrous tissue are formed between the dura mater and the arachnoid. There may be considerable effusion into the ventricles. A certain degree of opacity of the pia-arachnoid, especially marked along the longitudinal fissure, combined with increase in the number of Pacchionian granulations, is to regarded as a normal condition in old age.

This affection may be ranked among the "insidious diseases." The SYMP-

TOMS may not point to any cerebral affection or they are not distinctive of this form of disease. The diagnosis is difficult. Pain in the head is usually more or less prominent as a symptom, but this is common to many affections. There may be little or no fever. Vomiting may be a prominent symptom, but this too occurs in various pathological connections. Mental apathy, amounting sometimes to stupidity, indisposition to either mental or physical exertion, and a notable change in disposition, characterize certain cases. Paralysis of the face and hemiplegia sometimes occur, but they are effects of other forms of disease. This statement also applies to epileptiform convulsions.

I have known a case of chronic meningitis to be considered as a case of chronic gastritis, vomiting being a prominent and persistent symptom. The patient fell into coma before death, but this was attributed to exhaustion. Meningitis was not suspected prior to the autopsy. In another case in which an ante-mortem diagnosis was not made, the patient dying suddenly with apoplexy from extravasation, the case had been considered as one of latent or irregular intermittent fever, chills occurring from time to time. A hospital patient had no symptoms referable to the head except mental inertia bordering on imbecility. He gradually fell into coma, and after death the case was ascertained to be one of chronic meningitis.

As regards DIAGNOSIS, close observation ought to lead to the conclusion that there exists a chronic cerebral affection. Paralysis is less likely to occur than in cases of tumors within the skull. The absence of paralysis is therefore of some account in the diagnosis. This statement will measurably apply to convulsions. If cephalagia, impairment of the mental faculties, and gradually-developed coma follow an injury of the head, chronic meningitis is probable. A traumatic cause is likely to give rise to inflammation. Chronic meningitis is developed in a small proportion of cases of the affections of the kidneys embraced under the name Bright's disease. The existence of renal disease is therefore of some account in the diagnosis; but in cases of renal disease cerebral symptoms referable to uræmia—namely, epileptiform convulsions and coma—are not to to be considered as evidence of meningitis. The existence of syphilis is to be ascertained, inasmuch as this is a cause of chronic meningitis.

The TREATMENT consists of moderate counter-irritation, the iodide of potassium, and perhaps mercurialization, quietude of mind and body, and the hygienic regulations appropriate to chronic affections generally.

Tuberculous Meningitis.

The distinctive feature of this variety is the presence of miliary tubercles in the pia mater in connection with meningeal inflammation. Hence the name *tuberculous* or *tubercular meningitis.* As the inflammation, in the majority of cases, is chiefly at the base of the brain, it is called by some authors *basilar meningitis;* but this name is not to be recommended, inasmuch as meningitis at the base is not necessarily tuberculous. An old name for this affection is *acute hydrocephalus*, as there is usually found a considerable fluid accumulation in the ventricles. Tuberculous meningitis affects children more frequently than adults, and is treated of fully in works devoted to infantile diseases. It is of rare occurrence in the first year of life, occurring especially after two and before seven years of age. It is not, however, confined to childhood.

ANATOMICAL CHARACTERS.—Tuberculous meningitis attacks by preference the pia mater covering the fossa Sylvii, the optic chiasm, the interpeduncular space, and the under surface of the cerebellum; but it is by no means confined to these situations, being often found on the convexity of the brain, although, as a rule, in a less degree than at the base. Tubercles may be present also in the choroid plexuses, and even in the substance of the brain. The tubercles appear as grayish-white or yellowish granulations in the course of the small arteries of the pia mater. They are usually of about the size of a millet-seed, but nodules the size of a pea may be formed by the confluence of many granulations. Some of the tubercles are so small as to be recognizable only by the aid of the microscope. While usually found without difficulty, they may be present in such a small number as to require careful search for their detection, and on the other hand their number may amount to thousands. A sero-fibrinous or a fibrino-purulent exudation is found, as a rule, at the base, and may be present upon the convexity of the brain. The amount of exudation varies much in different cases. Although there is reason to believe that tubercles precede the inflammatory exudation, there seems to be no necessary connection between the number of tubercles and the degree and extent of inflammation. Tubercles may be present in large number without any apparent inflammation, and, on the other hand, the inflammatory exudation may be abundant without the coincidence of many tubercles. It can serve, however, no useful purpose, from a clinical standpoint, to distinguish between miliary tuberculosis of the meninges (without apparent inflammation) and tuberculous meningitis, inasmuch as the former usually produces the same symptoms as the latter, and, in fact, is accompanied not infrequently by the microscopical evidences of inflammation. In the majority of cases of tuberculous meningitis (in 75 per cent., according to Seitz), the ventricles are found dilated and containing a large quantity of serous effusion, either clear or moderately turbid from pus-cells. When the ventricular effusion is abundant the surrounding brain-substance is often found greatly softened and sometimes almost diffluent. It is probable that this softening is cadaveric and without clinical significance. In proportion to the amount of fluid effusion in the ventricles the convolutions are flattened and the sulci shallow. Minute extravasations of blood are frequently found in the pia mater, and sometimes in the substance of the brain.

The microscopical examination of the tubercles shows that they consist usually of a nodular accumulation, not always distinctly circumscribed, of lymphoid and endothelial cells in the walls and perivascular sheaths of the small arteries of the pia mater, especially where they divide. The lumen of the artery will often be found compressed and thrombosed at the seat of tuberculous infiltration. The inflammatory products are serum, fibrin, and pus.

Tuberculous meningitis is due to the invasion of the blood-vessels of the meninges by tubercle bacilli. Sometimes, especially in children, the affection of the meninges is the sole manifestation of the tuberculous infection, but more frequently, and almost without exception in adults, the meningeal tuberculosis is secondary to tuberculosis of some other part of the body. In children the primary tuberculosis is often of the cervical, bronchial, or mesenteric lymph-glands; in adults it is usually of the lungs; but the primary tuberculous focus may be in any part of the body subject to tuberculous infection, as in the genito-urinary apparatus, the serous membranes, the vertebræ, etc. Although it is probable that in these cases the tubercle bacilli are carried from the primary focus by the blood-current, it is a curious fact that the meninges are often involved without other evidence of the invasion of the blood by tubercle bacilli. Still, in not a few cases general miliary tuberculosis is likewise present. In cases of primary tuberculous meningitis it is not understood how the tubercle bacilli reach the meninges. In a few of the apparently primary cases there will be found tuberculous disease of the middle ear. It has been suggested that sometimes the tubercle bacilli reach the cerebral meninges through the ethmoidal foramina from the upper nasal passages.

Tuberculous inflammation of the cerebral meninges is nearly always accompanied by a similar affection of the spinal meninges, so that the disease is in reality a tuberculous cerebro-spinal meningitis. The affection of the spinal meninges is usually less intense than that of the cerebral meninges, but it may give rise to characteristic symptoms.

CLINICAL HISTORY.—In the progress of this disease, as in simple meningitis, the symptoms undergo a marked change due to the pressure of the exudation and effused liquid; and this change will serve as the basis of a division into two stages. The development of the meningitis may be preceded by the symptoms and signs of pulmonary tuberculosis, or, less frequently, of tuberculosis in other situations, as in the lymphatic glands, serous membranes, and the genito-urinary apparatus. The meningeal affection, even when it does not distinctly supervene upon tuberculous disease of the lungs, is usually preceded by pallor, loss of weight, impaired appetite, irritability of temper, muscular debility, indisposition to exertion, and other general symptoms for a variable period; but in a certain proportion of cases the symptoms denoting the head affection appear suddenly, the previous health having been apparently good. When the meningitis occurs after a prodromic period extending, as it does in some cases, over several weeks and even months, during which the general health becomes considerably impaired, an antecedent tuberculous affection in other organs is to be inferred, or it may be that the miliary granulations within the skull existed for some time prior to the development of meningeal inflammation. The latter is probable if the prodromic period have been marked by cerebral symptoms. On the other hand, the meningitis, when it is developed abruptly—that is, without a prodromic period, the previous health having been apparently good—is usually incident to a primary tuberculous infection of the meninges.

The development of meningitis, as a rule, is denoted by pain in the head, referred generally to the forehead. Convulsions are less likely to occur at the onset than in simple acute meningitis. Vomiting in most cases is an early and sometimes a prominent symptom. More or less fever occurs. The axillary temperature is raised, but the rise is less than in simple acute meningitis. Notable diurnal remissions are likely to occur, the exacerbations taking place at night. These symptoms continue for several days. The cephalalgia is frequently severe. Lancinating pains occur from time to time, causing infants to give a sudden, sharp, and short cry, which has been known as the *cephalic cry*. A common exclamation is, "Oh my head!" The hands are often carried to the head. There is increased sensibility to light and sound. The expression is frowning. In children raising the lids and exposing the eyes to the light occasion crying. The conjunctiva is often suffused. The pupils in this stage are frequently contracted. The face may be flushed, and in some cases paroxysmal flushing of the face is a notable symptom. I have observed this to precede convulsions. During this stage vigilance is frequently a prominent symptom, and the sleep obtained is disturbed by dreams causing patients to awake suddenly in terror. It is important to note that vomiting and increased sensibility to light, although generally marked, may be wanting.

Drowsiness and somnolence mark the supervention of the second stage. There is now less suffering from cephalalgia, but occasional lancinating pains may still occur. The pulse may fall below the average frequency of health. It is not infrequently faltering. It fluctuates in frequency and may be irregular in rhythm. The respirations, which in the first stage may have been accompanied by sighing, are now often irregular in rhythm and may present the Cheyne-Stokes phenomena. In young children masticatory movements are common, and after dentition grinding of the teeth. Rolling the head or boring the pillow is a symptom in infants. The somnolency increases. The patients are reluctant to be aroused. If able to speak, they reply to questions slowly or in monosyllables, like a person half awakened from profound sleep. When aroused they have an expression of stupidity. Incoherence is common, and occasionally drowsiness alternates with paroxysms of delirium. The pupils become dilated, and not infrequently present disparity as regards size. They respond sluggishly to light. As the somnolency deepens the movements of the eyeballs are not in unison, strabismus frequently occurs, and oscillations of the balls are occasionally observed. The eyelids are but partially closed, the eyeballs being raised so that the cornea is partially covered by the upper lid, and in some cases the patients lie with the lids widely opened. Vision is gradually impaired, and frequently is lost. Various ophthalmoscopic appearances have been described, of which the most common are neuro-retinitis and a swelling of the optic papilla, called choked disc. Subsultus, carphologia, and fumbling with the bed-clothes are frequent symptoms. Convulsions frequently occur, especially in children. They are either limited or general, and they may be either severe or slight. Paralysis of one side of the face and incomplete hemiplegia are occasionally observed. Not infrequently the muscles of the posterior part of the neck become rigid, and the patient lies with the head drawn backward. Contraction of certain of the muscles of the limbs occurs in some cases. This is sometimes observed on one side, while the muscles of the opposite side are relaxed. Occasional vomiting is a symptom of the second stage. As a rule, there is complete anorexia, but in some cases which I have observed food has been taken with apparent relish. The teeth and lips often become covered with sordes. Constipation is the rule. In young children the dejections are sometimes greenish and resemble spinach in appearance. The abdomen is retracted. Retention of urine is not uncommon.

Sooner or later the patient becomes comatose; but in some cases before the occurrence of coma remissions occur, in which the improvement as regards intelligence and in other respects is such as to appear to the friends to afford ground for much encouragement in hoping for recovery. The occurrence of profound coma is followed by impairment and loss of deglutition, with more or less embarrassment of respiration. The pulse becomes frequent and feeble, and the mode of dying is by apnœa and asthenia combined.

The duration of the disease is variable. It rarely is less than a week. In the majority of cases it does not exceed two weeks. It rarely extends beyond three weeks, but exceptionally it may be prolonged considerably beyond this period. The termination is almost invariably fatal. In some of the cases of supposed recovery it is probable that the affection was simple meningitis.

DIAGNOSIS.—The diagnosis involves the discrimination from an essential fever. With the latter it is not infrequently confounded. In making this differential diagnosis, meningitis is to be excluded by the absence of sensibility to light, of the characteristic lancinating pains which belong to the first stage, and of the strabismus, affection of the pupils, and other symptoms belonging to the second stage. In fever, drowsiness and delirium are not preceded and accompanied by the symptoms just referred to. Moreover, in typhoid fever, diarrhœa, abdominal tympanites, and iliac tenderness are likely to be present—symptoms not belonging to the clinical history of tuberculous meningitis.

It is to be discriminated from the so-called *hydrencephaloid affection* incident in children to exhaustion from diarrhœa. With this affection it was formerly confounded. In this affection the symptoms denoting inflammation are wanting, and the drowsiness or stupor is not accompanied by strabismus, inequality of the pupils, embarrassment of respiration, irregularity of the pulse, and other symptoms, exclusive of the somnolence, which belong to the second stage of tuberculous meningitis.

To discriminate tuberculous from simple meningitis is by no means always easy. The gradual development of the former, the prodromic symptoms, the longer duration of the first stage, and the coexistence of pulmonary tuberculosis in a certain proportion of cases must serve as a basis of this differential diagnosis. The meningitis is probably simple if it can be distinctly traced to a traumatic origin. It is, however, to be borne in mind that in most cases it is possible to recall some injury of the head within a period of weeks or months prior to the occurrence of any disease in childhood; and it is customary to refer the affection to an injury often when there is not sufficient ground for such an opinion. Tubercles in the choroid coat are sometimes discovered by ophthalmoscopic examination. Their presence decides the differential diagnosis; but inasmuch as they are not always present in cases of tuberculous meningitis, their absence is not sufficient to exclude this affection.

PROGNOSIS AND TREATMENT.—If the diagnosis be positive, the encouragement for successful treatment is slight. It is difficult, of course, to decide upon measures which will be likely to be useful in a disease tending intrinsically, like this, to a fatal issue, and when in cases of apparent recovery we are obliged to distrust the correctness of the diagnosis. The chief source of encouragement for successful treatment, in fact, in a case which the practitioner supposes to be one of this disease, is the possibility that the diagnosis may be incorrect. With reference to this liability to error in diagnosis the measures indicated in simple meningitis are admissible. If, however, the practitioner be thoroughly convinced of the correctness of the diagnosis, he is hardly justified in resorting to bloodletting or measures which occasion suffering, such as blisters and frequent cathartics. Under these circumstances measures to palliate distress and prolong life are alone indicated. In this country the iodide of potassium is generally given largely in the second stage of the disease. Cases have been reported in which this remedy was apparently successful; but there is room for the supposition that in these cases either meningitis did not exist or the disease was simple meningitis. It is certain that in the immense majority of cases the remedy is unsuccessful. I do not, however, concur in the opinion held by most writers, that tuberculous meningitis is necessarily fatal. At least there seems to me ground for considering as probable recovery in some instances. I have reported an instance of apparent recovery in another work.[1] Recovery in no instance can be demonstrated, since it can always be said that the diagnosis may have been incorrect. Of course if recovery invalidate the diagnosis, the disease must be considered as always ending fatally.

Acute Spinal Meningitis.

It is doubtful whether acute inflammation of the spinal meninges ever occur as a primary or idiopathic affection. It has already been mentioned

[1] Vide *Clinical Medicine*, by the author.

that tuberculous inflammation of the spinal meninges is usually present in a greater or less degree in cases of tuberculous cerebral meningitis. Acute spinal meningitis is a prominent lesion in epidemic cerebro-spinal meningitis, an affection which may occur sporadically and will be subsequently described. Acute spinal meningitis, as well as acute cerebral meningitis, may occur, although rarely, as a complication in various infectious diseases, such as lobar pneumonitis, pyæmia, septicæmia, typhoid fever, typhus fever, other exanthematous fevers, gangrene of the lungs, and purulent pleuritis. Finally, acute inflammation of the spinal meninges may be secondary to disease of neighboring parts, in which case the inflammation is conducted to the membranes by continuity. Thus, acute spinal meningitis may be consecutive to acute myelitis or to diseases and injuries of the vertebræ, such as cancer, caries, and fracture, or to inflammations in the neighborhood of the vertebræ. In the latter case the inflammation may extend to the meninges through the intervertebral foramina without involving the vertebræ. When the inflammation is secondary to affections of the vertebræ or adjoining parts, there are generally both spinal pachymeningitis and leptomeningitis. In the other cases the pia-arachnoid membrane is the part chiefly inflamed. Although acute spinal meningitis has been often attributed to prolonged exposure to cold, it is doubtful whether this be ever an efficient cause of the disease.

The inflammatory products are fibrin, serum, and pus. In the ordinary form of the disease these products are accumulated in the subarachnoidal spaces, chiefly upon the posterior surface of the spinal cord. When the inflammation is secondary to fractures and diseases of the vertebræ, the exudation is usually both upon the outer and upon the inner surface of the dura mater. Sometimes the exudation is very abundant between the dura mater and the bone (acute peripachymeningitis). With acute spinal leptomeningitis there is generally more or less inflammation of the adjacent parts of the spinal cord (meningo-myelitis).

The local SYMPTOMS of acute spinal meningitis are chiefly referable to effects manifested through the spinal nerves, and they correspond to the functions of the posterior and anterior roots of these nerves. More or less pain is felt in the spine, radiating therefrom to the extremities. The pain is increased, not so much by pressure over the spinal column as by movements of the body. The sensibility of the surface is morbidly increased (hyperæsthesia). These effects are manifested through the posterior roots of the spinal nerves. Other effects are manifested through the anterior roots or by reflex action through the posterior—namely, tonic contraction of the muscles of the extremities, clonic contractions in some cases, spasm of the muscles of the back so as to give rise to opisthotonos, and sometimes rigidity of the thoracic muscles, causing dyspnœa. As regards the muscular contractions or spasms, there are exacerbations and remissions. These symptomatic phenomena are attributable to a morbid excitation of the spinal nerves, and they belong to the early part of the affection. Subsequently, paralysis occurs as the effect of pressure upon the cord by the products of the inflammation. The paralysis may be limited to the lower extremities (paraplegia) or it may be general; that is, affecting both the upper and lower extremities. The paralysis is rarely complete. It is usually limited to motion, and with it, indeed, exaggerated sensibility or hyperæsthesia may persist, as well as tonic contraction of muscles. With these local symptoms there is more or less fever, together with anorexia and prostration. The pulse is sometimes abnormally infrequent, and this may be found in striking contrast with the temperature of the body as determined by the thermometer. In the course of the disease, however, the pulse becomes frequent and feeble. Profuse perspirations often occur. Reflex and electrical excitability are generally preserved.

The diagnostic features point to the spinal cord as the seat of the disease and denote an inflammatory and a grave affection. Hysterical opisthotonos lacks the evidences both of inflammation and of gravity. Moreover, various antecedent and concomitant phenomena betoken the existence of hysteria. It is to be differentiated from tetanus, which is a very grave disease. The latter, however, is distinguished by the early occurrence of trismus and tonic spasms, by the absence of local and general symptoms denoting inflammation, and by the traumatic origin of the affection in the majority of cases. The diagnostic points involved in discriminating acute spinal meningitis from acute myelitis will be noticed in treating of the latter in the next chapter. It may, however, be remarked here that acute spinal meningitis and acute myelitis very rarely occur independently of each other. The two affections are generally associated; but the inflammation may be primarily seated in either situation, and it may predominate in one or the other.

Acute spinal meningitis often runs a rapid course, its duration not extending beyond a week. It ends fatally in a large proportion of cases. Life is destroyed by apnœa in some cases in which the respiratory function is notably compromised by either spasm or paralysis of the muscles concerned in respiration or by an extension of the inflammation to the medulla oblongata. If the latter occur, dysphagia is likely to be a symptom. In either case death is preceded by intense dyspnœa, which, on a physical examination of the chest, is found not to proceed from any adequate pulmonary or cardiac complication. In other cases asthenia predominates in the mode of dying.

The danger is to be estimated by the extent of the diffusion of the inflammation and the severity of the symptoms. The immediate danger depends on the extension of the inflammation upward so as to interfere with respiration. In cases of a moderate intensity of the disease recovery may take place rapidly and completely. The disease may continue and become chronic, or recovery may take place, leaving as sequelæ more or less atrophy and paresis of the muscles which had been affected.

The indications for TREATMENT are essentially the same as in acute cerebral meningitis. Considering the rapid course of the disease in severe cases, and the danger in the direction of apnœa, bloodletting, general or local, or both, may be employed if contraindicating circumstances relating to the pulse, the blood, and the constitutional strength be not present. Mercurialization is warrantable, if not desirable. If employed, it should be rapidly induced, and for this end inunction may be added to the administration of calomel at short intervals. Ergot and belladonna are supposed to diminish hyperæmia by causing contraction of the small arteries, and in this way to be useful. When the symptoms denote the presence of the products of inflammation, the vesication successively of different sections of the spine, and the iodide of potassium in large doses, are indicated. Sustaining treatment is of course indicated in proportion as the duration of the disease is prolonged and the symptoms show a tendency to death by asthenia. Retention of urine is to be prevented, and cystitis may claim appropriate treatment.

Chronic Spinal Meningitis.

It is now believed that chronic spinal meningitis with clinical manifestations is a much less frequent affection than was formerly supposed. This view is based upon the following facts: The symptoms described as characteristic of chronic spinal meningitis are also produced by diseases of the spinal cord and by diseases of peripheral nerves, and many of these diseases, being recognized only by careful microscopical examination, may readily escape observation at post-mortem examinations. Some of these diseases have been discovered only within a comparatively few years. Again, the lesions which have been considered as evidence of chronic spinal meningitis, such as thickening and opacity of the pia-arachnoid membrane, calcareous plates in this membrane, and adhesions between this membrane and the spinal cord on the one hand and the dura mater on the other hand, are extremely common, and are usually present without any evidence during life of a spinal affection. Chronic spinal meningitis is not infrequent as a secondary affection. It may follow epidemic cerebro-spinal meningitis or it may accompany chronic diseases of the spinal cord or of the vertebræ. In these cases, however, there are usually no distinctive symptoms referable to the affection of the meninges. But, although chronic spinal meningitis is doubtless an affection of much less clinical importance than was formerly supposed, and although the symptoms formerly referred to a primary chronic inflammation of the meninges are usually dependent upon some other affection, nevertheless, there remain certain cases which are still to be considered clinically as instances of chronic spinal meningitis.

The LESIONS of chronic spinal leptomeningitis are, thickening and opacity of the pia-arachnoid membrane, increased subarachnoid fluid, and adhesions between the pia mater and the spinal cord and between the arachnoid and the dura mater. As already mentioned, these lesions, as well as calcific plates in the arachnoid membrane, are common, especially in elderly persons, without any corresponding symptoms during life.

The CAUSES which have been assigned for chronic spinal meningitis are injury to the spinal column, exposure to cold, abuse of the sexual function, and chronic alcoholism.

The SYMPTOMS are similar to those of acute spinal meningitis, but they are of longer duration, less intense, and not accompanied by acute febrile reaction. The symptoms are pain and rigidity of the neck and back, pain in the extremities, hyperæsthesia, and muscular spasms. These symptoms of irritation gradually give place to symptoms of loss of function, such as anæsthesia, paresis of the extremities, incontinence or retention of urine, and incontinence of feces. There may be a sensation of constriction, as of a tight belt, around the body. If marked paralysis and anæsthesia exist, it may be assumed that the spinal cord is involved.

The following points are urged in distinguishing the paraplegia of myelitis from that of meningitis: The former is accompanied by less pain in the spine, limbs, and extremities; the paralysis is developed more rapidly, and is greater in degree; cutaneous anæsthesia and analgesia occur earlier, and are more marked; and the muscles sooner become atrophied.

In a considerable proportion of cases of paraplegia dependent upon chronic spinal meningitis the termination is unfavorable, but in general the prognosis is better than in myelitis.

In the TREATMENT of chronic spinal meningitis the indications for blood-letting, local or general, are rarely present. Dry cupping, blisters, sinapisms, and irritating liniments applied to the back are considered as highly useful while the inflammation continues. The application of the hot iron is recommended as an efficient method of counter-irritation. During this period the avoidance of exercise and of exposure to cold is important. When there is reason to think that inflammation no longer continues, it is an object of treatment to promote absorption of the inflammatory products. The iodide of potassium is supposed to be of use in this way. Mercury may be tried, with proper precautions to avoid ptyalism. Nothing should be done to impair the general condition, and, if it be impaired, restorative remedies and hygienic measures are indicated. Tepid baths seem to be of use. The improvement sometimes derived from mineral waters, used either internally or externally, probably does not depend on any special efficacy, but to a greater or less extent on the hygienic advantages which are associated with their use.

As regards treatment addressed directly to the paralysis, the measures are the same as in all paralytic affections whenever the measures are not contraindicated by conditions standing in a causative relation to the paralysis, the object being to maintain the nutrition and functional capacity of the affected muscles.

Cervical Hypertrophic Pachymeningitis.

This affection was first described by Charcot in 1871. It is a rare disease. It is characterized anatomically by the formation of layers of new connective tissue from the inner surface of the dura mater. This membrane thus becomes greatly thickened, and it is usually adherent to the pia-arachnoid membrane, which may also become thickened in a similar way. The cord lies imbedded in a mass of dense fibrous tissue, by which it is compressed. The cord may be simply anæmic, but frequently it is the seat of a chronic transverse myelitis. Below the myelitis the cord presents the lesions of a descending secondary degeneration. The nerve-roots which are surrounded by the new fibrous tissue are likewise compressed and atrophied. The disease is usually confined to the cervical region.

The causation is not known. Exposure to cold and the abuse of alcohol have been assigned as causes, but without sufficient proof.

The compression and irritation of the posterior roots of the cervical nerves give rise in an early stage of the disease to severe shooting pains in the back of the neck, extending to the occiput and to the arms. There is often complaint of formication and of numbness in the arms. At a subsequent period the compression of the anterior roots of the cervical nerves causes paralysis with atrophy of the muscles of the upper extremity, most marked, as a rule, in the muscles supplied by the ulnar and the median nerves. The atrophied muscles rapidly lose reaction to the faradic current. The hand may assume a position of exaggerated extension. There may now be anæsthesia of the upper extremities. In consequence of the cervical part of the spinal cord being involved, the motor nerves going to the lower extremities may lose their power of conduction; and then follows a spastic paralysis of the lower extremities. If there be a complete transverse myelitis, anæsthesia of the lower extremities and paralysis of the bladder and rectum follow. Bed-sores may form. The paralyzed muscles of the lower extremities do not undergo atrophy, as do those of the upper extremities.

Cervical hypertrophic pachymeningitis may be confounded with tumors pressing upon the cervical part of the spinal cord, with caries of the cervical vertebræ and with amyotrophic lateral sclerosis. The main points in diagnosis are the situation and the radiation of the pain and the characters of the paralysis in the upper and in the lower extremities. Amyotrophic lateral sclerosis is distinguished by the absence of sensory disturbances, by the preservation of the vesical functions, and by the frequent development of the symptoms of bulbar paralysis.

The disease is of long duration. The usual termination is in death, either by apnœa or by asthenia. The symptoms may, however, be arrested, at least for a long time, and cases have been reported in which recovery is said to have resulted.

It is not likely that any measures of treatment directly affect the lesion. Iodide of potassium and electricity are the therapeutical agents usually employed. Counter-irritation to the back of the neck by means of the hot iron has been recommended.

Cerebro-spinal Meningitis.

The name cerebro-spinal meningitis denotes an inflammatory affection of the arachnoid and pia mater of both the brain and the spinal cord. As a sporadic and purely local affection, it is considered rare, but of late years sporadic cases of cerebro-spinal meningitis are met with in New York not very infrequently. The name is generally understood as designating an epidemic disease characterized by cerebral and spinal meningeal inflammation. In the latter application it denotes an infectious disease in the sense in which the term infectious is at present defined. (Vide p. 84.) Epidemic cerebro-spinal meningitis is a general not a local disease. Its appropriate nosological place is among the essential fevers. It is with propriety called by some writers cerebro-spinal fever. The evidence of its having existed prior to the present century is not clearly apparent in medical literature. Since the beginning of the nineteenth century epidemics have occurred at different periods in various parts of the Old World and in this country. It prevailed in New England between the years 1807 and 1816, and was then known as *spotted fever.* For a quarter of a century after that period this country was exempt from its visitations; but within the last forty years it has prevailed from time to time in different districts of the United States and in Canada. This article will have reference to cerebro-spinal meningitis as an epidemic disease, or to cerebro-spinal fever.

ANATOMICAL CHARACTERS.—In the majority of cases an exudation of a purulent character, similar to that in simple meningitis, is found in the meshes of the pia mater both of the brain and cord. The extent and the abundance of the exudation vary much in different cases. The inflammation affects the pia-arachnoid membrane both of the convexity and of the base of the brain, but it is usually most intense at the base, and it may be limited to this situation. Within the spinal canal the inflammatory exudation is limited chiefly to the posterior surface of the cord and to the membranous envelopes of the posterior nerve-roots. The purulent exudation, however, is rarely distributed uniformly over the posterior surface, but is most abundant in the lumbar, lower dorsal, and lower cervical regions of the cord, while it may fail in the rest of the cervical region or be present there only in scattered patches. The exudation consists predominantly of pus, together with serum and fibrin, mixed usually with a considerable number of extravasated red blood-corpuscles. In recent cases mucin may be present in considerable amount, so as to render the exudation gelatinous. The membranes as well as the substance of the brain and cord are generally hyperæmic, and frequently the seat of small hemorrhagic extravasations. The substance of the brain and cord has usually an abnormally soft consistence, which may be particularly marked at certain points. The sheaths of the blood-vessels which penetrate the brain and the spinal cord are infiltrated with pus-cells, which may accumulate in sufficient number to form small abscesses in the brain or in the cord. The ventricles usually contain an increased amount of fluid, somewhat turbid in appearance or rarely decidedly purulent. The central canal of the spinal cord may be distended with purulent fluid.

In cases terminating within a few hours (fulminant cerebro-spinal meningitis) the anatomical evidences of the disease may be slight. In such cases the exudation is mainly serous, although the microscope will show that emigration of white blood-corpuscles has already actively begun. In these fulminating cases the brain-substance will be found, as a rule, œdematous and anæmic. In cases of prolonged duration the ventricles are usually found greatly distended with serum. The previously creamy, purulent exudation is perhaps now desiccated and caseous, or more frequently has nearly disappeared.

The following embrace the most important of the morbid changes existing in other parts of the body: The skin often presents herpetic vesicles and petechiæ. The voluntary muscles, especially of the neck and back, are dark-red in color and often the seat of granular degeneration. As in other infectious diseases, parenchymatous degeneration of the heart, liver, and kidneys is usually found. The lungs present no constant changes, but are frequently the seat of bronchitis, atelectasis, lobular pneumonia, and hypostatic congestion. The spleen, in the majority of cases, is not much enlarged, but is of lax consistence. Sometimes the spleen is found greatly swollen and softened. A moderate enlargement of Peyer's patches and of the solitary follicles is common. Abscesses in the joints and elsewhere have been occasionally observed. Endocarditis and inflammation of serous membranes are rarer complications. Suppurative inflammation of the middle and internal car, choroiditis, and iritis are to be mentioned as occasional concomitants or sequelæ.

CLINICAL HISTORY.—The characteristic features of epidemic cerebro-spinal meningitis are remarkably distinctive. In many cases the disease begins with a sudden attack, the patient being at once stricken down with grave symptoms; but the access may be more or less gradual, the development occupying from one to eleven days. The prodromic symptoms, enumerated in the order of their relative frequency, are—cephalalgia, chills without rigor or chilliness, nausea and vomiting, pain in the spine and limbs, vertigo, and diarrhœa. Cephalalgia is usually the most prominent symptom when the disease is developed. The pain is intense, lancinating, and accompanied with a sense of tension. It is referred either to the frontal region, the occiput, or the whole head. The pain persists without intermissions. It is increased by light, sounds, and movements of the body. Pain referable to the spine, or rachialgia, is generally added to the cephalalgia. The pain may extend over the whole of the spine, or it may be referred to either the lumbar, dorsal, or cervical portion. The extent and situation of the spinal pain probably correspond with the extent and situation of the spinal meningitis. The rachialgia is wanting if the disease prove fatal without inflammation extending to the spinal membrane. The spinal pain is not always increased by pressure over the vertebral column, but notably by movements of the body. Pain is frequently felt in different parts of the body, especially in the lower extremities. The pain in situations other than the head and spine is often intense. Hyperæsthesia of the surface of the body is often a marked symptom, rendering the slightest contact a source of suffering. Not infrequently vomiting accompanies the cephalalgia at the outset of the disease.

Delirium in some cases quickly supervenes, but in other cases it occurs after a variable period. Sooner or later it occurs in a large proportion of the cases which are not quickly fatal. It varies much in character and intensity. At first there is simply difficulty in collecting ideas, but after a time the patient either remains taciturn, making no response to questions, or replies incoherently or becomes actively delirious, shouting and struggling against restraint. The latter form of delirium occurs in paroxysms, with calm intervals. The paroxysms occur especially at night. They are accompanied by hallucinations in some cases, and insane delusions. Occasionally the delirium is hilarious. The mind is usually desponding and apprehensive if the intelligence be preserved. Loquacity and erotic desires with priapism have been observed. Repeated seminal emissions occurred in a case which came under my observation. In a certain proportion of cases the patient becomes quickly somnolent and falls into coma, which may be of temporary duration or continue until the disease ends fatally. More or less stupor, frequently amounting to coma, occurs not infrequently on the first day of the disease. The vision is rarely lost; double vision sometimes occurs; the pupils are frequently dilated, but sometimes contracted; the conjunctiva is often injected; and temporary deafness is an occasional symptom. In one of the cases of sporadic cerebro-spinal meningitis which I have observed lateral and vertical oscillation of the eyeballs was a notable symptom. The coma in some cases is without stertor, and resembles that of hysteria. The patients seem to be in a quiet sleep, crying out if any attempt be made to alter the position of the body. A patient in one case which came under my observation had remained in precisely the same position for forty-eight hours, and in another case for three days. Mobility of the iris may remain, although the patient is in this comatose or deeply somnolent state.

Important symptoms relate to the muscles. Tonic contraction of the muscles of the neck and back is of frequent occurrence, giving rise to retraction of the head and opisthotonos in a greater or less degree. Trismus sometimes occurs, and in rare instances pleurosthotonos. Contractions of muscles of the extremities are not infrequently observed. Pain in the muscles of the legs and thighs, the spinal muscles, and those of the neck is a source of suffering if the patient be conscious. Muscular tremor is an occasional, and strabismus not an infrequent, symptom. Subsultus and carphologia occur in cases in which the disease is protracted. Convulsions are not of frequent occurrence. Paralysis is also infrequent. Hemiplegic paresis and ptosis have been observed.

Nausea and vomiting are more or less prominent throughout the disease in a minority of cases. Constipation is the rule. The appetite is generally lost. However, a desire for food is sometimes expressed, although convalescence is not near at hand. The tongue is frequently large and flabby, showing indentations made by the pressure of the teeth. It becomes coated, and in the progress of the disease dry and dark. The teeth and lips frequently present sordes.

The respirations are sometimes suspirious. They are usually increased in frequency even when no pulmonary complication exists, and they are sometimes irregular. Stertor is rare. The pulse in the early stages of the disease is generally not more frequent, and is often less frequent, than in health. In the progress of the disease it becomes moderately accelerated, rarely exceeding 100 per minute, until toward a fatal termination, when it is frequent and small. Fluctuations in frequency at different periods of the day constitute a striking feature in some cases. Palpitations are occasionally observed. The quantity of urine may be greater than in health, and the urates are deposited in abundance. In some cases there is slight albuminuria, and exceptionally glycosuria has been observed. In a fatal case among those which I have observed there was hæmaturia, the hemorrhage being abundant and not occurring in any other situation.

The temperature of the skin varies in different cases and at different periods in the progress of the disease. At the beginning the temperature rarely exceeds, and often falls below, that of health. Notable heat of the surface is very rarely observed; the thermometer, however, shows the heat of the body to be more or less raised. Thermometric examinations in the axilla were made by Githens in 44 cases at 6 A. M., at 12 M., and at 6 P. M. daily, with the following results: The maximum of increase was 105° F.; and in only 2 cases was this height reached. The maximum in 15 cases was between 104° and 105°; in 7 cases, between 102° and 103°; in 6 cases, between 101° and 102°; and in 2 cases it was below 100°. The difference between the morning and evening temperature was less than in most of the essential fevers. "A rapid fall was the sure precursor of collapse."[1] As regards dryness and moisture, cases vary on different days and at different periods of the same day. Sweating is rare except in some cases shortly before death. The face is often suffused and of a dusky hue.

Petechial spots are frequently observed, but they are by no means constant. They appear to be of more frequent occurrence in some epidemics than in others. They vary in size from that of a pin's head to spaces a quarter of an inch in breadth or even larger. They are evidently due to an extravasation of blood or of blood-coloring matter, such as occurs in scorbutus, purpura, in some cases of continued fever, and occasionally in other affections. They are hardly entitled to be called an eruption. A rose-colored papular eruption resembling that of typhoid fever has been occasionally observed. Herpes of the face occurs often in some, and rarely in other epidemics. As herpes occurs very rarely, if ever, in other forms of meningitis, this symptom may be of diagnostic importance. Urticaria, erythema, and bullæ have been observed, but their occurrence is evidently accidental; that is, they do not belong to the clinical history of the disease. In some of the cases which I have observed sudamina were abundant.

A survey of the clinical history of this affection, exclusive of the cases in which death quickly ensues in apoplectic coma, shows a correspondence with the symptoms of acute cerebral meningitis, and the addition of those due to inflammation of the meninges of the spinal cord. The duration of the disease is very variable. Of fatal cases, the minimum duration is stated by Tourdes to be twenty hours. Ames states the minimum to be fifteen hours. The maximum duration in fatal cases is stated by Tourdes to be one hundred days, and by Ames more than forty days. Of the cases analyzed by Ames, 16 terminated fatally on the second or third, and 19 on the fourth day.[2] Of 160 cases analyzed by Dr. S. B. Hunt, 12, or 1 in 13, died within the first twenty-four hours; 92, or more than half, died before the close of the fifth day; 14, or 1 in 11, died before the close of the tenth day; 4, or 1 in 40, before the close of the fifteenth day; and 18 survived for various periods thereafter.[3] In Upham's cases the duration varied between thirty-six hours and six weeks.[4] Thus in the majority of fatal cases the disease runs a rapid course, ending prior to the fifth day. In cases which end in recovery the convalescence is generally tedious and the patient remains for a long period in feeble health. Relapses have not been observed.

It is not easy to divide the career of the disease into well-marked stages, and the arrangement of cases into different classes, such as inflammatory, congestive, malignant, etc., is not attended by any practical advantage. Remissions are stated to occur by different observers. They vary as regards the time of their occurrence, their duration, and the degree of amelioration of the symptoms. Not infrequently the improvement for a few hours, and sometimes for a day or so, is so marked as to give encouragement to hope that convalescence is about to take place. These remissions are most likely to occur on the second or third day. In a case under my observation the temperature and pulse became normal, the intelligence returned, and for twenty hours the patient seemed convalescent; the symptoms, however, returned and the case ended fatally.

PATHOLOGICAL CHARACTER.—All at present known of the pathological character of epidemic cerebro-spinal meningitis is embraced in the statement that it is an infectious disease and belongs among the essential fevers. The inflammation of the pia mater of the brain and cord is a local manifestation or an effect of an underlying general morbid condition. It is reasonable to suppose that the specific infectious agent is a micro-organism, but with the exception of a few observations of micrococci in the exudation nothing is positively known concerning such an organism. It must be admitted that the phenomena of the disease are largely symptomatic of the cerebro-spinal inflammation.

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[1] Vide *Am. Journ. of Med. Sciences*, July, 1867.

[2] Vide *New Orleans Med. and Surg. Journal*, 1848.

[3] Article by Dr. Hunt in the medical volume of the *Sanitary Memoirs of the War*.

[4] *Epidemic Cerebro-spinal Meningitis in the Camps in and around Newbern in 1862–63*, by J. Baxter Upham, M. D., Boston, 1863.

CAUSATION.—The prevalence of the disease as an epidemic implies the existence of a special cause. With respect to the source of the special cause and the circumstances concerned in its production we have no positive knowledge. Nearly all who have observed the disease concur in the belief that the causation does not involve a contagium. The disease has prevailed in situations in which periodical fevers rarely occur. It is, however, not improbable that in malarial districts the special cause may be combined with that giving rise to periodical fevers, and that the phenomena of the latter are sometimes associated with those of cerebro-spinal meningitis.

In France the disease has been observed to prevail especially among soldiers, and particularly among new recruits. In this country it has prevailed among troops stationed at several different points. It attacks persons in all conditions and at all periods of life. Infants are not exempt, but the liability is increased after seven years of age. In a series of cases the larger number are between the ages of twenty and thirty years. Of the persons attacked, the larger proportion are males. The difference, however, is not great. The statistics of Sanderson, cited by Hunt, show 164 cases among males, and 154 among females.

Epidemics occur much oftener in the winter and spring than during the summer months. Of the epidemics which have occurred in this country, nearly all were in the spring and winter months. Of 182 epidemics in Europe, analyzed with reference to this point by Dr. John Simon, 10 were in August and September; 24 were in October and November; 46 were in December and January; 48 were in February and March; 30 were in April and May; and 24 were in June and July. The disease, as stated by Hunt, who cites the foregoing statistics, "has its favorite habitat in prisons and barracks." "In the four or five years following 1837 it invaded nearly all the crowded barracks in France. It has appeared frequently among the galley-slaves of Toulon."

Facts pertaining to the localization of epidemics and the occasional occurrence of sporadic cases show conclusively that auxiliary causes which pertain to overcrowding, defective ventilation of dwellings, poisoning of the atmosphere by sewer emanations, etc. have a potent causative influence in association with the special cause; and it is an important fact that these auxiliary causes are by no means always confined to the habitations of the poor.

DIAGNOSIS.—Difficulty in making the diagnosis of cerebro-spinal meningitis relates chiefly to sporadic cases and to those in which the disease aborts or is unusually mild. Abortive cases sometimes occur, and the disease is sometimes so mild that its usual diagnostic features are not marked.

The disease need never be confounded with typhoid fever. The suddenness of the attack in many cases, the intensity of the cephalalgia, and the occurrence of symptoms denoting inflammation of the meninges of the brain and cord, distinguish it alike from typhoid and typhus fever. The characteristic abdominal symptoms of typhoid fever are wanting—namely, diarrhœa, meteorism, and iliac tenderness. Absence of the latter symptoms does not exclude typhus, but other diagnostic points belonging to the latter disease are wanting. The characteristic eruption of typhus, often copious, appearing on the third or fourth day after taking to the bed, does not occur in cerebro-spinal meningitis. The spots of ecchymosis on which the name spotted fever is based cannot be confounded with the eruption of typhus. Tonic contraction of the muscles of the neck and back, rachialgia, convulsions, and furious delirium, which occur in cases of cerebro-meningitis, are very rare in cases of either typhoid or typhus fever.

In the cases in which the patient falls quickly into coma and dies after the lapse of a few hours the affection resembles, in this fact, a paroxysm of pernicious intermittent fever. In general, however, the latter does not destroy life in the first paroxysm, and the occurrence of a paroxysm from which the patient has emerged suffices to establish the diagnosis. It is of course only in districts in which periodical fevers exist that the practitioner is called upon to make this differential diagnosis.

The occurrence of trismus and opisthotonos may suggest tetanus; but in tetanus the cerebral symptoms which belong to cerebro-spinal meningitis are wanting. Moreover, tetanus in other than tropical climates is exceedingly rare save as a traumatic affection, and it never prevails as an epidemic.

PROGNOSIS.—There are few epidemic diseases so destructive to life as cerebro-spinal meningitis. In the epidemic observed by Tourdes the proportion of deaths was 60 per cent. Ames gives the same proportion of fatal cases in the epidemic which prevailed in Alabama in 1848. In an epidemic observed by Lefèvre four-fifths died at the beginning, and two-thirds toward the end of the epidemic. Of 366 cases analyzed by Dr. S. B. Hunt, the deaths were 243 and the recoveries 123, making the percentage of fatal cases 70. Stillé states that ten epidemics between 1838 and 1848 gave an average mortality of 70 per cent., and a similar number of epidemics between 1855 and 1865 gave an average of about 36 per cent. He cites these statistics as showing a gradual decline of power in the epidemic cause. All observers agree as regards a large death-rate in cases of this disease. Mild cases, however, may occur during the prevalence of an epidemic, but even in these cases the physician should be cautious in giving a prognosis. The prognosis in all cases in which severe symptoms occur, such as notable delirium, coma, convulsions, and muscular contraction, is exceedingly grave. The disease is most likely to prove fatal in infants and in persons in middle and advanced life. It is stated that the proportion of fatal cases is greater among females than among males. The mode of dying, in the cases in which coma and death take place speedily, may be by apnœa, but in the majority of cases it is chiefly by asthenia.

Deafness and blindness are not very rare sequels of this disease. In the large majority of cases the deafness is bilateral. The blindness may be in both eyes, but oftener it is unilateral. The blindness is due to purulent choroiditis or to optic neuritis, and the deafness to purulent inflammation of the labyrinth of the ear.[1]

TREATMENT.—As might be expected in a disease so severe and fatal as this, various potent therapeutical agents have been employed. No reliable abortive or curative treatment has as yet been discovered. Experience has, however, shown the utility of certain measures in the employment of which the physician is to be governed by the rational and symptomatic indications present in individual cases.

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[1] Vide article by Dr. H. Knapp in the *New York Medical Record*, Aug. 15, 1872.

Great severity of symptoms, the patient having been attacked in full health and the pulse being strong or hard, renders bloodletting appropriate. Wet cups may be applied to the neck or leeches behind the ears in lieu of venesection when the indications for bloodletting are less clear. The abstraction of blood is contraindicated if the cerebro-spinal symptoms be not severe, if the pulse be readily compressed, and if the patient were anæmic or feeble when attacked.

Quinia in large doses has appeared in some cases to be useful. Its apparent success is perhaps, in a measure, to be accounted for by supposing that pernicious intermittent fever may have been mistaken for the disease, and by the fact that malarial effects may be associated with the latter. The utility of this remedy may sometimes be due to its antipyretic operation. For the reduction of a high temperature in this disease sponging the body and the wet sheet are preferable. These measures are indicated by intensity of fever.

The testimony in behalf of opium in large doses is discrepant. As regards its utility, however, there is very general agreement among physicians of large experience. Prof. Stillé recommends one grain of opium every hour in grave cases, and every two hours in cases of moderate severity. This recommendation is based on the study of about 120 hospital cases. He has not known any approach to narcotism in the cases thus treated. The hypodermic administration of morphia is to be preferred. It is important to avoid narcotism. The bromides, given in large doses, are regarded with favor by many physicians with whose practice I have become acquainted through professional intercourse.

The utility of cold applications to the head and back is conceded by all physicians. The extent to which this measure is to be carried is to be determined by the sensations of the patient and the effect upon the symptoms.

The foregoing remarks relate to the treatment when the symptoms denote cerebro-spinal excitation. When the symptoms are those of oppression, the objects of treatment are the same as in cases of simple cerebral and spinal meningitis—namely, limitation of morbid products and their removal by absorption. The iodide of potassium, given in doses as large as are well tolerated, is indicated. Mercury, internally, and by inunction, may do something toward effecting the objects just stated, and if so it is desirable to secure its usefulness, however small it may be.

Support by alcoholics and nutritious alimentation is indicated in proportion as the symptoms denote asthenia.

Whenever an epidemic or a sporadic case of this disease occurs, local auxiliary causes should be sought and removed. Removal of residents to places without the area of the infected situation, if practicable, is to be recommended.

Hydrocephalus—Hydrorrhachis—Syringo-myelus.

By the term hydrocephalus or hydrocephalus internus is understood an excessive accumulation of serous fluid in the ventricles of the brain, particularly the lateral ventricles. The name hydrocephalus externus is sometimes applied to great increase of fluid in the subarachnoid space. The serous effusion of hydrocephalus may be due to a variety of different processes, such as venous congestion, atrophy of the brain, changes in the walls of the blood-vessels, and inflammation of the lining membrane of the ventricles or of the pia mater. In some, especially the congenital, forms of hydrocephalus we are unable to determine the cause of the disease. There is but little tendency to dropsical effusions within the cranium in cases of general dropsy.

Hydrocephalus occurs either as a congenital affection or in early infancy, leading in some cases to an enormous enlargement of the cranium and giving rise to a characteristic deformity of the head. The reader is referred to works on the diseases of children for an account of this affection.

The name hydrocephalus *ex vacuo* is given to an effusion of serum into the ventricles as a result of wasting or atrophy of the brain-substance, such as occurs in old age. Under these circumstances, as the effused serum simply fills an unoccupied space, the brain does not suffer from compression. An abundant serous effusion is an important element in certain cases of tuberculous meningitis (acute hydrocephalus), and sometimes in other forms of meningitis. The exudation of fibrin and pus in some cases is very small, and the amount of effused serum large; but these are not properly cases of hydrocephalus. It was formerly thought that serous effusion sometimes occurs suddenly in sufficient quantity to occasion apoplexy (serous apoplexy). This was the theory in certain cases which are now explained by our knowledge of thrombosis, embolism, and uræmia.

Serous effusion into the lateral ventricles may occur in consequence of pressure upon the veins carrying the blood from the ventricles into the sinuses (venæ Galeni), as from a tumor of the pineal gland, solitary tubercles of the cerebellum, etc. A chronic accumulation of serum in the lateral ventricles, combined with a granular condition of the ependyma, has been described under the names *granular ependymitis* and *chronic ependymitis*. It occurs especially in cases of chronic alcoholismus and in old age. A similar serous accumulation may be present in chronic cerebral meningitis with or without a granular ependyma. The growth and coalescence of the granulations may cause obliteration of portions of the ventricles—for example, the anterior horns of the lateral ventricles—or may lead to the formation of bands of adhesion between the ventricular walls.

Extensive serous accumulation within the spinal canal is called *hydrorrhachis*. The effusion may be in the central canal of the spinal cord (hydrorrhachis interna or hydromyelus) or in the subarachnoid or subdural space (hydrorrhachis externa). These affections, when congenital, are often combined with deficiency of portions of the vertebral column, constituting the affection commonly known as *spina bifida*. In this affection a tumor consisting of the serous accumulation with its enveloping membranes (hydro-myelocele) protrudes through the fissure, most frequently in the sacral or dorsal regions. The affection is treated of in works on the diseases of children and on surgery. An accumulation of fluid in the central canal of the cord attends some cases of myelitis.

A peculiar affection of the spinal cord is designated by the name *syringomyelus*. In this disease a cavity is formed in the gray matter of the cord. This cavity may run longitudinally throughout the greater part of the cord, and it is not always easy to distinguish it from a dilated central canal. The cavity may be due to the breaking down of a gliomatous formation which usually originates near the central canal. Another explanation is that the tubular cavities of syringo-myelus are due to congenital anomalies of the central canal. The symptoms of syringo-myelus are usually of a complicated nature, depending upon the part of the cord invaded by the abnormal canal, this part being most frequently that occupied by the posterior horns of gray matter. The diagnosis cannot be made during life.

CHAPTER III.

INFLAMMATORY AND STRUCTURAL DISEASES OF THE BRAIN.

Encephalitis.—Abscess of the Brain.—Inflammatory Softening of the Brain.—Diffuse Sclerosis of the Brain.—Cerebral Paralysis of Children.—Infantile Spastic Hemiplegia.—Chronic Bulbar Paralysis.—Acute Bulbar Paralysis.—Intracranial Tumors.—Cerebral Syphilis.

Encephalitis—Abscess of the Brain.

THE terms encephalitis and cerebritis denote inflammation of the substance of the brain. It has already been mentioned that in cerebral meningitis the inflammation frequently extends more or less from the pia mater into the substance of the brain.

Encephalitis may present itself—1, as inflammatory softening; 2, as abscess; 3, as sclerosis or induration of the brain. In the present article abscess of the brain will be considered, and afterward the other forms of encephalitis.

Suppurative encephalitis may occur in any part of the brain. It is most common in the cerebral hemispheres and in the cerebellum. It is rare in other parts of the brain. Recent abscesses have a shaggy wall of softened cerebral tissue. Old abscesses are usually invested by a fibrous capsule. In the majority of cases the abscess is single, but sometimes there are several distinct abscesses, which are then usually small in size. Abscesses have been described which involve nearly a whole cerebral hemisphere, but in the majority of cases the size varies between that of a pigeon's egg and that of a hen's egg. Abscesses may perforate into one of the ventricles or may burst upon the exterior of the brain. The pus is sometimes fetid, especially when the abscess is secondary to putrid affections of the lungs. The pus may be gelatinous in consistence, and it is sometimes acid in reaction.

The most frequent cause of cerebral abscess is injury to the head attended by wound of the soft parts or fracture of the skull. Sometimes the abscess follows injury without wound or fracture. Suppurative inflammation of the brain is often secondary to caries of the cranial bones and to disease of the middle or internal ear, and less frequently to disease of the orbit or of the nose. It may be secondary to empyema, to gangrene of the lungs, and to bronchiectases with putrid secretion. In these cases, as well as in cerebral abscesses complicating pyæmia and acute ulcerative endocarditis, the abscess is probably due to infectious emboli lodging in the cerebral arteries. Other acute infectious diseases, such as typhoid fever and the exanthematous fevers, are very rarely followed by cerebral abscess.

Cerebral abscess may occur as an apparently primary or idiopathic disease. In a certain number of these cases it has been suggested that the abscess is the result of infection with the special poison of cerebro-spinal meningitis.

Cerebral abscess is more common in males than in females, and during the period from twenty to forty years of age than before or after this time. It is very rare after sixty years of age.

Even large abscesses of the brain, particularly of the primary variety, may remain latent for a long time, and they are sometimes discovered accidentally at autopsies. When the abscess is secondary to severe injury to the head or to middle-ear disease, it is generally difficult to distinguish the affection from acute cerebral meningitis, which, in fact, is often also present. In these cases the course of the disease is generally acute.

Cerebral abscess often pursues a chronic course, and then more or less characteristic symptoms are present. Some of these symptoms are referable to the abscess *per se;* others are dependent upon its situation. Of the first group of symptoms the most important are—headache, which may be severe or of a dull character, vertigo, vomiting, rigors, and fever. These symptoms are not always present. Rigors are not common. Fever, if present, is usually not high and is of an irregular type, with remissions or intermissions. The symptoms which are dependent upon the situation of the abscess are manifold. If the motor regions be involved, muscular spasms occur, followed by paralysis (which may be complete or incomplete) in the form of hemiplegia or of monoplegia. Epileptiform convulsions are often present if the motor cortical zone be affected. Abscess seated in the occipital lobe may give rise to hemianopsia, and seated in the temporal lobe or involving the third frontal convolution on the left side, to aphasia. Disturbances of the pupils and of vision are common. (For further points which bear upon the localization of cerebral abscess, the reader is referred to the article on Topical Diagnosis, p. 672.)

Abscess of the brain may give rise to somnolence deepening into coma. The coma may develop suddenly, so as to give rise to the suspicion of hemorrhage or of embolism.

The DURATION of cerebral abscess is variable. The acute form may terminate fatally within a few days following the onset of the disease. The chronic form generally lasts for several months, and it may be for years.

The DIAGNOSIS involves especially the discrimination from cerebral meningitis, tumor of the brain, and thrombic softening. As already mentioned, the diagnosis of acute cerebral abscess, following traumatism and disease of the middle ear, from cerebral meningitis is difficult and often impossible. The main points to be considered are that in meningitis the cephalalgia, delirium, and fever are usually more intense, whereas in abscess symptoms dependent upon a local affection of the brain, such as hemiplegia, monoplegia, hemiopia, etc., are more common than in meningitis.

A capital point in the discrimination of abscess from tumor of the brain is the causation. When the symptoms follow injury to the head, ear disease, pulmonary disease, and the other causes of abscess which have been mentioned, the probability is in favor of abscess. The existence of rigors and of fever is in favor of abscess. Choked disc is rare in abscess and is common in tumor. When the diagnosis is doubtful, especially when none of the causes of abscess can be determined, the probabilities are in favor of tumor, inasmuch as tumor is much more frequent than primary abscess. Cerebral embolism and thrombosis can often be excluded by attention to the causation. Coma resulting from abscess is generally preceded by marked cerebral symptoms, which are absent or less prominent in embolism or thrombosis.

Cerebral abscess in nearly all cases ends fatally after a variable period. The immediate cause of death may be general œdema of the brain, producing coma, or rupture of the abscess either into one of the ventricles or upon the surface of the brain. The abscess may become encapsulated, and remain stationary for an indefinite time.

The TREATMENT is generally to be directed to symptoms. Depletory and debilitating measures are to be avoided. Tonic remedies, a nutritious diet, repose of the mental faculties, and stimulants if the patient be in danger from depression of the vital powers, should constitute the treatment.

The only possibility of effecting recovery is to trephine the skull and make an incision into the abscess. The operation is justifiable if the diagnosis be reasonably certain and if it be probable that the abscess is in an accessible situation. These conditions are not likely to be fulfilled except when the abscess is the result of injury or of disease of the bones or of the middle ear. Surgical works are to be consulted for the operative treatment. Several successful operations have been recorded.

Inflammatory Softening of the Brain.

Formerly, nearly all varieties of cerebral softening were considered to be inflammatory in their origin. It is now known that the majority of cases of softening are not inflammatory, but the result of suspended nutrition (necrobiosis), attributable either to embolism or to thrombosis. There remain, however, rare cases of primary softening the cause of which cannot be found in changes in the blood-vessels, that are still classified as inflammatory, although our knowledge is very imperfect as to their genesis and their distinctive pathological characters. It is possible that in these cases the softening is analogous to that present in many cases of myelitis. This encephalitic softening is generally reddish in color, and hence is called inflammatory red softening. The affected part of the brain, which may be either the white or the gray matter, is softened, somewhat swollen, of a diffuse reddish-yellow color, and frequently dotted with red points of capillary blood-extravasations. The region of inflammatory softening is not distinctly circumscribed, but is surrounded by a zone of œdematous brain-tissue. The microscopical examination reveals hyperæmia, blood-extravasations, broken-up nerve-fibres, fatty and disintegrating ganglion-cells, pus-cells in greater or less number, compound granular corpuscles, and sometimes pigment-granules. The histological characters do not suffice to distinguish inflammatory from non-inflammatory softening. The lesion has nothing to do with abscess, which, in the brain as well as elsewhere, is due to some specific infection. Whether this form of softening can terminate in resolution or complete restitution of the normal structure is unknown, but such a termination does not seem probable.

The SYMPTOMS resemble those of thrombic softening, and of course are various according to the situation of the disease. They do not enable us to make a diagnosis of inflammatory softening.

The cases which are designated popularly as softening of the brain are often cases of thrombic softening. The term "softening of the brain," however, is often used in a vague sense to designate mental weakness, which may be dependent upon a variety of morbid conditions.

As is evident, the TREATMENT of encephalitic softening must be directed to symptoms, and it does not differ from that applicable to softening from thrombosis. The general health of the patient should be maintained so far as possible. The phosphide of zinc, chloride of ammonium, and bromide of potassium have been recommended.

It is customary to include also under the term encephalitis certain cases in which cerebral symptoms, such as paresis of one or more of the extremities or of the face, muscular spasms, disturbances of speech, appear, and then after a variable period, usually of some weeks or months, wholly or in great part disappear. The pathogeny of these temporary forms of cerebral disorder is not understood, but they cannot depend upon severe structural lesions.

Diffuse Sclerosis of the Brain.

Sclerosis of the brain is characterized by an increase of the neuroglia. Sclerosis may affect the whole or the greater part of the brain, when it is called diffuse cerebral sclerosis, or it may occur in isolated, disseminated patches, and it is then distinguished as multiple or insular sclerosis. Multiple cerebral sclerosis is generally accompanied by a similar lesion of the spinal cord. The disease is then called cerebro-spinal sclerosis, which is a well-characterized affection, and will receive consideration in a subsequent article. At present only the diffuse form of cerebral sclerosis will be considered.

In diffuse cerebral sclerosis the brain presents an abnormally firm consistence. The brain is usually more or less atrophied, and the ventricles are frequently dilated. The microscopical changes are often less marked than the gross appearances would lead one to suspect. When these changes are marked, the lesion is found to be an increase of the neuroglia, usually, but not always, with atrophy of the nerve-elements. The centrum ovale is generally most affected, but the cortex may also be sclerotic.

Diffuse cerebral sclerosis is most common in the brains of idiots, old epileptics, and patients affected with the general paralysis of the insane. A certain amount of diffuse sclerosis with dilatation of the ventricles is not uncommon in the brains of those dying from chronic alcoholism. Diffuse cerebral sclerosis occurs in children as well as in very old persons.

The SYMPTOMS vary according to the extent and the intensity of the lesion. There is generally mental impairment which may amount to dementia. Epileptic convulsions are common. Rhythmical muscular spasms or chorea may be present. There is frequently hemiplegia, and when both hemispheres are affected there may be paresis on both sides of the body. The paralysis is often of a spastic nature. Speech is frequently disordered. The cutaneous sensibility is usually but little or not at all affected. Defective vision and deafness are not uncommon.

The COURSE of the disease is chronic. The duration is long, extending often over many years.

The TREATMENT relates only to symptomatic indications.

Spastic Cerebral Paralysis of Children.—Infantile Spastic Hemiplegia.

In the present article will be considered a peculiar form of hemiplegia occurring in children which resembles in many particulars the so-called spinal paralysis of children. As the latter affection is caused by a special form of myelitis involving the ganglion-cells of the anterior horns of the spinal cord, so the disease under consideration is the result of a special form of encephalitis involving the motor region of the cerebral cortex.

No post-mortem examination has as yet been made in an early stage of the disease, but the symptoms render it probable that the lesion is primarily an acute encephalitis of the motor cortex. In cases of long standing there have been found atrophy and sclerosis of the central convolutions in whole or in part. The affected convolutions are shrunken and are firm in consistence. The microscope shows increase of neuroglia and atrophy of the ganglion-cells, especially of the large pyramidal ganglion-cells. The disease may be either unilateral or bilateral, but is generally more marked in one hemisphere than in the other. Secondary degenerations are found in the pyramidal tracts.

The disease seems to be confined to children and begins usually within the first four years of life. The onset is generally acute and marked by fever, and often by convulsions. The existence of paralysis is not usually detected until after the febrile paroxysm, which is ordinarily of only a few days' duration, has subsided. The arm and the leg are paralyzed upon the side opposite to the lesion. The face is less frequently affected. The paralysis may be in the form of a monoplegia. The paralysis is accompanied with more or less muscular rigidity and contractures, not, however, as a rule, involving the fingers. The paralyzed muscles, although atrophied, do not present the reaction of degeneration to be subsequently described. Spasms of the paretic muscles, particularly in the form of athetosis or of chorea, are common. The children may become epileptic. Sensation is not affected. The tendon reflexes are usually increased. Temporary aphasia is common with right-sided hemiplegia.

Infantile spastic hemiplegia is distinguished from the spinal paralysis of children by the hemiplegic form of the paralysis, by the absence of the reaction of degeneration, and by the increased tendon reflexes.

There may be some improvement in the degree and extent of the paralysis soon after it first appears. The paralysis which remains after this early improvement is permanent. The paralyzed members are less developed than the others. The mental development may be normal, but it is often defective.

The TREATMENT at the onset of the disease consists in cold applications to the head and in the administration of mild purgatives. For the subsequent paralysis electricity and massage are of benefit. Bromide of potassium is useful if epileptic convulsions occur.

Chronic Bulbar Paralysis—Glosso-labio-laryngeal Paralysis.

This form of paralysis was described first by Duchenne in 1860 under the name "progressive muscular paralysis of the tongue, the palate, and the lips." Since that date a variety of names have been proposed, among which may be mentioned chronic progressive bulbar paralysis, progressive bulbo-nuclear paralysis, progressive atrophic bulbar paralysis, and glosso-labio-laryngeal paralysis. The disease is characterized by progressive paralysis and atrophy of the muscles of the lips, tongue, soft palate, pharynx, and larynx. The symptoms and the anatomical lesions are analogous to those of progressive muscular atrophy, with which, in fact, chronic bulbar paralysis is not infrequently combined. Chronic bulbar paralysis is also often associated with amyotrophic lateral sclerosis. The anatomical basis of chronic bulbar paralysis is to be found in an atrophy of ganglion-cells belonging to the nuclei of origin of certain motor nerves of the medulla oblongata. While there is no doubt that the essential change is an atrophy and disappearance of motor ganglion-cells, it is not certain whether the morbid process consists in a primary atrophy of these cells or in a primary interstitial myelitis with secondary degeneration of the nerve-cells. The medulla oblongata may present no distinct alterations to the naked eye, or perhaps there may be detected a few ill-defined gray and atrophic spots near the floor of the fourth ventricle. Microscopical examination is required for an accurate localization and study of the lesions. Atrophy and disappearance of the ganglion-cells have been found in the main and accessory nuclei of the hypoglossal nerves, in the motor (anterior) nuclei of the pneumogastric, spinal accessory, and glosso-pharyngeal nerves, in the nucleus of the facial, and in the motor (masticatory) nucleus of the trigeminal nerves. The changes are bilateral. Of these nuclei the main nucleus of the hypoglossus seems to be earliest and most extensively involved. Sometimes it possesses not a single ganglion-cell. Some of the above-named nuclei may escape—for example, the motor nucleus of the fifth nerve. In the process of atrophy the ganglion-cells become rounded, devoid of processes, filled with pigment and diminished in size, and finally they disappear. In the nuclei of origin, some increase of the neuroglia (interstitial myelitis), corpora amylacea, and granular corpuscles usually attend the degenerative changes in the ganglion-cells. The degenerated spots are most readily recognized on microscopical sections stained with carmine by their red color, which is deeper than that of the normal parts. The nerve-fibres in the roots and trunks of the nerves whose centres of origin are affected undergo, to a greater or less extent, degeneration and atrophy. Although the muscular fibres in the tongue are atrophied, the organ may preserve nearly its normal dimensions in consequence of a formation of fat-cells in the interstitial tissue—a change recognized by the yellow color on section. The atrophy which takes place in the muscular fibres in this affection is of the same nature as that to be described for progressive muscular atrophy.

Clinically, the disease is characterized by progressive diminution of voluntary power over the tongue, the muscles of the palate, and the lips. The disease begins insidiously. The paralytic effects are bilateral and symmetrical. The loss of power, at first slight, gradually increases. After a time there is much difficulty in articulation. The patient is unable to pronounce words and the letters of the alphabet, especially the consonants requiring for their enunciation movements of the tongue and lips. Speech at length is impossible, and the only means of communication is by signs or writing. The paralysis of the orbicularis oris renders the lips immovable during emotions of grief or mirthfulness. The mouth is elongated and gives to the face an expression as if about to weep. The saliva is increased and dribbles over the lips. Food collects between the teeth and cheeks, and the fingers are required to dislodge it. The ability to protrude the tongue or to curl the tip upward is lost. Fibrillary contractions of the muscles of the tongue and of the lips can usually be observed. When the paralysis of the tongue and orbicular muscles of the mouth has increased to a certain extent, or even from the outset and before the paralysis has extended to the lips, the muscles of the palate are affected. Deglutition then is more or less difficult, food and drink being often returned through the nostrils. The laryngeal muscles become paralyzed, causing aphonia, with either diminution or cessation of the respiratory movements of the glottis. Paresis of the muscles of respiration may follow. The acts of respiration are feeble, and the power of coughing is impaired.

Portions of food and drink and the salivary fluids are often drawn into the larynx, and are with difficulty expelled, owing to the inability to cough effectively. For this reason, if the patient be attacked with bronchitis, there is danger from the accumulation of mucus in the bronchial tubes. Paralysis may occur in other parts of the body and may extend more or less over the voluntary muscles. The progress of the affection is unattended by fever. The affection advances steadily, but more or less gradually, toward a fatal termination. Death may be caused by apnœa from impaired ability to carry on respiration, and from accumulations of foreign material in the bronchial tubes and air-cells, causing bronchitis, aspiration-pneumonia, or pulmonary gangrene; or the mode of dying is by asthenia or starvation, in consequence of inability to ingest an amount of aliment sufficient to sustain life. At an advanced period the action of the heart may be notably disturbed. Paroxysms of feeble, irregular action, together with cyanosis, sometimes occur, and death may take place by syncope.

The intelligence remains unaffected. Although the paralyzed muscular fibres present the reaction of degeneration, this is generally difficult to determine on account of the preservation of a certain number of fibres with normal electrical excitability. Reflex excitability is generally lost, but in some instances it is retained, and in rare instances is increased. The paralyzed muscles are atrophied. The general sensibility of the mouth, tongue, and pharynx is not affected; the gustatory sense and the other special senses are preserved; and the appetite and digestion may be unimpaired.

The affection is readily distinguished from double facial paralysis (*diplegia facialis*) by the fact that the orbicular muscles of the eye and the muscles of the face, other than the orbicularis oris, are not affected. In its progressive character and the occurrence of paralysis in other situations, it bears a resemblance to general cerebral paralysis, in which the muscles concerned in speech are often the first affected. It differs from the latter affection in the fact that the intellect remains unaffected; and paralysis of other muscles than those of the tongue, mouth, pharynx, and larynx occurs incidentally in only a certain proportion of cases. The group of symptoms which characterize bulbar paralysis may occur in the course of progressive muscular atrophy. In the latter disease, however, the muscles of the extremities or trunk are also affected. In other words, spinal disease accompanies that of the medulla oblongata. Chronic bulbar paralysis may be preceded or followed by progressive muscular atrophy. In like manner the spinal diseases, called *amyotrophic lateral sclerosis* and *disseminated sclerosis of the cord*, may involve by extension the medulla oblongata, and give rise secondarily to the symptoms which belong to the history of bulbar paralysis. Embolism or thrombosis of the vertebral arteries, occluding the branches going to the medulla oblongata, and hemorrhage limited to the latter, have been known to cause the group of phenomena which characterizes bulbar paralysis. The differential points pertaining to thrombosis and hemorrhage are, the sudden occurrence of the paralysis of the tongue, lips, and pharynx, and the occurrence simultaneously of paralysis either unilateral or bilateral of the limbs. The name acute bulbar paralysis has been applied to these cases. (See next article.) Tumors in the medulla oblongata, in connection with the symptoms of bulbar paralysis, give rise to those denoting cerebral disturbance and to ophthalmoscopic appearances which are wanting in the latter disease.

With reference to CAUSATION, the fairest statement is a confession of ignorance. The disease very rarely occurs before forty years of age, and the male is more liable to it than the female. Occupation and station in life have no causative influence. Beyond these facts nothing can be positively affirmed in regard to the etiology. It is, however, reasonable to suppose that exposure to cold, excesses of all kinds, over-exertion, either physical or mental, and, perhaps, syphilis, have more or less agency in the development and progress of the disease.

In chronic bulbar paralysis involving irremediable anatomical changes in the medulla oblongata, all that can be hoped for is that the disease will cease to be progressive, or, next to this, if the progress continue, that this will be slow. The extreme limit of the duration of the disease is five years. The prognostics which show the fatal termination to be not far distant are, complete dysphagia, great debility from inanition, embarrassment of respiration, and disturbances of the heart's action from syncope.

The objects of TREATMENT are, *first*, the arrest of the progress of the disease, and, *second*, if this object be unattainable, retardation of its progress. With reference to these objects, the patient should, as far as possible, be withdrawn from every agency which may be supposed to have a causative operation. All unsanitary influences pertaining to body and mind are to be removed. The system should be well nourished, and measures are to be employed for restoration of the general health if this be in any way impaired. These injunctions embody, perhaps, the most important part of the treatment, with our present knowledge. It is not certain that any remedies have a special influence on the disease. An able observer (Erb) warns against the administration of strychnia and phosphorus; yet the latter is undoubtedly useful in some cases of a similar disease seated in the spinal cord—namely, progressive muscular atrophy—and also in multiple sclerosis. It is doubtful if much benefit be derived from the nitrate of silver, the iodide of potassium, ergot, and belladonna, but it is proper to make trial of these remedies. There is very little ground for expecting benefit from counter-irritation. Most neurologists attach importance, *par excellence*, to electricity, but it does not seem, thus far, to have effected great results. Persistent trial of this agent, however, is not to be omitted.

When deglutition becomes difficult, nourishment should be introduced into the stomach by means of the œsophageal tube, and rectal alimentation may be resorted to. In order to avoid the danger of foreign substances entering the bronchi, it is well not to delay too long before resorting to the use of the œsophageal tube.

Acute Bulbar Paralysis.

Various lesions of the medulla oblongata may give rise to the group of symptoms which has received the name of apoplectiform or acute bulbar paralysis. Such lesions are hemorrhage, softening from embolism or thrombosis of the vertebral and basilar arteries, acutely developed compression of the medulla oblongata, and acute inflammation. In three cases of acute bulbar paralysis examined by Leyden there were found in the medulla oblongata a number of small foci of inflammatory softening. These foci may be discoverable only upon microscopical examination. The term acute bulbar paralysis is sometimes limited to the inflammatory variety, which will be taken as the basis of the following description of the symptomatology.

The SYMPTOMS develop acutely, and often rather suddenly. The disease may be ushered in by pain in the head and back of the neck, vomiting, and dizziness. The characteristic bulbar symptoms soon appear. These are difficulty in swallowing, difficulty in articulation producing the disturbances of speech called dysarthria and anarthria, and often irregularity in the rhythm of respiration and of the pulse, which is frequent, small, and intermittent. There is often, but not always, elevation of temperature. The extremities may or may not be paretic. Not infrequently there is paralysis of facial and of ocular muscles. Paralysis of the diaphragm has been repeatedly observed.

The DURATION of the disease is from four days to two or three weeks. The termination appears to be always fatal, and is generally due to paralysis of respiration.

No measures of TREATMENT have thus far been attended with success. Counter-irritants to the back of the neck may be employed. The indications are essentially the same as in acute myelitis.

Tumors within the Cranium.

Most of the tumors which have been found in other parts of the body may also develop within the brain. Among the most frequent and characteristic of cerebral tumors is *glioma*. It develops from the cerebral substance, and not from the membranes. It is met with elsewhere only in the spinal cord and retina. It is the sarcoma of the brain, differing from ordinary sarcomata by the presence of that finely-fibrillated and molecular connective substance called neuroglia. A peculiarity of glioma is that it often appears more as a diffuse infiltration than as a circumscribed growth. The consistence of the tumor may be either hard or soft. The hard variety may be mistaken for cerebral sclerosis, and the soft for cerebral softening. The tumor is often very vascular, and may be the seat of hemorrhages. Sometimes the hemorrhage is so great as to destroy the larger part of the tumor and to present the appearance of a primary cerebral hemorrhage. In such a case the microscopical examination of the tissue immediately surrounding the hemorrhage will usually reveal gliomatous structure. In some cases of glioma there can be demonstrated large numbers of the branching cells, called brush-cells and spider-cells, which characterize the normal neuroglia. In other cases the tumor seems to consist chiefly of small round cells imbedded in a finely-fibrillated granular matrix. In the latter cases proper methods of demonstration will often reveal the characteristic branching cells. Glioma is often combined with sarcoma, constituting the so-called *glio-sarcoma.* All varieties of *sarcoma* may develop in the brain, either as primary or as secondary growths. They originate more frequently in the membranes than in the brain-substance. *Gummata* are among the most important of cerebral tumors. They take their origin almost invariably from the membranes, but not infrequently grow from the pia mater into the brain-substance. They also form in many cases diffused growths. They are usually combined with chronic inflammatory changes (gummatous meningitis). They usually present spots of cheesy degeneration. *Solitary tubercles* consist of nodules, varying in size from a pea to an apple, which present a cheesy centre and a periphery of connective tissue rich in small round cells. In the peripheral zone there are sometimes miliary tubercules containing endothelioid cells and giant-cells. The tubercle bacilli have been found in solitary tubercles. Tubercles may be present in other parts of the body. Solitary tubercles may occur in any part of the brain. They are found with especial frequency in the cerebellum. The brain and its meninges may be the seat of secondary *carcinoma.* Tumors presenting a structure very similar to carcinoma may develop primarily in the membranes. They probably take their origin from endothelium in the perivascular sheaths (endothelioma). The tumor called *cholesteatoma* is misnamed, as it by no means always contains cholesterin. The appearance of cholesterin is afforded by the presence of flat epithelial-like cells, often arranged in concentric masses. *Psammoma* derives its name from the presence of little calcareous granules resembling particles of sand. The calcareous masses often present a concentric arrangement. They appear to be connected with the blood-vessels. The last two varieties of tumor grow from the membranes, especially from the dura mater. Of other intracranial tumors may simply be mentioned fibroma, myxoma, lipoma, lymphoma, osteoma, angioma, neuroma, papilloma, and tumors of the pituitary body. (The reader is referred to Part I. p. 42 *et seq.* for a brief consideration of most of the tumors found in this situation.) In this connection may be mentioned the occurrence in the brain of the parasites cysticercus cellulosæ and echinococcus. The cerebral arteries are occasionally the seat of *aneurism,* aside from those distinguished as miliary. Aneurismal tumors are generally connected with arteries at the base of the brain, the basilar, the internal carotid, the middle cerebral, and the circle of Willis. Generally the tumors are small, but they are sometimes of the size of a pullet's egg.

The different tumors produce morbid effects by pressure on the brain, and their effects, thus produced, other things being equal, are proportionate to the size of the tumor and the rapidity of its growth. Special morbid effects depend on the situation of the tumor. They cause additional injury to the brain, either by exciting circumscribed cerebritis, and thereby inducing softening of the brain-substance surrounding them, or by interfering with nutrition, and thus leading to non-inflammatory softening; and as a result of the softening hemorrhagic extravasation is not uncommon. These morbid effects are produced alike by different tumors; that is to say, the morbid effects have no relation to the particular kind of tumor. Hence the kind of tumor in any case cannot be determined by the symptoms which it occasions. For this reason the several tumors are considered collectively, instead of receiving each a separate consideration.

Of the SYMPTOMS which accompany tumors within the skull, cephalalgia is usually prominent. The pain is frequently limited to a circumscribed space which sometimes, but not always, corresponds to the site of the tumor. Pain is prominent as a symptom in proportion as the tumor gives rise to circumscribed meningitis or increases the intracranial pressure. Other cerebral symptoms more or less prominent are—vertigo, difficulty in preserving an equilibrium in walking, momentary loss of consciousness, tinnitus aurium, the feeling of formication, numbness in different parts of the body, and scintillations; hyperæsthesia of the surface followed by blunted sensibility; mental irritability, to which succeed dulness and progressive impairment of the faculties of the mind; sometimes active delirium, and frequently epileptiform convulsions. The so-called compelled movements, either backward, forward, or rotatory, or in a circle (circus movements), are sometimes, although rarely, observed. Vomiting may be a prominent symptom, occurring irrespective of the ingesta and not accompanied by symptoms denoting gastritis. This symptom is likely to be prominent in the cases in which cephalalgia and vertigo are unusually marked. The pulse is often slower than normal. Constipation is the rule. Polyuria and glycosuria have been noted, especially in tumors pressing on the pons or medulla oblongata.

Paralysis more or less extensive, affecting the special senses and voluntary muscles, occurs sooner or later. Hemiplegia, generally incomplete, is a frequent form of the paralysis. The hemiplegia is often developed from successive monoplegias. In some cases the two sides are successively affected, constituting double hemiplegia. The hemiplegia may have been preceded by facial paralysis, and if the latter do not precede it may either accompany or follow the hemiplegia. The tumor may be so situated as to press upon certain of the cranial nerves exterior to the brain. The paralysis of the parts supplied by these nerves will then be on the same side as the tumor and opposite to the hemiplegia, whereas if the paralysis be due to pressure of the tumor within the substance of the brain the cranial nerves affected are those on the same side as the hemiplegia and the side opposite to the hemisphere in which the tumor is situated. The paralysis caused by pressure upon nerves beyond their central connections have the distinctive characteristics of a peripheral paralysis. The affected muscles atrophy and speedily lose electrical excitability. The paralyses may be limited to the seventh or portio dura nerve, or other cranial nerves may be involved. Loss or impairment of vision in one or both eyes occurs when the tumor either involves destruction of the optic nerve or interrupts its function by pressure. Neuralgia and paralysis of sensation on one side of the face result from similar effects upon the fifth nerve. The third and the fourth nerves may be involved, and not infrequently the paralysis affects, separately or collectively, the senses of hearing, smell, and taste.

The differences in different cases, as regards the parts affected with paralysis, of course depend on the seat of tumors, their size, and the direction in which they extend. With reference to the differential diagnosis of tumors and inflammatory softening or other lesions, the extension of the paralysis to different parts in succession after variable intervals is a highly significant fact. The paralysis affecting successively parts supplied by different cranial nerves, the members in some cases becoming affected first on one side and then on the other side, and the continued increase of the degree of the paralysis of different parts, point with significance to tumors, because such a history is in correspondence with their continued growth and the progressively increasing amount of pressure and of destruction of cerebral substance. Aphasia in some cases is an effect of tumors, and this symptom renders it probable that they affect the posterior part of the inferior convolution of the left frontal lobe. The ophthalmoscope is useful in determining the existence of intracranial tumors. Morbid changes in the optic discs, either congestive, inflammatory, or atrophic, are found in a very large proportion of cases, even though vision be not appreciably affected. The ophthalmoscope may be, therefore, of much value in the diagnosis, even before there are any subjective symptoms referable to the eye. Swelling of the optic papilla, producing the appearance called choked disc, is a result of intracranial pressure, and this may occur wherever be the seat of the tumor. Choked disc is an important aid in diagnosis. There may be descending optic neuritis. Atrophy of the optic nerve may be either secondary to the foregoing or a direct effect of the pressure of the tumor on the optic tract. The pupils are often dilated, and not infrequently they differ in size and respond feebly to light. With reference to the localization of tumors the reader is referred to the article on the topical diagnosis of cerebral lesions. (Vide p. 672.) It cannot be claimed, on the basis of clinical experience, that, by means of the so-called topical symptoms, tumors or other lesions can be always accurately localized. Aside from the imperfections of our present knowledge of the functions of the different parts of the brain, their relations with each other are such that a lesion in a particular situation may affect other situations more or less remote, and the parts thus secondarily affected may give rise to prominent symptoms. Nevertheless, experimental physiology and clinical observations have furnished facts which have led to some approximation to accuracy in localization.

As regards DIAGNOSIS, abscess of the brain may present a series of clinical events which render it difficult thereby to differentiate this affection from tumor. If the affection have speedily followed injury of the head, if it be connected with otitis, or if there be caries of the skull in any situation, the affection is probably abscess. If these etiological relations be wanting, tumor is more probable than abscess. Cerebral hemorrhage and softening from thrombosis or embolism are distinguished by this fact—namely, paralysis with or without an apoplectic seizure occurs suddenly without prodromes, whereas paralytic and other marked effects of tumor occur in succession and after prodromic symptoms which have existed for a longer or shorter period. In cases of hemiplegia from the affections just named nerves of the special senses and other cranial nerves are rarely involved, as they often are in cases of hemiplegic paralysis from tumor. Paralysis of the face on one side and of the limbs on the other side (alternate paralysis) is more likely to occur from tumor than from hemorrhage or softening.

With respect to the character of the tumor in individual cases, carcinoma is probable if the patient be of middle or advanced age, if cancer be hereditary, and if it exist elsewhere. The evidence afforded by the symptoms of a rapid growth of the tumor is in favor of the conclusion that it is carcinomatous. The tuberculous character of the tumor is to be inferred from the existence of tuberculous disease in the lungs and other organs, and from the youth of the patient. Tumors of this kind rarely occur after puberty or at less than three years of age. The syphilitic character of the tumor is rendered probable by the fact that the patient has had syphilis and by the absence of evidence of carcinoma and tubercle. Aneurismal tumors rarely occur except in persons between fifty and sixty years of age. Where no data exist as to the variety of tumor, glioma is to be inferred, as being the most common form of cerebral tumor not referable to syphilis or to tuberculosis. Cerebral tumors are more common in males than in females.

As regards digestion, nutrition, and the functions of the body in general, cases differ. Patients may retain their weight and an aspect of health for a considerable period after the occurrence of paralysis and other symptoms referable to the brain. In other cases debility and emaciation occur early. The general condition will be likely to deteriorate speedily in the cases in which the tumor is either carcinomatous or tuberculous, parts other than the brain being the seat of the disease. It is to be added that the tumors within the skull may remain latent, as regards both local and general symptoms, until they have attained a considerable size. The slowness of their growth favors their latency. On the other hand, local morbid conditions incident to the growth of the tumors—namely, inflammation and softening of the surrounding brain-substance, congestions, circumscribed meningitis, and extravasation of blood—give rise not only to paralysis affecting different parts in succession and progressively increasing in degree, but to exacerbations of pain and other cerebral symptoms varying much in frequency and intensity in different cases—to somnolency or coma in some cases, either of transient duration or lasting for several days, and occasionally to active delirium.

The termination in most cases of tumor within the skull is, sooner or later, fatal, the duration of life varying much in different cases according to the nature of the tumor, the rapidity of its growth, and the incidental local morbid conditions. In some cases death takes place after a few months, and in other cases not until after many years. An exception to this statement is to be made in behalf of syphilitic tumors. These are amenable to curative treatment if they have not attained to much size and have not led to damage of the cerebral substance in proximity to them. Death may take place after gradual exhaustion; it may occur suddenly from cerebral hemorrhage; epileptiform convulsions sometimes determine the fatal termination; and patients sometimes die from secondary meningitis.

Syphilitic tumors offer much encouragement in the way of successful medicinal treatment. Cases in which are presented hemiplegia, paralysis of cranial nerves, aphasia, epileptiform convulsions, and delirium are by no means hopeless if the affection be traced to syphilis. I have met with instances successfully treated in which these symptoms were present. It is very important to resort to antisyphilitic treatment in all cases in which there is any ground for suspecting the previous existence of syphilitic disease.

The indications for the treatment of non-syphilitic tumors relate to the palliation of symptoms and the prolongation of life by analeptic and supporting measures. It is not certain that any known remedies have a special influence upon the tumors themselves or the brain lesions which they produce. It is, however, claimed that certain remedies—namely, the iodide of potassium, the bichloride of mercury, and arsenic—do have such an influence in some cases. Trial may be made of these remedies, taking proper precautions as regards a deleterious effect upon the general condition of the patient.

Cerebral Syphilis.

Although in previous articles mention has been made of various syphilitic affections of the brain, nevertheless, for the sake of clearness and in view of the frequency and importance of these affections, it is thought best to bring them together in a separate article.

Cerebral syphilis may be a manifestation of congenital or of acquired syphilis. In the majority of cases it is one of the late, tertiary lesions, occurring sometimes ten to twenty years after the primary infection. Cerebral syphilis in the secondary stage of syphilis is rare.

The two leading syphilitic lesions of the brain are gummata and obliterating endarteritis.

Intracranial gummata originate usually either in the dura mater or in the pia mater. They may develop in the cranial bones. Gummata originating in the substance of the brain are very rare. They then develop primarily in the walls of the blood-vessels. At first a gumma is composed simply of soft, grayish granulation-tissue, but in a short time central caseation and fibroid metamorphosis occur, producing a mass of firm consistence and opaque yellowish color. The gumma may develop as a circumscribed nodular tumor or as a diffuse growth. In either case there is usually more or less diffuse inflammatory thickening of the adjacent membranes, so that the term syphilitic or gummatous meningitis appropriately describes the lesion. There may be a single gumma or many gummata in the membranes. It is important to note that gummata, although almost invariably starting in the cerebral membranes, frequently grow into the substance of the brain, which they compress and destroy.

Obliterating endarteritis of the cerebral arteries is a common syphilitic lesion, but it is not peculiar to syphilis. The arteries at the base of the brain are the ones oftenest affected. The affected arteries are hard, of an opaque whitish color, and thickened. The change consists in a thickening of the intima due to a new growth of connective tissue. The thickening may affect the circumference of the artery symmetrically or be most marked at one point in the circumference. The artery may become entirely obliterated, either by the continued growth of the new connective tissue or by thrombosis. Small gummata are frequently found in the media and the adventitia of arteries which are the seat of syphilitic endarteritis. Small aneurisms may form as the result of the arterial disease. The occlusion of the arteries, especially of those of the base which belong to the terminal arterial system, leads to softening of the region of the brain supplied by the obstructed vessel.

Finally, it may be mentioned that syphilitic disease of the arch of the aorta, or syphilitic endocarditis or myocarditis attended by the formation of thrombi, may give rise to embolic cerebral softening. Cases of cerebral syphilis without any coarse lesions in the brain have been described, but here it is probable that the small arteries are diseased.

From this description of the syphilitic lesions of the brain it is evident that the SYMPTOMS are manifold and variable in individual cases, so that it is impossible to give a clinical description applicable to all cases. The following may be mentioned as some of the common types.

The grouping of symptoms in cases of gumma may be that of other forms of cerebral tumor. When the gumma is seated at the base of the brain, there are affections of various cranial nerves, such as facial paralysis, neuralgia and paralysis of the trigeminus, paralysis and twitching of the eye-muscles; disorders of vision, such as diplopia, hemianopsia, amaurosis, irregularity of the pupils; sometimes impairment of hearing, of smell, or of taste; and rarely paralysis of the hypoglossal nerve Involvement of the crus cerebri causes hemiplegia. Choked disc is the rule. A gumma involving the motor cortical zone causes monoplegia or hemiplegia, usually accompanied with motor spasms and frequently with epileptic convulsions. When the centres for speech are affected there is aphasia. (The previous article may be consulted for other symptoms of syphilitic cerebral tumor.)

In a second group of cases the symptoms are referable to softening of the brain caused by occlusion of one of the cerebral arteries. These symptoms do not differ from those of ordinary thrombic softening.

In a large group of cases the symptoms are not those of a focal disease, as in cases of tumor and of softening, but belong to some chronic diffuse disorder of the brain. Here the clinical history may resemble that of chronic cerebral meningitis, of multiple cerebral sclerosis, of chronic insanity, or, in women, of hysteria.

The DIAGNOSIS cannot be made from the cerebral symptoms alone, although the transitory nature of many of the symptoms, their rapid development, and amelioration are in a certain degree characteristic. The diagnosis must be based upon a syphilitic history and the recognition of manifestations of syphilis in other parts of the body. The ophthalmoscopic examination may aid in the diagnosis, as, in addition to choked disc, a syphilitic choroiditis has been observed in some cases of cerebral syphilis.

Cerebral syphilis is often markedly amenable to TREATMENT. If, however, parts of the brain have been seriously impaired in their structure by syphilitic lesions, it cannot be expected that these parts will be restored, so that in a considerable number of cases the termination is unfavorable.

The treatment consists in the use of mercury and of iodide of potassium, according to the recognized principles of antisyphilitic therapeutics.

CHAPTER IV.

INFLAMMATORY AND STRUCTURAL DISEASES OF THE SPINAL CORD.

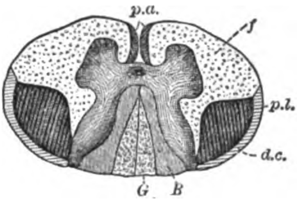
General Considerations relating to Inflammatory and Structural Diseases of the Spinal Cord.—Myelitis, Acute and Chronic.—Cerebro-spinal Sclerosis.—Locomotor Ataxia, or Posterior Spinal Sclerosis.—Hereditary Ataxia.—Acute Anterior Poliomyelitis.—Acute Anterior Poliomyelitis in the Adult.—Subacute and Chronic Anterior Poliomyelitis.—Progressive Muscular Atrophy.—Progressive Unilateral Facial Atrophy.—Spastic Spinal Paralysis.—Amyotrophic Lateral Sclerosis.—Compression of the Spinal Cord.—Intraspinal Tumors.—Pseudo-hypertrophic (or Myosclerotic) Paralysis.—Scleroderma.—Myxœdema.

General Considerations Relating to Inflammatory and Structural Diseases of the Spinal Cord.

IN order to understand the localization of certain of the diseases of the spinal cord which are to be described in this chapter, it is necessary to know the situation of certain bundles of nerve-fibres entering into the composition of the white columns of the cord. The accompanying diagram (Fig. 5) represents these bundles upon a transverse section of the cord at the level of the sixth cervical nerve.

In the anterior column of each half of the spinal cord adjacent to the anterior median fissure is a narrow tract of nerve-fibres, marked *p. a.* in the diagram, called the pyramidal fasciculi or pyramidal tracts of the anterior column. These bundles are called also the uncrossed or direct pyramidal tracts and the columns of Türck. They may be absent in normal cords. In the posterior part of the lateral column are situated the pyramidal fasciculi of the lateral columns or the crossed pyramidal tracts, marked *p. l.* in the diagram. The pyramidal tracts are composed of motor-fibres which originate in the central convolutions of the brain, descend through the centrum ovale to the posterior division of the internal capsule (see p. 676), pass thence through the pes pedunculi, the pons, and the anterior pyramids of the medulla oblongata to the crossing of the pyramids, where the majority of the motor-fibres cross over and descend in the pyramidal tracts of the lateral columns of the cord, while a smaller but variable portion remains uncrossed and descends in the pyramidal tracts of the anterior columns of the spinal cord. The latter pyramidal tracts cross in their course through the spinal cord, so that eventually all of the motor-fibres of the pyramidal tracts which originate in one cerebral hemisphere cross to the opposite half of the spinal cord. The fibres of the pyramidal tracts are connected with the ganglion-cells of the anterior horns of the spinal cord. From these ganglion-cells originate the anterior nerve-roots. When the pyramidal tracts are destroyed in any part of their course, as by a hemorrhage in the posterior division of the internal capsule, they become the seat of a descending degeneration which can be traced from the seat of the lesion downward to the termination of the tracts.

FIG. 5.



After Eichhorst.

Between the pyramidal tracts of the lateral columns and the periphery of the cord are seated the direct cerebellar fasciculi, marked *d. c.* The remaining parts of the anterior and of the lateral columns are called the fundamental fasciculi of the antero-lateral column or the anterior root-zone (*f.* in the diagram).

Two divisions are recognized in the posterior columns—namely, the triangular columns on each side of the posterior median fissure, called the funiculi graciles, or columns of Goll (*G.* in the diagram), and the wedge-shaped columns between these and the posterior horns, called the funiculi cuneati, or columns of Burdach (*B* in the diagram).

The columns of Goll and the direct cerebellar fasciculi contain centripetal nerve-fibres, and are the seat of an ascending degeneration above any lesion which destroys their continuity.

The pyramidal tracts diminish in size from above downward, and in the lower dorsal region the uncrossed pyramidal tracts disappear. In the lumbar and the lower dorsal regions the division of the posterior columns into the columns of Goll and the columns of Burdach cannot be made out. The direct cerebellar fibres make their first appearance in the lumbar enlargement, and increase in size from below upward.

The divisions of the white columns of the cord which have been described are based chiefly upon the study of embryonic cords and of the secondary degenerations of the cord. These divisions are of importance, because each one represents a group of functionally distinct nerve-fibres, which may be the seat of some special disease.

Two great classes of diseases of the spinal cord are recognized—namely, the *systemic diseases* or *system-diseases*, and the *unsystemic*, *mixed*, or *diffuse diseases*.

The system-diseases of the spinal cord are characterized by the fact that the pathological lesions are confined to certain definite tracts or parts of the cord which represent distinct physiological systems or groups of nerve-fibres or of ganglion-cells. To the category of system-diseases belong the affection of ganglion-cells in the anterior horns, called poliomyelitis anterior; the sclerosis of the posterior columns constituting the essential lesion of locomotor ataxia; the degeneration of the pyramidal tracts, either alone as in secondary descending degenerations, or in combination with anterior poliomyelitis in amyotrophic lateral sclerosis; and the secondary ascending degenerations of the columns of Goll and of the direct cerebellar fibres. We must assume that in the system-diseases the special cause of the disease acts primarily upon the nerve-elements, and that the interstitial tissue is secondarily involved.

In contrast with the system-diseases, the lesions in the unsystemic or diffuse diseases are not confined to any definite group of nerve-elements, but affect irregularly either a large or a small part of the spinal cord. To this second class of diseases belong hemorrhages, tumors, the transverse, the diffuse, and the disseminated forms of myelitis, embolic or thrombic softening, and other affections.

Before considering the individual diseases of the spinal cord it will save repetition and assist in a clearer understanding of these diseases if the important symptoms of various affections of the cord be first considered with reference to certain peculiarities, and especially to their topical diagnosis.

An important distinction is made between two kinds of paralysis. In one variety of paralysis the paralyzed muscles rapidly undergo atrophy and assume the so-called reaction of degeneration. This variety may be called atrophic paralysis. In the second kind of paralysis the paralyzed muscles waste only after a long period from disuse, and they retain their electrical excitability as long as muscular substance is preserved.

The *atrophic form of paralysis*, attended with the reaction of degeneration, is always due either to disease of the ganglion-cells in the anterior horns of the spinal cord or to an interruption of the connection between these ganglion-cells and the muscles; that is, to diseases of the peripheral nerves. If the paralysis be due to any lesion of the motor tracts from their origin in the cerebral cortex to their termination in the ganglion-cells of the anterior horns of the spinal cord, there will be no rapid atrophy of the paralyzed muscles and no reaction of degeneration. The explanation of this difference is found in the fact that the ganglion-cells of the anterior horns exert some unexplained trophic influence upon the muscles with which they are in connection. A peculiarity in cases of paralysis due to disease of these ganglion-cells or to disease of the peripheral nerves is that the affected nerves from the seat of lesion and the paralyzed muscles undergo rapid degeneration; a degeneration, however, which may be recovered from if the primary lesion be repaired before the degeneration has advanced too far.

Another important difference is, whether the paralyzed muscles be relaxed and flabby or in a rigid, spastic condition. *Spastic paralysis* is generally associated with increased reflex action, and it occurs when certain inhibitory fibres which come from the brain and run in the lateral columns are interrupted by some lesion in their continuity. It is assumed by many that these inhibitory fibres are identical with those in the pyramidal tracts of the cord; but this view is not proven. For the production of spastic paralysis it is necessary also that the nerve-fibres and ganglion-cells which conduct and originate impulses for reflex action of the paralyzed muscles should not be destroyed. The conditions for spastic paralysis are present in many cases of cerebral paralysis, in most cases of transverse myelitis or compression myelitis in the upper dorsal and the cervical regions, and in sclerosis of the lateral columns of the cord. Spastic paralysis does not occur when the centripetal reflex fibres in the posterior nerve-roots, posterior columns, and gray matter, the ganglion-cells of the anterior horns which transmit reflex impulses, and the centrifugal reflex fibres of the anterior nerve-roots are diseased so as to arrest their functions.[1]

Loss of sensation is a symptom of spinal disease when the function of the sensory fibres in the cord is destroyed or impaired. The sensory fibres enter the cord by the posterior nerve-roots, from which a part enter directly into the posterior horns and a part pass into the posterior columns. The sensory fibres of the posterior columns probably also enter the posterior horns at some point distant from the entrance of the corresponding nerve-roots. Unlike the majority of the motor fibres, the sensory fibres decussate in the spinal cord. Loss of sensation dependent upon spinal affections, therefore, points to some disease of the posterior columns or of the posterior horns of gray matter, more particularly to disease of the latter. It is an interesting fact that tactile sensation may be lost while the sense of pain is preserved. The loss of sensation to pain is called analgesia. If a lesion destroy only one-half of the spinal cord—which is a very rare occurrence—the paralysis of motion will be on the side of the lesion, and the paralysis of sensation (tactile and for pain) will be on the opposite side.

Conditions affecting *reflex action* have already been mentioned in connection with spastic paralysis. Reflex action is increased if the connection between the reflex centres and the brain be interrupted. The reflex centres are situated in the gray matter of the cord. Destruction of the gray matter, therefore, abolishes the reflex action of those muscles which are in nervous connection with the diseased part of the gray matter. A similar effect is produced by disease of centripetal nerve-fibres which carry impressions from the periphery to these centres, as in the case of disease of the posterior columns and posterior horns of gray matter in locomotor ataxia. The two most important forms of reflexes for diagnostic consideration are cutaneous reflexes and tendon reflexes. *Cutaneous reflex* is generally studied by noting the contraction of the muscles of one of the lower extremities upon tickling the sole of the foot. Other cutaneous reflexes which may be examined are the contraction of abdominal muscles upon passing the finger along the skin of the abdomen or the contraction of the cremaster muscle upon passing the finger along the upper part of the inside of the thigh.

Tendon reflexes are examined at the knee and at the ankle. In most healthy individuals a vigorous contraction of the quadriceps extensor muscle is produced by striking quickly with the ulnar side of the hand or with a percussion hammer the ligamentum patellæ when the leg is flexed and the muscles are relaxed. This contraction, called the *knee-jerk* or *patellar reflex*,[2] may be increased or may be diminished or abolished in certain diseases of the spinal cord. The patellar reflex may be impaired even when cutaneous reflexes are intact. The centre for the patellar reflex is in the gray matter of the lumbar portion of the spinal cord. Diseases of the gray matter in this situation, and also disease of the centripetal nerve-fibres in the posterior columns of the lumbar enlargement cause the patellar reflex to disappear. In the various conditions causing spastic paralysis the patellar reflex is increased.

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[1] It is the rule, therefore, that disease of the posterior columns and of the gray matter extending over a considerable longitudinal extent of the spinal cord prevents the occurrence of spastic paralysis. An apparent exception is amyotrophic lateral sclerosis; but here it must be assumed that at the time of spastic paralysis the ganglion-cells of the anterior horns are not diseased sufficiently to prevent the transmission of reflex impulses. In the explanation of spastic paralysis some writers lay more stress upon the irritation of certain nerve-fibres and ganglion-cells than upon the abolition of inhibitory impulses. It is probable that both irritative lesions and the withdrawal of the inhibitory influence of the brain may cause spastic paralysis with increased reflexes.

[2] It is assumed that the knee-jerk is a reflex phenomenon, although this view is not proven.

The so-called *foot-clonus*, *ankle-clonus*, or foot-phenomenon is produced by making passively a vigorous dorsal flexion of the foot, when, if the clonus be marked, the foot, upon relaxation of the flexion, will assume a position of extension in consequence of contraction of the gastrocnemius muscle. There may follow repeated movements of flexion and of extension, so that there is a continuous rapid vibration of the foot from one position to the other. This reflex, produced by irritation of the tendo Achiilis, is more frequently absent in health than the knee-jerk, but in spastic paralysis it is often a marked phenomenon.

The use of *electricity* has become not only a valuable therapeutical agent, but also a most important aid in the diagnosis and the prognosis of various forms of paralysis. For diagnostic purposes it is necessary to make use of both the induced (or faradic) current and the constant (or galvanic) current. The polar method of application of the electric current is the most exact. In this, one electrode is placed over the nerve or the muscle to be irritated, and the other electrode at some distant part of the body, as the sternum or the nape of the neck. The galvanic battery should be supplied with a commutator, so that either electrode can be made positive or negative at will.

In order to understand the electrical reactions in disease it is necessary to be familiar with the so-called *law of contraction* for motor nerves and muscles in health. This law relates to the responses to the constant current applied by the polar method. This current produces muscular contractions only when the circuit is opened or is closed. The occurrence and the strength of these contractions depend upon the direction and the strength of the electrical current. If the constant current be very weak, no contraction is produced. If the strength of the current be gradually increased, the first contraction appears upon closing the descending current; and this is called the cathodal closure-contraction (C. C. C.), the cathode or negative pole being applied to the nerve or muscle when the circuit is closed. If the strength of the current be still further increased, the next contractions appear upon closing and upon opening the ascending current, sometimes one and sometimes the other causing the first response. These contractions are the anodal closure-contraction (A. C. C.) and the anodal opening contraction (A. O. C.), the anode or positive pole being applied to the nerve or muscle. Contraction upon opening the descending current or cathodal opening contraction (C. O. C.) appears last, and not until the current is so strong as to cause tetanic cathodal closure-contractions. The galvanic current therefore causes contractions in the following order: 1, C. C. C.; 2 and 3, A. C. C. and A. O. C., sometimes one coming first, and sometimes the other; 4, C. O. C. The main points to remember are that in health the C. C. C. exceeds the A. C. C., and the A. O. C. exceeds the C. O. C. The contractions occur in the same order whether the electrode be applied to the nerve or to the muscle.

The faradic current applied to nerve or to muscle in health causes tetanic spasm of the muscle, the intensity of the spasm being in proportion to the rapidity of the interruptions and the strength of the current.

The changes in electrical reactions in disease may be quantitative or qualitative. The *quantitative changes* consist in simple increase or simple diminution of electrical response. Simple increased electrical excitability is common in the early stages of various forms of paralysis; but when unaccompanied with qualitative changes in electrical reaction it is not of much diagnostic importance. So, too, diminished electrical response, when of diagnostic import, is generally, although not necessarily, accompanied with qualitative changes in muscular excitability.

Far more important than the simple quantitative changes are the combined quantitative and qualitative changes in the electrical excitability of nerve and of muscle which constitute the *reaction of degeneration*. This reaction is so called because it occurs only when the nerve or the muscle (or usually both) has undergone degeneration. The degeneration of the nerve is indicated by a diminution, and finally a disappearance, of its excitability both to the faradic and the galvanic currents. In cases of peripheral paralysis the electrical excitability of the nerve may be completely lost in from one to two weeks, so that it is impossible by electrical excitation of the nerve to produce any muscular contraction. During this time the reaction of the muscle to the direct application of the faradic current also diminishes, and finally disappears. This is due to degeneration of the intramuscular nerve-fibres. The reaction of the muscle to the direct polar application of the galvanic current undergoes important alterations, which indicate degeneration of the muscle, going on at the same time as nerve-degeneration. At an early stage of the degeneration there may be slight diminution in the galvanic excitability of the muscle, but this soon gives place to a decided and characteristic increase in the response to the galvanic current when directly applied to the muscle. This increased response is attended by certain noteworthy peculiarities. The first peculiarity is, that the anodal closure-contraction (A. C. C.) increases, so that after a while it equals, or even exceeds, the cathodal closure-contraction (C. C. C.), thus reversing the conditions in health. The cathodal opening contraction is also often increased, so that it may equal or exceed the anodal opening contraction. A second peculiarity is, that the muscular contractions, instead of being immediate in their appearance and short, are sluggish and prolonged, sometimes continuing during the entire duration of the current, instead of only at the opening or the closing of the circuit.

If the degeneration go on to complete destruction of the muscular structure, the increased galvanic excitability of the muscle gives place to diminution and final disappearance of all electrical excitability, the last to disappear being the anodal closure-contraction.

If repair of the degenerated muscular and nervous tissue occur, the exaggerated and altered galvanic muscular reaction gives place to the normal reaction, faradic excitability of the muscle returns, as well as the galvanic and the faradic reaction of the nerve. The nerve becomes capable of transmitting the impulses of the will, causing volitional contractions, before the normal faradic and galvanic excitability is restored.

Usually, both the nerve and the muscle simultaneously undergo degeneration. If the nerve be alone degenerated, there will be simple diminution or loss of its faradic and galvanic excitability and diminution or loss of the faradic muscular excitability, without qualitative change in the galvanic muscular excitability. If the muscle be alone degenerated, the faradic and the galvanic excitability of the nerve and the faradic excitability of the muscle will be preserved, while the above-described characteristic changes following direct galvanic excitation of the muscle are present. The latter reaction is sometimes called partial reaction of degeneration.

The reaction of degeneration is present only in paralysis due to disease of the peripheral nerves and in that due to disease of the ganglion-cells in the anterior horns of the spinal cord. It is absent in all forms of cerebral paralysis, in all spinal paralyses due to disease of the white matter of the cord, and in functional paralyses. If paralysis be due to disease involving a segment of the gray matter of the spinal cord, as in transverse myelitis, the reaction of degeneration is present only in the nerves and muscles which are connected with that segment, while it is absent in the paralyzed muscles whose nerves originate below the diseased segment. It is thus apparent that the study of the electrical phenomena of nerve and of muscle in cases of paralysis affords most important information as to the seat of disease. The reactions to electricity also indicate the pathological condition of a nerve or of a muscle; and this may prove of great assistance in forming a judgment as to the possibility of recovery.[1]

Myelitis.

Inflammation of the substance of the cord is called *myelitis*. Myelitis appears as an *acute* and as a *chronic* affection. These two forms, however, cannot be sharply discriminated in all cases, since acute myelitis often passes into the chronic form. Other divisions are based upon the extent of the inflammation and the part of the cord involved. Thus the inflammatory changes may be either circumscribed, diffuse, or disseminated; the white matter, the gray matter, or both, may be affected; or the disease may tend to extend transversely or in a longitudinal direction. When both white and gray matter of the greater portion of the cord are inflamed, the process is called *diffuse myelitis*; when the inflammation affects only or chiefly the gray matter, the disease is *central myelitis;* and when confined to the anterior horns of gray matter, it is *anterior poliomyelitis* or *anterior cornual myelitis*. In *transverse myelitis* the whole thickness of the cord over a circumscribed space is involved. *Cortical myelitis* or *meningo-myelitis* is the name given to inflammation of the peripheral portion of the cord, the inflammation being consecutive to spinal meningitis. When a number of separate foci of inflammation exist in the cord the change is designated as *disseminated* myelitis. Myelitis extending from below upward is called *ascending*, and extending from above downward it is called *descending*.

As regards the tissue primarily affected, Charcot, following Virchow's classification, distinguishes two varieties of myelitis—namely, *interstitial* and *parenchymatous myelitis*. In interstitial myelitis the connective tissue or neuroglia is first inflamed, and the nerve-elements suffer secondarily; and in the parenchymatous form the ganglion-cells and nerve-fibres are primarily involved, and the interstitial changes are secondary. The final result in both cases is the same.

The gray substance of the cord, on account of its greater vascularity and functional importance, is more frequently the seat of primary myelitis than is the white matter. Inflammation of the gray matter is particularly prone to extend in the vertical direction (central myelitis).

Acute myelitis, in the majority of cases, appears under the form of softening of the cord. Chronic myelitis, on the other hand, appears as induration or sclerosis. The changes which characterize acute myelitis will be described first, and afterward those of the chronic form. Softening and acute myelitis are not to be regarded as synonymous, since there exists non-inflammatory as well as inflammatory softening of the cord. Exceptionally, the consistence of the acutely inflamed cord is found to be normal or even increased. The color also varies. It may be that of the normal cord, in which case, if the consistence be unchanged, the existence of inflammation can be determined only by microscopical examination. Most frequently the color is reddish or yellow, but in cases of prolonged duration it may be grayish or white. The red color is due to the presence of blood. In very acute cases of central myelitis the softened part may contain so much extravasated blood as to present the appearance of a primary hemorrhage into the cord. The name *hæmato-myelitis* is given by Hayem to cases of acute myelitis attended by extensive hemorrhage. From the sudden development of the symptoms, it is also called *apoplectiform myelitis.* The stage of red softening gradually gives place to that of yellow softening. The yellow color is due to alterations in the blood-pigment, to fatty degeneration, and to the presence of pus-cells and granular corpuscles. The affected portion of the cord is usually, but not always, swollen. The pia-arachnoid membrane is, as a rule, involved to some extent in the inflammatory process. The dorsal and the lumbar regions of the cord are the favorite seats of the disease.

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[1] For further information on this subject consult the excellent work of A. Hughes Bennett, *A Practical Treatise on Electro-diagnosis in Diseases of the Nervous System*, London, 1882.

The microscopical examination in cases of acute myelitis reveals dilatation of the blood-vessels, extravasated red blood-corpuscles, granular corpuscles, fat-granules, pus-cells, disintegrated nerve-fibres and nerve-cells, swollen and apparently proliferating neuroglia-cells, granular material, especially around the vessels (regarded by Lockhart Clarke as coagulated albuminous exudation, and called by him "granular disintegration"), blood-pigment, and corpora amylacea. In the early stage, and in the neighborhood of softened foci in the later stages, the axis-cylinders of some of the nerve-fibres are much swollen, presenting the appearance called varicose or fusiform hypertrophy. The ganglion-cells also may be found swollen, spherical, pigmented, vacuolated, and devoid of processes. They finally undergo atrophy and disappear. The recognition of the granular corpuscles (also called fat-granule bodies or inflammatory corpuscles of Glüge) is of great assistance in distinguishing ante-mortem softening from the softening so often produced artificially in removing the cord. These corpuscles are usually spherical or oval in shape, vary greatly in size, and are so densely filled with small fat-globules as to appear opaque by transmitted and bright by reflected light. They may be formed from emigrated white blood-corpuscles, from neuroglia-cells, from cells in the walls of the blood-vessels, and perhaps in other ways. They are not distinctive of inflammation, and they may be present in simple degenerative processes.

If acute myelitis pass into the chronic stage, the red and the yellow give place to white softening. Cysts filled with a whitish, milky fluid may be formed as in cerebral softening. By absorption of the inflammatory exudation, by atrophy of the nerve-elements, and the production of fibrillated connective tissue acute myelitis may terminate in sclerosis, as has been demonstrated by Leyden. It is probable that very mild cases of myelitis may be followed by complete restoration to the normal condition. Certain observations tend to show that nerve-fibres in the cord which have been destroyed by the myelitic process may be regenerated. Suppuration or abscess is an extremely rare termination of acute myelitis, and when present it is either pyæmic or consecutive to suppurative processes in the membranes, vertebræ, or surrounding parts.

Transverse myelitis of sufficient duration (probably at least a month) is followed by the secondary degenerations of Türck. These degenerations affect both centripetal and centrifugal fibres. They are therefore ascending, and when the myelitic focus does not reach the lower extremity of the cord, descending. The ascending degeneration occupies the columns of Goll and the direct cerebellar fasciculi; and the descending degeneration follows the course of the pyramidal fasciculi of the anterior and of the lateral columns. These tracts, when degenerated, appear gray and are finally atrophied, the process consisting in a gradual atrophy of the nerve-fibres combined with increase of the neuroglia and the appearance of granular corpuscles. When the myelitis occupies the whole thickness of the cord the degenerations are bilateral. Myelitis confined to one-half of the cord is very rare.

Chronic myelitis includes, according to common usage, the processes also called sclerosis and gray degeneration. It is characterized by proliferation of the neuroglia-elements, resulting in the production of fibrillated connective tissue and the atrophy of nerve-elements. Granular corpuscles and corpora amylacea may also be present. The consistence of the affected part is usually firm, its size is diminished rather than increased, and its color is gray. Sometimes there are no morbid changes visible to the naked eye. As regards their clinical history, certain cases of slowly-developing softening of the cord are to be referred to chronic myelitis, although Leyden considers all instances of inflammatory softening of the cord as acute in their origin.

Chronic myelitis embraces affections of the cord which vary greatly as to the situation, extent, and nature of the pathological process. Certain forms have an exact localization, and follow the course of certain definite groups of nerve-fibres and of nerve-cells. Others are characterized by irregularity in distribution and extent. Of the forms which have more or less of an exact localization may be mentioned posterior spinal sclerosis or locomotor ataxia, amyotrophic lateral sclerosis, and anterior poliomyelitis. These will each receive separate consideration. The secondary degenerations of Türck, although regarded by many writers as the result of chronic myelitis, are primarily degenerative. The name *funicular sclerosis* is given to sclerosis following certain definite columns of the cord, such as lateral and posterior spinal sclerosis. It is probable that in these cases the primary change is in the nerve-fibres. Otherwise it would be difficult to understand why inflammation should be confined to the course of definite nerve-tracts. The most important examples of chronic myelitis with irregular distribution are—chronic transverse myelitis, circumscribed sclerosis, and disseminated or multiple sclerosis. Multiple sclerosis will be considered separately, as it has a well-defined clinical history. Myelitis resulting from slow compression (angular curvature resulting from Pott's disease, cancer of the vertebræ, or intraspinal tumors) is usually of the ordinary transverse variety and accompanied by secondary degenerations.

The CLINICAL HISTORY of *acute myelitis* presents variations corresponding to those which belong to the anatomical characters of the disease. The latter furnish the interpretation of the former, and from the symptoms the seat of the inflammation, its extent, and the particular parts of the cord which are involved may be inferred with considerable accuracy. Certain symptoms belong alike to acute spinal meningitis and to acute myelitis. In many if not in most cases these two affections are associated. In meningitis the inflammation extends from the membranes to the cord, and in myelitis the membranes are likely to be secondarily involved. Practically, at the bedside, having diagnosticated an acute intraspinal inflammation exclusive of intracranial inflammatory diseases, the diagnostic problem which remains is to differentiate myelitis from meningitis; that is, to decide that the inflammation has its origin in and is predominant in the cord. The symptoms, therefore, which distinguish myelitis from spinal meningitis especially claim attention.

Disorders of sensation are often the first local symptoms. These are pain in the back, the sensation of a girdle around the trunk, and tenderness over certain of the vertebræ. Preceding paralysis the muscles which are to be paralyzed are sometimes affected with tremor or spasms.

The early occurrence of paralysis is a distinguishing feature. It occurs within a day or two, and sometimes within a few hours, after the symptoms have pointed to a spinal affection. In most instances the form of the paralysis is paraplegic, showing the seat of the affection to be in the dorsal or in the lumbar portion of the cord. According to the seat at the outset or the subsequent extension of the inflammation upward (ascending myelitis), the muscles of respiration and of the upper extremities may be affected. The speedy occurrence of paralysis is evidence that the cord has been primarily affected. Sudden and complete paralysis denotes that the myelitis is central and accompanied by hemorrhage (hæmato-myelitis).

Paralysis affecting the bladder and rectum is more constant and occurs earlier in myelitis than in spinal meningitis. Loss of cutaneous sensibility is more frequent in the former than in the latter. It is rare for sensory paralysis to be wanting. Its absence shows that the myelitis does not involve the posterior columns or the posterior cornua of the cord. Sensation may not be lost, but is more or less diminished, and it may be characterized by retarded transmission of sensory impressions. Exceptionally, there is cutaneous hyperæsthesia, this being attributable to complicating meningitis. Priapism is a symptom of not infrequent occurrence. Symptoms relating to reflex movements are of significance as regards the localization and the extent of the lesions. The continuance or increase of the reflex movements shows either that disorganizing changes have not taken place, or that the disorganization has not extended to the lower end of the spinal cord. On the other hand, loss of reflex excitability is an effect of destruction of the gray matter in the lower part of the cord. This extension is shown by the loss of reflex excitability after the occurrence of the motor paralysis.

Spasm of the muscles of the lower extremities, constituting the so-called spinal epilepsy of Brown-Séquard, is a prominent symptom in some cases of transverse myelitis not involving the lower lumbar region.

Notable wasting of the paralyzed muscles is indicative of destruction of the gray matter; and in this case the reaction of degeneration appears in the muscles the nerves of which originate in the diseased gray matter. Hence this reaction is absent in the muscles of the lower extremities in dorsal and cervical myelitis, and is present in lumbar myelitis involving the anterior horns.

In myelitis, earlier and in a greater degree than in spinal meningitis, the parts exposed to pressure—sacrum and trochanter—take on gangrenous inflammation. Not infrequently this occurs within a few days after the attack. The urine is retained and within a short period it becomes alkaline. The presence of muco-pus shows the existence of cystitis.

Fever in cases of acute myelitis is rarely high, and is generally of a low grade. The propriety of the term acute in this connection has reference to the rapid development of grave symptoms, rather than to the elevation of temperature and other evidence of constitutional disturbance. Fever symptomatic of gangrenous inflammation (septic fever) and of cystitis occurs in the progress of the disease.

Acute myelitis may run a rapid course, destroying life in a few days, or death may take place as a result of gangrenous inflammation or of cystitis after the lapse of several weeks. Complete recovery is rare. In a certain proportion of cases the disease becomes chronic. It may end in more or less complete atrophy of the cord, and patients may live indefinitely with a permanent paraplegia, the general health being good.

In the foregoing account of the symptomatology of acute myelitis reference has been had especially to the parts involved in the differentiation from acute spinal meningitis. These points have been already referred to in the account given of the latter disease. (Vide p. 702.) The affection to be noticed in connection with the functional affections of the spinal cord, called acute ascending paralysis (vide Chapter V.), closely resembles acute ascending myelitis; but in the latter affection disturbances of sensation and bladder and rectal symptoms are generally not marked. The symptoms which relate to reflex and electrical excitability, to the paralysis of the bladder and rectum, and to the occurrence of cystitis and of bed-sores at an early period, are sufficiently characteristic of acute myelitis as contrasted with so-called reflex and functional paralysis. Acute myelitis, the inflammation limited to the anterior horns of the gray matter of the cord (anterior poliomyelitis), constituting the affection known as infantile paralysis and an analogous affection in adults, will be considered separately in this chapter.

Acute myelitis is a rare disease, especially if cases of traumatic origin be excluded. Aside from injuries of the cord from fractures, contusions, penetrating wounds, etc., prolonged exposure to cold, as in sleeping upon damp ground, seems to be the only cause clearly apparent. The adequateness of other agencies supposed to be causative, such as muscular efforts, excessive venery, and exposure to a high temperature, is problematical. Acute myelitis is occasionally observed as a sequel of diphtheria and of other infectious diseases. The disease occurs when it cannot be traced to any causative agency.

The TREATMENT in the early stage of acute myelitis has for its objects diminution of the intensity of inflammation and the prevention of disorganization resulting therefrom. Dry cupping over the spine, and in full-blooded subjects the local abstraction of blood by cups or leeches, are indicated. The application of the ice-bag to the spine is recommended. Ergot and belladonna have been supposed to be useful by causing contraction of the arteries of the cord. Mercurialization, by giving calomel and also by inunction, is to be advocated in view of the danger and the frequently rapid progress of this disease, on the ground that the notions formerly held in regard to the antiphlogistic effect of mercury are not without some foundation, and that the evils of the remedy are of little moment, provided it have a favorable influence upon the inflammatory process, however small this influence may be.

It will often happen that the acute stage is too brief to afford much time for treatment with reference to the objects just stated. This may be inferred when complete paralysis has taken place. Revulsive applications to the spine may be employed, but, considering the liability to gangrenous inflammation, they should not be made in situations exposed to pressure. To obviate the effects of pressure is an important part of the treatment. Relieving the bladder of an accumulation of urine is important, as well as proper attention to the early symptoms of cystitis.

Absolute rest is an important part of the treatment. Brown-Séquard advises recumbency on the abdomen, but the continuance of this decubitus is hardly practicable. There is no difference of opinion as to the impropriety of the employment of electricity in acute myelitis. Strychnia is contraindicated.

Chronic myelitis, in the most comprehensive sense of the term, includes several distinct affections, among which are sclerosis of the posterior columns of the cord, multiple cerebro-spinal sclerosis, sclerosis of the lateral columns, anterior poliomyelitis, and progressive muscular atrophy. These will be considered in this chapter under separate headings. Exclusive of these affections, the term chronic myelitis denotes an inflammatory affection localized in a segment, or sometimes in more than one segment, and extending transversely more or less throughout the cord (transverse myelitis). The form of paralysis resulting therefrom is generally paraplegia; but if the cervical portion of the cord be the seat of the affection, general spinal paralysis is the result. In cases of the disease as thus restricted the clinical problem is similar to that in cases of the acute form—namely, to differentiate chronic myelitis from chronic spinal meningitis. Here, too, it is to be considered that in the latter affection the inflammation extends more or less from the meninges to the cord. and that in the former affection inflammation of the cord generally involves the meninges. The antecedent history is to be taken into account. Both chronic myelitis and chronic spinal meningitis may have been preceded by an acute form of disease, and of course the chronic has the same primary seat as the acute. If, therefore, the previous history show the acute disease to have been acute myelitis, the existing affection is chronic myelitis.

As regards the symptoms, they point to myelitis, instead of meningitis, in proportion as they represent more or less disorganization of the cord. Hence, completeness of the paralysis, sensory as well as motor paralysis, paralysis of the bladder and rectum, cystitis, wasting of the paralyzed muscles, and a tendency to gangrenous ulceration are diagnostic of myelitis rather than of spinal meningitis.

The practical importance of making this discrimination relates chiefly to the prognosis. The prospect of improvement and recovery is far better in cases of spinal meningitis than in cases of myelitis. In proportion as the symptoms denote disorganization of the cord (softening and secondary atrophy) the prognosis is unfavorable. As already stated, however, destruction of the cord, causing complete motor and sensory paralysis of the lower limbs, is not incompatible with long life and good general health. In the preceding editions of this work a case is cited in which complete paraplegia followed a fall of forty feet, and after death there was found atrophy of the cord below the twelfth dorsal vertebra. This patient lived more than fifty years after the accident, married, and had six children, amassed a little fortune by his industry, and finally died with double pneumonia. In another case, reported by Dr. Purple,[1] there was complete loss of motion and sensation below the fifth dorsal vertebra following an injury. In this case the patient, finding his lower limbs burthensome, persuaded a surgeon to amputate them near the hip-joint. The operation was wholly without pain and the stumps healed readily.

The measures of TREATMENT indicated in cases of chronic myelitis are rest as long as the symptoms denote an inflammatory condition, and moderate counter-irritation over the spine. Remedies given with a view to promote the absorption of morbid products—namely, the iodide of potassium and mercury—are not indicated; and severe counter-irritation—by the moxa, issues, setons, etc.—is of no use. Electricity may be useful in retarding atrophy of the paralyzed muscles. The application of water to the spine by sponging and compresses is to be recommended, but cold affusions and hydropathic packing are hurtful. Experience has shown the hurtfulness of thermal baths. Attention to the bladder is important. Indications pertaining to the general condition of the patient are to be met. If the patient be confined to the bed, precautions are to be taken against the effects of pressure.

Cerebro-spinal Sclerosis—Multiple or Insular Sclerosis.

Among the names for this affection may be mentioned multiple sclerosis, disseminated sclerosis, insular sclerosis of the brain and spinal cord, *sclérose en plaques disseminées*, cerebro-spinal sclerosis, and disseminated chronic interstitial encephalo-myelitis. The disease was recognized pathologically by Cruveilhier (1835) and by Türck (1855). From a clinical point of view it has been studied most thoroughly by Charcot and his pupils.[2] It is characterized anatomically by the development of many or of few nodules of sclerotic tissue in the central nervous system. These nodules are the result of a disseminated chronic interstitial inflammation. Charcot distinguishes, according to the situation of the nodules, three types of the disease—namely, the cerebro-spinal, the cerebral, and the spinal. The cerebro-spinal form is the most common type, its clinical history is best understood, and it is the one most readily diagnosticated. The spinal form, with our present knowledge as regards its symptomatology, is not readily distinguished from the ordinary forms of chronic myelitis.

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[1] *New York Medical Journal*, 1853.

[2] Charcot, *Gaz. des Hôpitaux*, 1868; Bourneville et Guérard, *De la Sclérose en Plaques disseminées*, Paris, 1869.

In the majority of cases the spots of sclerosis can be recognized by the naked eye. In exceptional cases they may be visible only microscopically. They are distributed irregularly throughout the spinal cord, medulla oblongata, pons varolii, cerebrum, and cerebellum. The sclerotic islands in the pons and medulla are rarely absent, and they give rise to characteristic symptoms. Their number in the cerebrum and cerebellum is greater in the white than in the cortical substance. In the cord they occupy, unsymmetrically, both white and gray matter, more frequently the former. They may be concealed in the interior, or, reaching the surface, they may be visible from the exterior. It is rare for the sclerosis to extend throughout the entire thickness of the cord. The nodules may be scattered at wide intervals or several may coalesce. The size of individual nodules may vary between microscopic dimensions and several inches in length. The color of the sclerotic spots is reddish-gray, the consistence, especially in the brain, firmer than normal, the cut surface smooth and somewhat translucent, and the borders irregular and in many cases well defined. The affected parts may be shrunken or of normal or even increased size. The membranes, especially the pia mater, may be thickened and opaque or they may be unchanged.

The microscopical examination of the gray sclerotic nodules reveals, as the essential change, increase of the interstitial tissue, together with atrophy of the nerve-fibres and nerve-cells. The interstitial tissue appears as a finely-fibrillated but dense connective tissue, mingled with finely granular material. Large stellate cells, called Deiter's cells or spider-cells, are often present. The walls of the blood-vessels are notably thickened, and are often infiltrated with fat-molecules and lymphoid cells. Granular corpuscles and corpora amylacea are usually present, but not in large number. In the sclerosed patches most of the nerve-fibres have disappeared,[1] those remaining being, for the most part, naked axis-cylinders, sometimes much hypertrophied. At the borders of the sclerotic nodules the neuroglia-reticula are swollen and the cells are apparently in proliferation. The ganglion-cells, when the gray substance is involved, become filled with yellow pigment, lose their processes, and finally disappear by atrophy. It has been pointed out by Frommann that sometimes a considerable quantity of fat in the form of free globules and in cells occupies the sclerotic islands. This he refers to a fatty degeneration of the elements of the neuroglia. Similar gray translucent spots of sclerosis have been observed also in the cerebral nerves and in the roots of the spinal nerves. Sclerotic nodules have been recognized with especial frequency in the optic nerves.

The individuality of this affection has been recognized only within a few years. Prior to a knowledge of its clinical characteristics, as pointed out by Charcot and others, it was confounded with locomotor ataxia, general paralysis of the insane, senile trembling, and especially paralysis agitans. Typical cases at the present time are readily discriminated. This statement will not apply to the rare instances in which the lesions are limited to either the brain or the spinal cord. I have met with an instance, the diagnostic features being extremely well marked, in which there were no symptoms, other than tremor, pointing to a cerebral affection, the duration of the disease extending over many years.

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[1] Later observations (Erb, Frommann) do not confirm the opinion of Charcot that insular sclerosis, in distinction from other forms of chronic myelitis, is characterized by the preservation of many of the nerve-fibres in the sclerotic islands.

The development of the disease is usually very gradual, and for a considerable period its peculiar characters may not be declared. Various indefinite symptoms referable to the brain or spinal cord may have existed for months, and even years, before a positive diagnosis is practicable. Among these symptoms are—cephalalgia, vertigo, mental disturbance, apoplectiform attacks, abnormal sensations in the limbs, muscular feebleness (paresis), lack of co-ordinating power (ataxia), etc. It is easy to understand that different cases offer a great diversity, as regards not only the initial symptoms, but those associated with the diagnostic characters of the disease, when it is considered how great are the differences as regards the number, size, and situations of the sclerotic patches. The interpretation of the diverse symptomatic phenomena is to be sought after in the pathological relations of the different parts of the brain, medulla oblongata, and spinal cord in which the lesions are liable to be seated. Charcot calls the disease polymorphous, in view of the great variety of the forms of disorder observed in different cases. Notwithstanding these variations, the diagnostic features, when these are present, render the differentiation from the several diseases just named not difficult.

Following incomplete paralysis of one or both of the lower limbs, and perhaps also of the upper limbs, accompanied often by rigidity of the muscles (spastic paralysis) and increased tendon reflexes, tremor occurs, and the most diagnostic feature relates to this symptom. The tremor is excited by volition, and is wanting as long as there is no exercise of the will (volitional tremor). The muscles are quiet or nearly so if the patient be entirely at rest, but whenever any action is excited by the will they take on automatic spasmodic movements more or less violent, and the patient's will is powerless for their arrest. These movements may extend from the limbs over the whole body. A hospital patient for a long time under my observation while sitting quietly was free from tremor, but as soon as he made any voluntary effort the motions of his limbs and body were so violent and grotesque that it was difficult to regard the spectacle as otherwise than ludicrous. The tremor which is thus characteristic of this disease is analogous to that of chorea in so far as it is automatic, but excited by the will. In chorea, however, automatic movements take place without as well as with the exercise of the will. The tremor differs from the inco-ordinate movements of ataxia in that the latter are not automatic. The tremor in cases of senile trembling and paralysis agitans differs in being independent of volition, taking place when the patient is entirely at rest. Moreover, in both these affections the movements are less violent, have less range, and are more rhythmical in character. The term *shaking tremor* expresses more correctly the movements in multiple sclerosis than the word tremor alone. This symptom is referable to lesions seated in the medulla oblongata.

A peculiar disturbance of speech is diagnostic of this disease. The patient speaks in a slow, monotonous manner, with intervals between syllables, as in scanning. The peculiarity is known as the "scanning speech." The voice is sometimes reduced to a whisper. Aphonia and the feeble, monotonous voice are referable to paresis of the vocal cords. In some cases the lips, tongue, and muscles of the palate become paralyzed, as in cases of bulbar paralysis.

Impaired vision (amblyopia) and double vision (diplopia) occur not infrequently, and oscillation of the eyeballs (nystagmus) is so frequent in this disease and so rare in other diseases that it has much diagnostic significance. These symptoms represent sclerotic patches affecting the optic tracts or nerves and nerves going to the muscles of the eyeballs. Inequality of the pupils is a not infrequent symptom.

Sensory paralysis is rarely marked, and is not infrequently wanting. If, however, the sclerotic patches involve the posterior columns of the cord, ataxia and more or less impairment of sensibility are associated. There may be little wasting of the muscles, but in some instances it is otherwise, the inference in these instances being that the anterior horns of the gray matter of the cord are involved. Cystitis and bed-sores do not usually occur until late in the disease, and they are often absent.

Disturbances referable to the brain are rarely, but sometimes, wanting. The mental faculties generally fail. Patients often alternately weep and laugh irrationally, and progressive deterioration of the mind ends at length in imbecility. Apoplectiform attacks are liable to occur, accompanied by hemiplegia. With these attacks there may be considerable fever, showing an intercurrent acute inflammatory condition. Patients usually recover from these attacks, but they sometimes prove fatal.

The DIAGNOSIS is to be based chiefly on the tremor, together with the peculiar disorder of speech, taken in connection with the varied motor, sensory, and mental disturbances. The diagnostic criteria probably proceed from sclerosis of the medulla oblongata and pons varolii. If these parts escape, which probably is rare, the clinical characteristics of the disease may be wanting. An important point in the diagnosis is the age of the patient. In the great majority of cases the beginning of the disease dates from a period between twenty and forty years of life.

Aside from the influence of age, nothing has as yet been definitely ascertained respecting the etiology.

The disease is usually progressive, but with a slow march, the duration sometimes extending over a period of from ten to twenty years. Long periods in which the disease appears to be stationary are not uncommon, and also periods in which the symptoms give delusive hopes of real improvement. Often the life of the patient is destroyed by some intercurrent affection. A fatal ending may be due to disturbances of respiration and deglutition incident to the occurrence of bulbar paralysis. Irrespective of these causes, death may be the result of exhaustion from cystitis, bed-sores, and the giving way of the processes involved in nutrition. Clinical experience thus far has furnished no ground for entertaining any hope of recovery from the disease.

The utmost to be hoped for in the way of TREATMENT is that the disease will remain stationary, or, if it be progressive, that the progress may be retarded. Various remedies have seemed to be of use in isolated cases. Among these are the nitrate of silver, arsenic, the chloride of barium, and the phosphide of zinc. The latter remedy, in a case under my observation, was repeatedly followed by distinct although limited improvement. Electricity and hydropathic treatment have appeared to be beneficial. More is to be expected from the removal as far as possible of all causes of disorder, from measures for the improvement of the general health, and from treatment governed by symptomatic indications, than from any remedies given with a view to a special influence upon the disease.

Locomotor Ataxia—Posterior Spinal Sclerosis.

This affection was described by Duchenne in 1858 and 1859 under the name of progressive locomotor ataxia, more fully than by any previous writer; hence Trousseau designated it Duchenne's disease. The principal symptoms of the disease, however, had been recognized previously by Romberg, who applied to it the old name *tabes dorsalis*. The distinctive character and the seat of the affection were clearly defined also by the late Dr. Todd prior to the researches of Duchenne. Other names of the disease are *gray degeneration of the posterior columns*, *sclerosis of the posterior columns*, and *posterior chronic leucomyelitis*.

The anatomical characters of locomotor ataxia consist of sclerosis of the posterior columns of the spinal cord, involving frequently a part of the lateral columns and of the gray substance. The posterior nerve-roots are atrophic and gray. The pia mater covering the posterior part of the cord is thickened, opaque, and is often adherent to the dura mater. In advanced cases the posterior columns appear shrunken, gray, and somewhat firmer than normal. The observations of Pierret (1872), under Charcot's direction, render it probable that the primary seat of the sclerosis is in two bands, each in the outer part of the posterior columns, and lying close to, and parallel with, the posterior horns of gray matter. These bands they call the *external* or *lateral bands*. This view of Pierret and Charcot is based, on the one hand, on the fact that sclerosis of the columns of Goll, although usually present in locomotor ataxia, does not cause ataxic symptoms, as shown in cases of ascending secondary degeneration and of primary sclerosis of these columns; and, on the other hand, on the observation of a case of locomotor ataxia in which the sclerosis was confined to the external bands, while the columns of Goll were normal. When, as is usual, the symptoms are most marked in the lower extremities, the changes in the posterior columns are most extensive and advanced in the lower dorsal and upper lumbar regions. Here the whole thickness of the posterior columns is involved. In very rare cases the symptoms are chiefly referable to the upper extremities, the sclerosis then being most developed in the cervical enlargement. As a rule, however, while the dorso-lumbar sclerosis occupies the greater part of the posterior columns, it is found that in the cervical region the sclerosis is confined either to the columns of Goll or to these together with the external bands. The almost constant involvement of the columns of Goll may be regarded as referable to an ascending secondary degeneration. The sclerosis may be traced continuously to the restiform bodies of the medulla oblongata, where it ceases. There is a tendency in the disease during the later stages to extend to the posterior part of the lateral columns. The posterior horns of gray matter are generally involved. The nerve-fibres in Clarke's columns are atrophied in all typical cases. Extension to the anterior horns of gray matter is a rare complication, causing muscular atrophy. Gray degeneration of the optic nerves, and rarely of the oculo-motor, hypoglossal, and certain other cranial nerves, has been observed.

Microscopical examination of the sclerosed parts reveals increase of the interstitial tissue, which appears as a dense but finely-fibrillated connective tissue, containing in the early stages many nuclei and granular corpuscles, and in the later stages abundant corpora amylacea. The walls of the blood-vessels are thickened, and the perivascular sheaths enclose a large number of fat-globules which have probably been derived from the products of fatty degeneration of the nerve-elements. The nerve-fibres lose their medullary sheaths, undergo atrophy, and for the most part disappear. The exact localization of the sclerosis as described above can only be determined by microscopical study. The morbid process is regarded by most writers as beginning in the nerve-fibres, either as a parenchymatous inflammation (Vulpian, Charcot, Erb) or as a degeneration (Leyden), the chronic interstitial inflammation being a secondary change.

Locomotor ataxia is not a form of paralysis, although it is generally associated with diminished sensibility, and not infrequently with more or less diminution of motor power. The distinctive characteristic of the affection is impairment or loss of the ability to combine and direct voluntary muscular movements. The patient may have as much muscular strength as in health, but there is either inability or difficulty in co-ordinating the action of the muscles by the will. A natural division is into the initial period or first stage, the ataxic period or second stage, and the paralytic or third stage.

Lancinating, shooting pains, usually at first in the lower, and later in the upper limbs, often constitute a prominent symptom. The patient often compares the pains to those caused by electric shocks. In consequence of their shooting, piercing characters and great intensity they have been called fulgurating or lightning pains. They are not positively characteristic, inasmuch as they occur in other forms of myelitis, and are not invariably present in cases of ataxia. The "girdle sensation," which is a symptom in other inflammatory affections of the cord, is often felt. Other events which may occur in the first stage are—incontinence of urine, frequent seminal emissions, anaphrodisia, or, on the other hand, a morbid excitability of the sexual organs, urethral and rectal neuralgic pains, a sense of fatigue in the muscles of the lower limbs after any exertion, attacks of vomiting accompanied by gastralgia (*crises gastriques* of Charcot), and paralysis of certain of the nerves of the eyeball (the oculo-motorius, the abducens, and rarely the trochlearis), giving rise to strabismus, diplopia, and ptosis. These paralytic symptoms may be transient, and they may disappear and return, sometimes remaining permanently. Impairment of vision in some cases occurs early, and it may end in amaurosis. The ophthalmoscope shows an opaque, pearly whiteness of the optic papilla, characteristic of atrophy of the optic nerve. Irregularity of the pupils is not infrequent. The pupil on one side may be contracted to the size of a pin's point, showing paralysis of the sympathetic; and with this symptom are sometimes associated dilatation of the vessels and increased heat on the corresponding side of the face. The pupil on one side may be contracted and on the other side dilated. Oftener both pupils are contracted. A symptom which possesses considerable diagnostic value is the absence of contraction of the pupil on exposure to light, while there is normal contraction in the acts of accommodation (Argyll-Robertson symptom). Difficulty of speech from paralysis of the hypoglossus, and paralysis affecting branches of the trigeminus and facial nerve, are occasional symptoms. The acoustic nerve is rarely involved. Absence of patellar reflex is an early and almost constant symptom of locomotor ataxia. These events are liable to occur in the first stage—that is, they may precede the development of ataxia—or their occurrence may be delayed until the second stage. They may precede for a long period, months and even years, the ataxic characteristics. The latter mark the second stage of the disease.

The ataxia is generally first manifested in the lower extremities. It may, however, appear first in the upper extremities. This was true of a case which I have observed. One lower limb is sometimes affected before the other, and both limbs on the same side may be first affected. When one limb is first affected, whether it be a lower or an upper extremity, it is on the left oftener than on the right side. The defect of co-ordination is apparent when any combined movements are undertaken. In proportion as the affection is marked the patient's gait in walking is uncertain, irregular, and grotesque. The lower limbs are thrown forward by forcible jerks, without definite direction. The body is sometimes swayed from side to side in the attempt to maintain an equilibrium, and the arms are thrown out like those of a person balancing on the tight rope. In cases less marked the greatest difficulty is experienced in beginning to walk, and after getting under way the patient is unable to advance slowly, but walks with precipitation. The legs are thrown forward with a quick, jerking movement, and the feet are brought to the ground on the heels with force and are liable to strike against each other. Notwithstanding the violence of the exertions, the muscular strength being retained patients are sometimes able to walk for long distances. In an extreme degree of the affection walking, even with the aid of a cane or assistants, is impossible, and the patient is confined to the chair or bed.

The defect in co-ordinating power over the upper extremities is shown by an inability to execute acts which require the combined movements of the fingers. The patient is unable to button or unbutton clothes, and yet the grasp may have not less force than in health. A patient whom I exhibited at a clinic tried in vain to unbutton his vest, but his grip was too strong to be borne. In extreme cases the patient is unable to guide the hand to his mouth, and requires to be fed by others.

In a certain proportion of cases the affection progressively extends over the voluntary muscular system, and increases in degree more or less slowly. The defective co-ordinating power may be much more marked at some times than at others. A patient for some time under my observation, who had suffered from this affection for five years, habitually walked with a certain amount of uncertainty and irregularity, and enunciated with labor and slowly, but distinctly. This patient at times, for the space of an hour or so, walked with great difficulty, presenting in a marked degree the characteristic gait, and the embarrassment in speech was such that he was with difficulty understood. These paroxysms recurred on some days repeatedly, and he was sometimes exempt from them for several successive days. This patient had occasionally double vision and night-blindness.

A symptom, even when the affection is slight, is inability to stand with the eyes closed. This symptom is not pathognomonic of locomotor ataxia. The patient, with the feet in apposition and the eyes closed, soon reels, and would fall if not supported. This has been attributed to the coexistence of more or less anæsthesia of the soles of the feet. Patients, however, with notable loss of sensibility of the soles, but who have not ataxia, are sometimes able to stand without difficulty when the eyes are closed.[1] A moderate amount of ataxia occasions want of confidence in the powers of locomotion. A patient under my care has the disease only in a sufficient degree to walk usually with what seems an eccentric gait; but were he to cross a street when it became necessary to hasten in order to get out of the way of carriages, he would scarcely be able to make any headway, and he carefully avoids such a contingency.

In most if not in all cases the cutaneous sensibility is more or less impaired. The amount of sensory paralysis varies in different cases. Generally both anæsthesia and analgesia are combined. There is also diminution of the sense of weight and of temperature. The so-called muscular sense is impaired. Retardation in the transmission of sensory impressions to the sensorium is usually marked. The difficulty in walking is increased by the impairment of sensibility. The patient's eyes are directed to the feet and ground in walking, and there may be inability to walk with the eyes closed.

As already stated, although distinct from motor paralysis, the latter may coexist with the ataxia, and may be either primary or secondary as regards the former. In cases of paralysis from different spinal affections, the difficulty in walking depends often measurably on defective power of co-ordination.

Impotence occurs, as a rule, sooner or later, either before or after the advancement of the disease to the ataxic stage.

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[1] Vide report of a case by Dr. Pepper, in *Am. Journ. of Med. Sciences*, July, 1871.

Affections of the joints occur in a proportion of cases, small, but sufficient in number to show a pathological connection. These have been described, especially by Charcot, under the name of ataxic arthropathies. They may occur before as well as after the development of the ataxic condition. Generally, the larger, but sometimes the smaller, joints are affected. Those attacked by preference are the knee and the shoulder. The joints are attacked suddenly, without any traumatic cause; they are swollen from intra-articular effusion; pain and fever are wanting; and recovery often takes place after several weeks or months. On the other hand, permanent structural changes may be the result, leading sometimes to dislocation[1] and the lesions of deforming arthritis. The patellar reflex is wanting. Percussion over the ligamentum patellæ or over the tendo Achillis does not occasion spasmodic movements as in health. This is a point of diagnostic importance. Cutaneous reflexes are generally normal or nearly so. As a rule, for a long period the nutrition of the muscles is well maintained. The electrical reactions are normal in uncomplicated cases.

In the advanced or paralytic stage notable paralysis is added to the ataxia. The muscles waste, the patient falls into a cachectic state, cystitis and bed-sores occur, and death takes place by exhaustion. Happily, the disease progresses to this stage in but a small proportion of cases. It is generally reached, if reached at all, after a long period. When a certain amount of progress has taken place, sometimes the disease remains without further progress indefinitely or during life. If the disease be progressive, its progress may be extremely slow. Notable improvement takes place in some cases. I have known the ataxia to be so great as to compel patients to keep the bed for several months, and such a degree of improvement to follow that they were able to walk without much difficulty. Complete recovery is extremely rare, but if the disease be not far advanced it is possible. Hence the name progressive, applied by Duchenne, is by no means characteristic of the disease. In the great majority of cases death is owing to some intercurrent affection. The disease may not interfere with the continuance indefinitely of appetite, nutrition, and good general health.

The DIAGNOSIS involves a discrimination from paraplegia, and in some cases from general paralysis. The differential diagnosis may be easily made. The ataxia is not a paralytic affection; and the difficulty in walking or in using any of the affected muscles depends not on deficiency of muscular power, but on the lack of ability to combine and direct the movements of the affected muscles. The difference in this respect is strikingly shown by causing two patients, one affected with paralysis and the other with locomotor ataxia, to walk side by side. The paralytic walks with a feeble, tottering, or shuffling gait, while the patient affected with ataxia throws out his limbs with force, but in an irregular, uncertain manner. The preservation of voluntary motor power in ataxia is shown by the resistance which the patient is able to make when the physician tries to bend the limbs, and also by the freedom and strength of movement when the patient is recumbent. If there be paralysis, more or less in degree, combined with ataxia, it is not a difficult problem to determine this fact and to estimate the relative prominence of either. The coexistence of mental aberration with general cerebral paralysis is a diagnostic feature of the latter. In that disease the voluntary muscles of the limbs, face, etc. are simultaneously affected. In locomotor ataxia different parts of the voluntary muscular system are successively affected, the affection being generally manifested first in the lower extremities. General paralysis of the insane and locomotor ataxia may occur together, either affection appearing first.

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[1] Charcot, *Leçons sur les Maladies du Système nerveux*, Paris, 1877.

Affections of the cerebellum giving rise to vertigo may occasion an embarrassment in walking and standing suggestive of ataxia. The embarrassment is due to vertigo, and is analogous to that caused by drunkenness. With proper attention this cannot be confounded with the ataxic difficulty. Moreover, the vertigo is usually accompanied by other symptoms pointing to cerebellar disease.

Sclerosis of the lateral columns, or spasmodic spinal paralysis, presents diagnostic characters which, as will be seen, are sufficient for the discrimination of this affection. The muscles in locomotor ataxia are notably relaxed.

Before the appearance of ataxia the diagnosis may be considered established if lancinating pains, absence of patellar reflex, and the Argyll-Robertson pupil are all present as symptoms.

The ETIOLOGY of locomotor ataxia relates to age, sex, over-exertion of the muscles, together with exposure to cold and sexual excesses. The disease occurs very rarely prior to adult life. In the great majority of cases the ages are between thirty and fifty years. Men are oftener affected than women. The disease has been observed to follow various affections—namely, rheumatism, pellagra, epilepsy, hysteria, etc.—but the diversity of these antecedent affections renders it doubtful that the sequence denotes any etiological connections. The personal history in many cases gives nothing definite as regards the causation. Syphilis seems to be an undoubted factor in the causation of a certain proportion of cases of locomotor ataxia—a proportion, however, probably not more than 30 or 40 per cent. of the cases. The analysis of the cases shows antecedent syphilis to have been generally mild, and that the ataxia occurs from five to ten years after the syphilitic infection.

TREATMENT.—The nitrate of silver, the iodide of potassium, arsenic, the chloride of barium, and phosphorus are remedies recommended in this disease. Each has been found useful by some, and not so by other observers. None are entitled to be considered as specially curative. Their degree of usefulness is limited in the cases in which they appear to be useful. The nitrate of silver may be given continuously in doses of from a third to a sixth of a grain for several weeks, and it should then be suspended for a season. Phosphorus has seemed to me to be useful in several cases. It may be given in the form of phosphide of zinc or of the oleaginous solution.

They who have had large experience in electro-therapeutics bear testimony to the usefulness of electricity. Both the faradic and galvanic forms are useful, the latter having the more testimony in its favor.

Thermal baths are generally regarded with disfavor by those who have observed their effects in a considerable number of cases. Sponging the body with cool or tepid water and the hydropathic packing, the latter employed with care and moderation, are recommended. The usefulness of these measures, as well as of electricity and the other remedies which have been named, is to be explained by their general influence upon the functions of the body, this influence reaching thus indirectly the local disease.

There is general agreement as to the inutility of local depletion and counter-irritation.

The lancinating pains, the attacks of gastralgia with vomiting, seminal emissions, the affections of the joints, the retention or incontinence of urine, and other complications, claim of course appropriate measures of treatment. Stretching the sciatic or other nerves which are the seat of severe pain is a surgical operation which has afforded great relief in some cases, and has been without benefit in other cases. It is only a palliative measure.

Measures to improve and maintain nutrition, to invigorate the general health, to remove coexisting disorders, and to palliate symptoms, doubtless do much toward preventing or retarding the progress of the disease.

Inasmuch as measures of treatment appear in not a few cases to prevent or retard the progress of the disease, it is obvious that an early diagnosis is highly important. It is important to recognize the significance of the symptoms which precede the ataxic stage, with the hope that as a result of judicious management the disease may not advance to this stage. There seems to be no good reason, *a priori*, why the disease might not become stationary, to say the least, at this as well as at the second stage.

Hereditary Ataxia.

This rare affection was first described by Friedreich in 1863, and is sometimes called *Friedreich's disease.* Although several cases of typical locomotor ataxia sometimes occur in the same family, in Friedreich's disease there are certain peculiarities in the symptoms and the pathological lesions which distinguish the disease from typical locomotor ataxia.

A number of members of the same family are usually affected. In an observation of Carré there were 18 cases of ataxia in one family, occurring during three generations. Dr. W. E. Smith has reported 6 cases of hereditary ataxia in one family.[1] The female members of the family seem to be more subject to the disease than the male, although this has not been observed in all instances. The first symptoms generally appear in childhood, often about the age of puberty, but sometimes earlier or later.

An initial stage of lancinating pains is wanting. Ataxia is the first symptom. Patellar reflex disappears. In typical cases sensation is intact. Eye-symptoms are absent. There is a peculiar disturbance of speech, due probably to inco-ordination in the movement of the muscles involved in articulation. Nystagmus has been repeatedly observed. Spastic paralysis with contractures, and often with muscular atrophy, occurs after the ataxia has existed some time. Spinal curvature, usually in the form of scoliosis, is a frequent symptom. Bladder and rectal disturbances are absent or they occur only in a late stage. The duration of the disease is very long, extending often over thirty years.

The pathological lesion consists in gray degeneration or sclerosis of the posterior columns, of the direct cerebellar fibres, and of the pyramidal tracts. Sometimes there is degeneration of the ganglion-cells in the anterior horns, but whether this be only a complication or not is uncertain.

The disease, so far as known, is incurable. The indications for treatment are similar to those in typical locomotor ataxia.

In this connection may be mentioned the occasional occurrence of so-called *acute ataxia*, observed most frequently after diphtheria, variola, and rarely other infectious diseases. The symptoms resemble those of ordinary locomotor ataxia. Recovery often takes place or the disease may become chronic. Nothing is known of the pathological lesions of acute ataxia.

Acute Anterior Poliomyelitis—Spinal Paralysis of Children.

The primary pathological LESION of infantile spinal paralysis is an acute circumscribed myelitis of the anterior horns extending over a variable portion of the cord. The few autopsies which have been made in an early stage of the affection reveal undoubted evidences of inflammation of the anterior cornua in the presence of leucocytes, proliferating neuroglia-cells, and hemorrhages. Opportunities for examining the cord at a late stage of the disease are not rare. At this period one or both anterior horns in a situation corresponding to the origin of the nerves supplying the paralyzed muscles are shrunken. Upon microscopical examination the ganglion-cells of the anterior horns in this situation are atrophied, and may have entirely disappeared. The surrounding tissue is sclerotic. The sclerosis may extend a little beyond the gray matter into the adjoining antero-lateral columns. The essential lesion, therefore, of infantile spinal paralysis is an acute interstitial myelitis of the anterior cornua resulting in atrophy of the motor ganglion-cells and sclerosis of the neuroglia. When the paralysis affects the lower extremities the poliomyelitis is in the lumbar enlargement. When the upper extremity is paralyzed there is poliomyelitis of the cervical enlargement.

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[1] W. Everett Smith, "Hereditary or Degenerative Ataxia," *Boston Med. and Surg. Journ.*, Oct. 15, 1885.

As would naturally be expected (see p. 730), the disease of the ganglion-cells of the anterior horns is followed by degeneration of the nerves and muscles in connection with the cells. The degeneration of the nerves is characterized by disintegration of the myeline sheaths, increase in the nuclei of the sheath of Schwann and of the endoneurium, atrophy of the axis-cylinders, and eventually new growth of interstitial connective tissue within the nerve-trunk. The degeneration of the muscles is characterized by similar changes—namely, atrophy of the muscular fibres, increase in the sarcolemma nuclei and of the interstitial cells, and a gradual replacement of muscular substance by connective tissue, which may be so infiltrated with fat-cells as to conceal the actual atrophy.

The SYMPTOMS of this disease in children are strikingly distinctive. The initial history of a typical case is as follows: A child is suddenly attacked with fever, perhaps in the night, having been at bedtime apparently perfectly well. The fever has considerable, it may be great, intensity. It is accompanied by cerebral symptoms—namely, vertigo, somnolence, coma, and sometimes convulsions. There is also pain in the spine and extremities. The fever and the concomitant symptoms subside and disappear after a minimum duration of a few hours or a maximum duration of a few days. The patient may have seemed to have had a febricula. As a sequel of the fever paralysis is discovered, generally quite unexpectedly. The paralysis may be at first confined to one extremity, much oftener a lower than an upper. It may quickly extend to another extremity, and it may involve the four extremities. The extension from one part to other parts takes place quickly—that is, within a few hours or at most a few days—and the paralysis as quickly becomes complete. There is no sensory paralysis. After a few days the liability to an extension of the motor paralysis ceases. As regards the parts paralyzed cases differ widely. The paralysis may be confined to one lower extremity; but oftener both lower limbs are affected. Cases occur, but they are extremely rare, in which either one or both of the upper limbs are alone affected. More or less of the muscles of the trunk may be involved. Not infrequently the paralysis does not affect all the muscles of the extremities, but is confined to certain groups of muscles.

There are occasional variations from the foregoing history as regards the fever and the concomitant symptoms. The fever may be slight, and in rare instances wanting. More or less of the cerebral symptoms may be absent. The latter do not represent any definite cerebral affection, and they are probably incident to the fever. The fever has been considered as symptomatic of acute spinal inflammation. The intensity of the fever in some cases, and the short duration, sometimes lasting only a few hours, militate against this view. It is more rational to consider the fever as essential or idiopathic. If the latter view be accepted, the spinal affection is to be regarded as a local manifestation of a general disease, holding to the latter perhaps a relation analogous to that of meningeal inflammation to cerebro-spinal fever or epidemic cerebro-spinal meningitis.

The clinical history after the development of paralysis embraces certain diagnostic features.

The susceptibility of the paralyzed muscles to reflex excitation of the skin and percussion of the tendons is quickly lost.

Owing to the degeneration of nerve and muscle the reaction of degeneration, as previously described (p. 733), rapidly makes its appearance. In seven or eight days the excitability of the nerve to the faradic current may be completely abolished. In consequence of the degeneration of the intramuscular nerve-filaments the response of the muscle to the direct application of the faradic current is also lost, but at a somewhat later period. The galvanic excitability of the nerve at the same time disappears. The galvanic excitability of the muscle, on the other hand, is increased, this increase lasting often for several months. The muscular contractions are sluggish and prolonged, and the anodal closure-contraction equals, and finally exceeds, the cathodal closure-contraction. After about three months the increased galvanic excitability is diminished in consequence of atrophy and destruction of the muscular fibres. The qualitative changes remain. There may be, finally, total disappearance of the galvanic muscular excitability.

If after two weeks the contractility of the muscle to the induced or faradic current be not destroyed, there is a good chance of restoration of the affected muscle. Restoration is also possible after the disappearance of faradic excitability during the period of increased galvanic excitability. Voluntary impulses can be transmitted through the nerves before the faradic excitability returns.

Speedy and notable wasting of the muscles which present the reaction of degeneration is a diagnostic feature. This occurs too early and goes on too rapidly to be referable simply to disuse; it denotes trophic disturbance, and is evidence of a connection of nutrition with the ganglion-cells in the anterior portion of the gray matter of the cord. The atrophy in some muscles may proceed to such a degree that all trace of the muscular tissue has disappeared. The temperature of the surface over the paralyzed parts is lowered, as determined by the thermometer as well as the touch, and the skin has a livid appearance.

Within a few weeks improvement begins either in all or in a certain number of the paralyzed muscles. In a small minority of cases the improvement in all the muscles goes on to complete recovery. In the large majority of cases the improvement is limited to muscles incompletely paralyzed and retaining more or less susceptibility to the faradic current. The muscles which, after the lapse of several weeks, are without any improvement, as a rule remain permanently paralyzed. As regards the number and the situation of the muscles so remaining different cases present great diversities. The permanent paralysis may be confined to one lower limb, to both lower limbs, to an upper limb, to an upper and lower limb, etc. It is rare for all the muscles of a limb to remain paralyzed in an equal degree Often the paralysis is limited to a certain group of muscles, especially to the peroneal and anterior tibial, and sometimes even to a single muscle.

The permanent paralysis of a greater or less number of muscles involves no disturbance of the general health. The assimilatory and the nutritive processes (exclusive of those in paralyzed muscles) may go on as completely as possible. In all respects save the paralysis and its local consequences the health may be excellent and the duration of life is not shortened.

The local consequences of the paralysis are, however, by no means trivial. A great variety of deformities are results of this disease. The growth of the bones is arrested. Owing to a want of a proper antagonism of the muscles, distortions of the feet and hands are common. The joints become loose and often admit of ready dislocations. The painful spectacles so often met with of shortened, flaccid limbs, club-foot, etc., if not congenital, are generally remote consequences of this disease.

The DIAGNOSIS of this disease, as regards other paralytic affections, after the development of paralysis is easy. The diagnostic points summarized are as follows: The paralysis following an initial fever and affecting muscles in rapid succession and in an irregular manner; the paralysis of a certain number of muscles rapidly becoming complete; the rapid appearance of the reaction of degeneration and the speedy disappearance of reflex and tendon reaction; early and progressive atrophy of paralyzed muscles; absence of anæsthesia; coldness and blueness of the skin over the paralyzed limbs; no tendency to gangrenous inflammation of parts exposed to pressure; beginning improvement within a few weeks in some, and sometimes in all, of the paralyzed muscles; permanent paralysis of certain of the affected muscles in the great majority of cases. The discrimination from the cerebral paralysis of children has been already considered.

We possess no actual knowledge of the ETIOLOGY of this disease beyond its being especially incident to childhood. Children, usually from one to four years of age, are attacked when apparently in perfect health and when nothing is apparent on which suspicion can rest as causing the attack. These facts render probable a special cause, the nature and source of which are unknown.

The disease involves very little danger to life. As regards PROGNOSIS, the danger relates to permanent paralysis with atrophy of a greater or less number of muscles and the deformities arising therefrom.

The object of TREATMENT in the early stage of the disease is the prevention of these; in other words, the prevention of the changes in the anterior gray columns of the cord, on which depend the permanent paralysis and atrophy of groups of muscles corresponding to the situation and the extent of the spinal lesions. For this object the treatment indicated in acute myelitis is recommended—namely, active purgation, local bloodletting by cups or leeches, the application of cold to the spine, dry cupping and vesication, mercurial inunctions, the internal administration of ergot, belladonna, and the iodide of potassium. The physician must use his discretion in the employment of these measures, having due regard to the age and the constitution of the patient. Could the diagnosis be made in the initial febrile stage, perhaps more would be accomplished by antipyretic treatment than by the measures just enumerated. Unfortunately, a positive diagnosis is rarely made in this stage.

The treatment after the occurrence of paralysis has for its object the restoration of motor power over the paralyzed muscles and the prevention of atrophy. For these objects the reliance is chiefly on electrical treatment. Neurologists are generally agreed upon the superiority of the galvanic current, but faradic electricity may be combined with advantage. The electrical treatment should be continued steadily for many months. Hydropathy employed with proper care, and massage, are useful. The system should be well nourished and invigorated by hygienic measures, together with tonic remedies whenever these are indicated by debility, want of appetite, feeble digestion, etc.

The treatment of the deformities which are likely to follow this disease falls within the province of surgery.

Acute Anterior Poliomyelitis in the Adult.

It was for a long time supposed that the form of acute atrophic spinal paralysis described in the preceding article was confined to children. It is now known that the same affection may occur in the adult. The pathological lesion is the same as that already described—namely, an acute anterior poliomyelitis.

This affection in the adult is rare. After childhood the cerebral symptoms which in children accompany the initial fever are less marked. The clinical characteristics, however, which relate directly to the spinal affection are the same. The development of the paralysis is rapid. The distribution of the paralyzed muscles is irregular, and, as regards their number and situation, different cases present much diversity. In more or less of the affected muscles reflex and electrical excitability is soon greatly lessened or lost. There is no impairment of sensation. The bladder and rectum are unaffected. The sexual function is not involved. After the lapse of from one to two weeks improvement in certain, or it may be in all, the paralyzed muscles begins. If improvement begin in all and progress, the recovery may be complete after several weeks or months. The improvement and recovery, however, may be limited to a certain proportion of the paralyzed muscles, a greater or less number remaining permanently paralyzed. These rapidly atrophy, and as remote consequences distortions occur, but in a less degree than in childhood.

The main difficulty in diagnosis is to distinguish acute anterior poliomyelitis in the adult from primary degenerative neuritis involving several nerves. In both affections there is muscular paralysis with rapid atrophy and the reaction of degeneration, but in neuritis there are usually severe neuralgic pains, and often other sensory disturbances lacking in poliomyelitis.

The objects of treatment and the measures to be pursued are the same as in the disease occurring in childhood.

Subacute and Chronic Anterior Poliomyelitis—Subacute and Chronic Atrophic Spinal Paralysis.

Duchenne first described, under the name of subacute anterior general spinal paralysis, an affection characterized by the gradual or by the rapid development of paralysis—first, usually, of the lower and then of the upper extremities—without marked disturbances of sensation, combined with progressive atrophy of the paralyzed muscles, and by loss of reflex and of faradic excitability. The recorded post-mortem examinations do not suffice for positive statements as to the anatomical basis of the disease. It is believed that the disease is pathologically a subacute or a chronic myelitis of the anterior horns.

The clinical characteristics are analogous to those of the acute affection, exclusive of symptoms denoting acuteness. Fever is absent. The development of the paralysis, as a rule, is not rapid but gradual, extending over months or even years. Groups of muscles of the lower extremities are usually first affected, and the paralysis sooner or later may extend to groups in the upper extremities and in the trunk. The irregular distribution of the paralyzed muscles is a feature of this as of the acute affection. The paralyzed muscles present the reaction of degeneration, either complete or partial, and atrophy occurs, evidently from trophic disturbance. Reflex excitability of the paralyzed muscles is extinguished. Sensibility is but little or not at all impaired. The bladder and rectum are not involved.

Complete recovery takes place oftener than in the acute affection: but, in a certain proportion of cases, the recovery is limited to a greater or less number of the paralyzed muscles, the others remaining permanently paralyzed. Distortions follow the disturbance of the normal equilibrium of antagonizing muscles. Death is sometimes caused by an extension of the paralysis upward, so that deglutition and respiration become affected.

This paralytic affection is distinguished from progressive muscular atrophy by the fact that paralysis precedes the atrophic degeneration of muscles, whereas in the latter affection atrophy of the muscles is the primary event, of which the loss of muscular power is an effect. Moreover, in the latter affection the atrophy affects parts of the muscles, more or less of the fibres retaining their susceptibility to reflex action and electricity. In chronic anterior poliomyelitis, on the other hand, the loss of motor power, together with that of reflex and electrical excitability, extends over the whole of the muscles affected. The main difficulty is to distinguish subacute and chronic anterior poliomyelitis from primary multiple neuritis. It is probable that many cases of the latter affection have been erroneously referred to poliomyelitis. The chief points in diagnosis are mentioned in the preceding article.

The objects and measures of treatment are the same as in acute poliomyelitis after the acute stage has passed. With our present knowledge, the chief reliance is on the judicious and persistent employment of electricity, especially of the galvanic current.

Progressive Muscular Atrophy.

Progressive muscular atrophy was described first under this name by Aran in 1850. In 1855, Cruveilhier also investigated the disease, particularly from a pathological point of view. The affection has been called by English writers *wasting* or *creeping palsy*. There has been much controversy as to whether the primary seat of the disease be in the muscles (the myopathic theory) or in the spinal cord (the neuropathic theory). We are indebted especially to Lockhart Clarke and to Charcot and his school for repeated and careful microscopical examinations of the spinal cord in cases of progressive muscular atrophy. These investigators have observed with great uniformity, as the essential lesion, atrophy of the ganglion-cells in the anterior horns, combined with chronic interstitial inflammation about the atrophied cells. The view held by Friedreich that these changes in the spinal cord are not primary, but secondary to the muscular atrophy, has not met with favor, and is not in harmony with the fact that muscular atrophy, when undoubtedly primary, may exist for a long time without alterations in the cord, and that the atrophy of ganglion-cells which follows amputations is not to be compared in extent and degree with the atrophic changes in progressive muscular atrophy. The *neuropathic* origin of typical cases of progressive muscular atrophy (that is, of the type of Aran and of Duchenne) may be considered, therefore, as demonstrated; but it is also certain that there are cases even of widespread muscular atrophy which may bear great resemblance to the Aran-Duchenne type, and in which the most searching microscopical examination has failed to reveal any atrophy of ganglion-cells or other changes in the spinal cord. A case of this nature of great similarity to typical progressive muscular atrophy, but differing from it in certain particulars, has been reported by Lichtheim.[1] He failed to discover any diminution in the number or size of ganglion-cells in the cord. We must therefore admit that there are certain forms of progressive muscular atrophy which differ more or less widely from the typical variety, and which are *myopathic* in their origin. Our information as to the points of distinction between the myopathic and the neuropathic forms of progressive muscular atrophy is as yet so imperfect that it would not be profitable, within the limits of this work, to attempt to classify and describe separately different forms of the disease. We may hope that in the future their distinctive characters will be better understood. In the present article the ordinary form of progressive muscular atrophy will be described. This may be considered to be neuropathic in origin.

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[1] *Archiv für Psychiatrie*, 1878, Bd. 8.

Progressive muscular atrophy may be *primary* or *secondary*. In the secondary form (deuteropathic of Charcot) the chronic anterior poliomyelitis is secondary to some other morbid process in the cord, such as primary lateral sclerosis(?), posterior spinal sclerosis, spinal meningitis, and insular sclerosis. Of the secondary form, that consecutive to lateral sclerosis is the most important, and is called amyotrophic lateral sclerosis. It will receive consideration in a separate article. The intimate connection between chronic bulbar paralysis and progressive muscular atrophy in symptomatology, development, and pathology has already been mentioned in treating of the former affection.

The ANATOMICAL APPEARANCES in the spinal cord in cases of progressive muscular atrophy are similar to those met with in the later stages of infantile spinal paralysis. The primary change is, however, best regarded as a degenerative atrophy of the ganglion-cells, and not as a myelitis. No changes may be visible to the naked eye, or a more or less distinct atrophy of the anterior horns may be discerned. The microscopical examination shows the various stages of pigmentary and sclerotic atrophy of the motor ganglion-cells in the anterior horns, the presence of a few granular corpuscles, and increase with induration of the interstitial tissue, especially well marked in the neighborhood of the atrophied cells or cell-groups. Gray degeneration of the anterior nerve-roots and of peripheral nerves supplying the affected muscles is also present. The changes in the muscles are not those of a simple fatty degeneration, as was at one time supposed. They consist in a simple atrophy of the muscular fibres, which become smaller and smaller, but preserve their striæ until much reduced in size. Many of the fibres possess a greatly increased number of sarcolemma nuclei, and some may present the characters of fatty degeneration. The interstitial tissue is at the same time increased in amount, and may contain some fat-cells. It is a noteworthy fact that the affected muscles do not undergo atrophy in their entire volume at once, as is the case in infantile spinal paralysis, but that individual fibres or bundles of fibres become atrophied, while adjoining fibres may remain of normal size. This peculiarity of atrophy of individual fibres or of single bundles in a muscular mass forms one of the distinguishing characters of progressive muscular atrophy.

Progressive muscular atrophy generally begins in one of the upper extremities, oftener in the right than in the left. The affection is at first usually limited to a certain number of muscles. In the larger proportion of cases some of the muscles of the hand are primarily affected. The muscles forming the ball of the thumb and the ball of the little finger and the interosseous muscles are first atrophied. Less frequently the muscles of the shoulder or of the arm or of the forearm are the first to become atrophied. Corresponding muscles of the other extremity are likely to become subsequently affected, the affection thus exemplifying the law of parallelism. More or less gradually the affection extends over the muscular system. Not only the muscles of the extremities, but those of the trunk, are likely to become atrophied, and in some cases the latter are first affected. The intercostal muscles and the diaphragm may be involved, and death may be produced by apnœa. The muscles of deglutition may become involved, and the affection may prove fatal by causing inanition. The affection is limited to the voluntary muscles.

A marked degree of atrophy of external muscles renders the appearance striking and characteristic. In a case which came under my observation the atrophy was limited to the muscles of the left shoulder, arm, and forearm. The right scapula was well covered by its muscles, but on the left side this bone was conspicuous, its boundaries distinct, and the spinous ridge projecting. The whole of the upper limb was attenuated, presenting a remarkable contrast with its fellow. In a case reported by Reade, the patient, a young man, when stripped to the waist "exhibited the neck, chest, and arms to the elbow-joints reduced to the most abject degree of emaciation. The emaciation was perfectly symmetrical; the greater and lesser pectoral muscles were little more dense than the strongest brown wrapping-paper; the muscles of the neck, anterior and posterior, proportionately attenuated; the muscles on the scapulæ, particularly the supra- and infraspinatus, were so much diminished as to show the spine of the bone with distinctness only less than the dry bone; all prominences from the deltoids were gone, and the muscles of the humeri were reduced to the cellular membrane, the mere elementary outline of the muscles, the biceps and triceps especially. From the elbows the muscles of the forearms and hands displayed the full development of a robust and vigorous man of his stature, with all the concomitant power, sensibility, and aptitude for use. All the muscles outside the pelvis and those of the inferior extremities were full, strong, and well formed."[1] A patient in Bellevue Hospital presents great attenuation of the arms and thighs, the forearms and legs retaining a large muscular development. In this case the muscles of the back are affected, so that in walking the spine is curved backward, and on raising the upper extremities the scapulæ are thrown outward in a remarkable manner, forming what has been called the "wing shoulder." In another case the atrophy is limited to the feet and about two-thirds of the muscles of the calves, the remaining third of the latter being unaffected. The feet have the appearances of bones covered only with integument. Cases have been reported in which the affection extended over the greater part of the voluntary muscular system. The tongue, the muscles of mastication, the facial muscles, and the muscles of the larynx may be included in the general atrophy. In a case cited by Thouvenel the power of moving the limbs and body was lost, and the patient was able only to move the head feebly. The tongue was involved in this case, and deglutition was difficult. Trousseau refers to a case treated by Bretonneau in which the power of speech and all voluntary movements, except of the head and of the forefinger of the right hand, were lost. This patient, a female, held communications with her family by indicating letters, in this way forming words and sentences. She dictated in this manner her last will and testament. When the atrophy is limited to a few muscles the loss of substance is rendered striking by the normal size of the adjacent muscles. Atrophy of one class of muscles—for example, the flexors or extensors of the limbs—causes distortion by the contraction of the muscles which are not affected, the contraction arising from the loss of antagonism. Distortions of the head, trunk, and extremities may be produced by the limitation of the atrophy to certain muscles, or by the progress of the atrophy being much greater in some muscles than in others. The characteristic appearances belonging to the affection give to it a physiognomy by which it is readily recognized in well-marked cases. Among the frequent deformities may be mentioned the claw-like hand, or *main en griffe*. The first phalanges are extended, the second and third are flexed. This deformity is due to paralysis of the interosseous and lumbrical muscles. In the early part of its progress the disease may escape recognition if a close examination be not made.

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[1] *Dublin Quarterly Journal.*

In general, the first evidence of trouble is weakness of the affected muscles, arising from the atrophy. Pain or soreness precedes and accompanies the affection in a certain proportion of cases. Generally the pain is not severe. It is referred to the affected muscles, and is of a neuralgic character. The affected parts are notably sensitive to cold, and their temperature is below that of other parts. Defective capillary circulation is sometimes shown by congestion of the surface. Quivering movements of the fibres of the affected muscles, called fibrillary contractions, are often observed, and sometimes subsultus of the tendons and slight twitchings of the parts to which the tendons are attached. These movements occur irrespective of the will, occasion no pain, and the patient may not be conscious of them. The sensibility of the surface is unaffected. The mental faculties remain intact, and there are no symptoms pointing to intracranial disease. The appetite, digestion, and nutrition may continue unaffected. There is no loss of power over the bladder or rectum, even when the affection is far advanced, nor does the heart become affected. Constipation may be produced by atrophy of the abdominal muscles. The affected muscles lose their electro-contractility only in proportion as they become atrophied and degenerated; and the fibres which are unaffected respond to the electrical current. For this reason it is difficult to make out the reaction of degeneration, which is confined to the atrophied fibres, but by careful examination it has been detected in muscles greatly atrophied. This apparent preservation of electrical contractility serves to distinguish the affection from the wasting of muscles incident to the acute and chronic forms of anterior poliomyelitis, which have been considered.

The progress of the affection is slow. More or less slowly increasing, it is thus, as a rule, progressive, as the name implies. This rule, however, is not without many exceptions. Often it remains stationary after having progressed to a certain extent. In a few cases recovery may be said to take place; that is, the affection ceases without leaving any permanent deformity or notable debility. It may continue, slowly progressing, a great number of years before tending to a fatal result. The prognosis is extremely unfavorable. The most to be hoped for is that it will cease to progress or that its progress will be very gradual. Retrogression is not to be expected after much change in the muscles has taken place. Life may be destroyed by apnœa if the respiratory muscles become affected, or by inanition from atrophy of the muscles concerned in deglutition. In other cases death takes place after a long and tedious confinement to the bed if the patient be not carried off by some intercurrent disease.

As regards DURATION, the following are the results of the analysis of 105 cases by Roberts: Mean duration of cases ending in recovery, one year and two months; of cases ending in permanent arrest, two years and three months; of cases ending fatally, five years and two months. Of the cases which recovered, the longest duration was two and a half years, and the shortest eight months. Of the cases proving fatal, the longest duration was twenty-three years, and the shortest twelve months. The longest case ending in arrest continued active for seven years, and the shortest for four months.

The DIAGNOSIS relates to the exclusion of other affections which are attended by muscular atrophy, such as diffuse myelitis, anterior poliomyelitis, primary neuritis, and primary muscular affections with atrophy. The chief points to be considered are the typical onset of the disease in the small muscles of the hand; the development of the atrophy first in individual muscles, and even in individual muscular fibres, and not in groups of muscles; the typical extension of the atrophy to the muscles of the shoulder and other parts; the development of bulbar paralysis in some cases; the apparent preservation for a long time of electric excitability; the absence of sensory disturbances; and the existence of paralysis only in proportion to the degree of atrophy.

The CAUSATION is obscure. The affection rarely occurs under adult age, and it is unusual after middle life. Males are much oftener affected than females. There is no connection with social condition or particular occupations. Several members of the same family in repeated instances have been affected, but most cases of hereditary muscular atrophy do not belong to the Aran-Duchenne type. It has been supposed in some cases to proceed from undue muscular exertion. Like most affections, it is sometimes attributed to the action of cold.

The great object of TREATMENT is the arrest of the progress of the affection. The measures for this object have reference to the affected muscles and the general condition. Undue exertion of the affected muscles is to be avoided, but a certain amount of exercise is important. The circulation and nutrition within the affected muscles are to be promoted by friction and shampooing and the use of stimulating embrocations. As regards treatment addressed directly to the affection, however, the most potential measure is the employment of electricity. Each of the different forms of electricity is useful, and they may be employed in alternation. Measures having reference to the general condition are those which invigorate the powers of the system. Here, as in cases of progressive locomotor ataxia, judicious management, although it may fail to effect recovery, may do much toward retarding the progress of the affection and prolonging the life of the patient. The point in the progress of the affection at which the arrest is effected is of great importance to the patient; hence the desirableness of an early diagnosis.

In one of the cases at Bellevue Hospital which has been referred to, in which the muscles of the arm, thigh, and back are greatly atrophied, not only has there been no progress of the disease during nearly ten years, but the patient is greatly improved, and although the atrophied muscles remain the same, he has been able for several years to do active duty as a hospital nurse and in other kinds of labor.

Progressive Unilateral Facial Atrophy.

In this rare disease the muscles are not primarily nor chiefly affected, but the atrophy begins at the surface and progresses inwardly, involving the bones as well as the soft parts. The skin is discolored and thinned. The beard, eyebrows, and hair on the affected side become white and often fall out. The subcutaneous fat disappears. The muscles are more or less atrophied, but in a less degree than other structures. The bones and cartilages are atrophied. The atrophy may extend to the tongue on the same side and also to the palate and uvula.

The disease occurs in women much oftener than in men, and very rarely after twenty-five years of age. The left much oftener than the right side of the face is affected.

The contrast between the two sides of the face renders the recognition of the disease easy after it has made considerable progress. It cannot be confounded with facial paralysis, inasmuch as drawing of the mouth to the opposite side is slight or wanting, and the muscular movements are not lost on the affected side.

The disease usually progresses very slowly. In all other respects the patient may be perfectly well.

The pathogeny is undetermined. It is yet to be settled whether the disease be primarily seated in the affected tissues or whether the atrophy be secondary to a neurotic affection. If the neurotic or trophic theory be adopted, it is not settled to what particular nerve or nerves the disease is to be referred. The trifacial, the facial, and the cervical sympathetic have each been supposed to be especially involved.

The disease thus far has proved intractable to treatment. An arrest of the progress is all that can be hoped for. It must be expected that the lesions which have taken place will be permanent. The progress of the disease does not endanger life, but the conspicuous deformity is not a small calamity.

Spastic Spinal Paralysis—Primary Lateral Sclerosis.

In 1875, Erb described a group of symptoms as a well-defined disease to which he gave the name *spastic spinal paralysis.* Charcot, who had previously recognized the main features of the disease, accepted Erb's description, but called the affection *spasmodic tabes dorsalis.* The leading symptoms are slowly-developing paralysis, rigidity of the affected muscles, contractures, and increased tendon reflexes. There is absence of sensory and of trophic disturbances in uncomplicated cases.

It is assumed by Erb and by Charcot that the anatomical characteristic of the disease is primary sclerosis of the lateral columns of the cord. This view is in harmony with the teaching of Charcot, that sclerosis of the lateral columns, whether primary or secondary, is attended by tension of the paralyzed muscles. The assumption, however, that Erb's spasmodic spinal paralysis depends upon primary lateral sclerosis has not as yet been confirmed by post-mortem examinations. Charcot himself has published a case in which he had diagnosticated this disease during life, and the autopsy revealed multiple sclerosis. A variety of affections of the spinal cord and of the brain may produce the combination of symptoms described by Erb, such as chronic hydrocephalus, transverse myelitis in the dorsal or cervical region, tumors of the cord, compression of the cord, multiple sclerosis, hydromyelus, and combined systemic diseases of the cord. In these cases the symptoms may be for a long time those of pure spastic paralysis, but eventually other symptoms, as a rule, are superadded. In the present state of the question it seems proper to recognize as a distinct symptomatic affection the clinical picture drawn by Erb, but it is necessary to await future investigations before we attempt to localize the anatomical changes which form its basis.

The combination of locomotor ataxia with lateral sclerosis has been described especially by Westphal. In these cases there is an extension of the sclerotic process from the posterior to the lateral columns. Muscular rigidity is added to the characteristic ataxic symptoms. Secondary lateral sclerosis has been repeatedly mentioned already as forming the descending secondary degeneration of Türck.

The combination of lateral sclerosis with anterior poliomyelitis constitutes the disease called amyotrophic lateral sclerosis, which will be described in the next article.

From a clinical point of view the symptoms described by Seguin in 1873, and called by him "tetanoid pseudo-paraplegia," subsequently described by Erb and called spasmodic spinal paralysis, and by Charcot under the name of spasmodic dorsal tabes, represent a distinct and easily-recognized paralytic affection. Cases presenting its distinctive features, as delineated by these authors, have fallen under my observation.

A synopsis of the clinical history is as follows:

Incomplete paralysis (paresis) begins in either the lower or the upper extremities—according to Erb, much oftener in the former. The paralysis very slowly increases. After having reached a certain degree it is accompanied by twitchings of the muscles and muscular rigidity. The twitchings occur in connection with voluntary movements as well as irrespective of the will, and especially at night. The muscular tension or stiffness is at first excited by motor acts, and, lasting for a few moments only, after a time becomes persistent, and at length leads to a permanently rigid extension of the limbs.

Pressure on the toes excites tremor of the foot and sometimes of the leg. Tremor is excited by a rapid flexion of the foot or by pressure with the hand upon the toes. This symptom is called ankle-clonus (p. 732). It is not, however, peculiar to this affection. Tremor occurring with a certain degree of violence, and sometimes extending to the other limb, has been called by Brown-Séquard by the inappropriate name "spinal epilepsy." It may sometimes be excited by mere contact of the fingers with the skin.

The tremor, stiffness, and spasmodic twitchings added to the paralysis increase the difficulty in walking, and give rise to what is termed by Erb the "spastic gait," which he thus describes: "The feet seem to cleave to the ground; the tips of the feet find an obstacle in every irregularity of the ground; every step is accompanied by a peculiar hipping elevation of the whole body; . . . . the patient immediately gets upon his toes, and steps forward on them, showing a tendency to fall forward. The legs are close together, held stiffly, the knees somewhat depressed forward, the upper part of the body slightly bent forward." This mode of walking is distinctive as contrasted with the purely paralytic and the ataxic gait.[1] The difficulty in walking is out of proportion to the impairment of muscular power, yet there is no resemblance to ataxia.

Reflex motions are abnormally excited by percussion of the tendons, the affection in this respect offering a contrast to ataxia. Sensibility remains intact. The bladder and rectum are unaffected. There is no tendency to bed-sores. The sexual functions may be retained. The muscles for a long period do not undergo atrophy. Cerebral symptoms are absent. The electrical excitability of the muscles is slightly diminished, but there is no reaction of degeneration.

The DURATION of the affection extends over many years. It may slowly progress in degree, and gradually be diffused over all the extremities, or, having reached a certain stage, it may remain stationary for an indefinite period. Patients generally die from some intercurrent disease. If, however, it be otherwise, and the affection continue to be progressive, confinement to the bed is at length necessary. The affection may extend to the medulla oblongata, and give rise to the symptoms of bulbar paralysis. Cystitis and bed-sores do not occur in typical cases.

The foregoing account applies to typical cases. The symptoms may be associated with those of ataxia in different degrees. The inference, then, is that the sclerosis also embraces the posterior columns. The affection may be unilateral, confined either to one limb or to the two limbs on one side. There may occur more or less speedily muscular atrophy. Under these circumstances the affection has been described by Charcot under the name amyotrophic lateral sclerosis. This form will be noticed under a separate heading.

The ETIOLOGY of this affection is not less obscure than that of the other sclerotic affections of the spinal cord. In the great majority of cases the age of patients is between thirty and fifty. Men are oftener affected than women, and the affection occurs in persons apparently in robust health. In a typical case which came under my observation the patient had suffered much from syphilis.

This form of paralysis admits of a favorable PROGNOSIS as regards a fatal termination. Erb states that recovery may be said to have taken place in two cases under his observation, and notable improvement in three cases. The chief source of danger relating to the disease is the liability to the occurrence of bulbar paralysis or muscular atrophy.

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[1] Vide *Ziemssen's Cyc. of Med.*, Am. ed., vol. xiii. p. 97.

The principles of TREATMENT are essentially the same as in the other varieties of sclerotic disease. Electricity, especially the galvanic current, is to be employed, and hydropathic measures may be tried with proper care and precautions. Strychnia is contraindicated.

Amyotrophic Lateral Sclerosis.

Progressive muscular atrophy, as has already been mentioned, may be a primary affection or may be secondary to other diseases of the spinal cord. Of the secondary or deuteropathic forms of progressive muscular atrophy, that designated by Charcot in 1874 as amyotrophic lateral sclerosis has received especial attention. In this disease the symptoms of progressive muscular atrophy are preceded by paralysis and accompanied by rigidity. In other words, the symptoms of lateral sclerosis and of progressive muscular atrophy are combined. Post-mortem examinations have revealed the lesions of progressive muscular atrophy (degeneration and atrophy of the ganglion-cells of the anterior horns), together with sclerosis of the pyramidal tracts in the anterior and the lateral columns. The antero-lateral sclerosis occupies the same situations as in Türck's secondary descending degeneration; that is, a narrow, wedge-shaped tract on each side of the anterior median fissure, known as the columns of Türck, and a triangular column in the posterior part of each lateral column, known as the pyramidal tract of these columns. (See p. 728.) Bands of sclerotic tissue connecting the diseased parts of the anterior horns with the sclerosed lateral columns have been observed. The microscopical changes are those which have already been described as characterizing progressive muscular atrophy and gray degeneration of the pyramidal tracts. In some instances the degeneration of the pyramidal tracts has been traced throughout their whole extent from their origin in the cerebral cortex to their terminations in the spinal cord. The large pyramidal ganglion-cells in the motor cortical zone have been found atrophied in these cases. The disease is, in the majority of cases, accompanied by the symptoms and lesions of chronic bulbar paralysis. The primary lesion is to be regarded as degeneration, and not inflammation.

The paralysis begins generally in the upper limbs, and extends to the lower, but in some instances the reverse is the case. The spastic symptoms are usually more marked in the lower extremities, and the atrophic symptoms in the upper extremities. The atrophy of muscles generally begins, as in progressive muscular atrophy, in the small muscles of the hand. In distinction from progressive muscular atrophy the tendon reflexes of the paralyzed muscles are increased. In an advanced stage of the disease contractures occur. Abnormal electrical reaction can be made out only in muscles which are markedly atrophied. In these the reaction of degeneration is apparent, but in most cases this is the so-called partial reaction of degeneration, the electric excitability of the nerves being normal or nearly so in consequence of the preservation of many nerve-fibres. (See p. 733.) Sensation is intact. There are no vesical disturbances. Finally, if life be sufficiently prolonged the symptoms of bulbar paralysis appear.

The affection, as compared with simple spasmodic paralysis, runs a rapid course. It arrives at an advanced stage within six months or a year, and death takes place in from two to three years.[1]

In no instance has recovery been known to take place. The indications for TREATMENT are those which belong to progressive muscular atrophy and to bulbar paralysis. Irrespective of the muscular atrophy and bulbar paralysis, the treatment is the same as in cases of uncomplicated spasmodic spinal paralysis.

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[1] Vide Charcot, *Leçons sur les Maladies du Système nerveux*, Paris, 1877, tome ii.

Compression of the Spinal Cord—Intraspinal Tumors.

Compression of the spinal cord may be produced suddenly or gradually. Fractures and dislocations of the vertebræ constitute the most frequent causes of sudden compression. Their consideration belongs to the domain of surgery. Meningeal hemorrhage and the rupture of aneurisms, abscesses, or cysts into the spinal canal may occasion rapid compression of the cord.

Of greater interest and importance is the study of the causes and results of slow compression of the spinal cord. The causes are many and diverse. Of the more important may be enumerated—*first*, tumors and inflammatory affections of the vertebræ, especially Pott's disease and cancer; *second*, extravertebral tumors, such as aneurism, cancer, or abscess, which make their way into the spinal canal either by erosion of the vertebræ or through the intervertebral foramina; *third*, tumors and inflammatory thickening of the meninges and of the perimeningeal tissue; and *last*, tumors of the spinal cord itself. Tumors of the cord itself are less frequent than those of the meninges. They include solitary tubercle, gumma, glioma, sarcoma, neuroma, and secondary carcinoma (p. 41 *et seq.*). Dilatation of the central canal is called *hydromyelus*, and is generally congenital. Cavities of variable size and length and more or less centrally located may be developed in the spinal cord in various ways. The name *syringo-myelus* or *syringo-myelia* is given to these pathological canals. (See p. 713.)

Of the tumors of the meninges and perimeningeal tissue, may be mentioned—lipoma, sarcoma, myxoma, fibroma, psammoma, gumma, enchondroma, secondary cancer, echinococcus, and cysticercus cysts. Calcific plates in the dura mater, and especially in the arachnoid, are not uncommon in elderly persons, and, as a rule, produce no symptoms. In the list of tumefactions in this situation which act as causes of compression should be included hemorrhage and certain chronic inflammatory processes. Hemorrhage in the form of a tumor (hæmatoma) may be the result of internal hemorrhagic spinal pachymeningitis. Of the inflammatory thickenings, that described by Charcot as *cervical hypertrophic pachymeningitis* deserves especial mention. This affection has already been described (p. 706).

Of the causes of gradual compression of the cord, the most frequent and best studied is *caries of the vertebræ*, or *Pott's disease.* Is was formerly supposed that compression of the cord was due to the narrowing of the spinal canal in consequence of the angular curvature of the spine, or kyphosis, so frequently produced in this disease. It is indeed certain that in rare instances the cord is compressed by the displaced vertebræ, but that this displacement is not necessarily or usually the cause is proven by the following facts: Extreme angular curvature may exist without symptoms of compression; these symptoms may be present in Pott's disease without curvature; and the symptoms may disappear while the kyphosis remains unchanged. We owe to Michaud,[1] a pupil of Charcot, the explanation of the mechanism of compression in these cases. By extension of the diseased process from the vertebræ to the perimeningeal tissue and the outer layer of the dura mater a peripachymeningitis and a pachymeningitis externa are set up, leading to the formation of new connective tissue and of pus, which undergo in part a caseous metamorphosis. The caseous purulent mass thus formed between the bone and dura mater and in the substance of the dura mater is really the agent of compression. There may be an extension of the inflammation to the inner layers of the dura and to the other membranes of the cord, but this is not necessarily the case.

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[1] *Sur la Méningite et la Myélite dans le Mal vertébral*, Paris, 1871.

Compression renders the cord anæmic and flattened, and if protracted leads to inflammatory changes which present the picture of a transverse myelitis followed by secondary ascending and descending degenerations. This is known as *pressure myelitis.* In a given case it is not always easy to determine how much of the disturbance is due to compression and how much to myelitis; but that compression often plays an important part is proven by the fact that the symptoms may rapidly disappear as soon as the cause of compression is removed.

It is obvious that the SYMPTOMATOLOGY in different cases of compression of the cord must vary very much according to the cause, the seat, the amount of pressure, the nature and extent of the induced changes in the cord, etc. Diseases of the vertebræ, as well as fractures and dislocations, are determinable causes. The existence of an intraspinal tumor can hardly be determined with certainty, and, assuming that a tumor exists, to determine its character from the symptoms is impracticable.

The prominent symptom in cases of compression from different causes is paralysis, greater or less in degree (paresis) or complete. The extent of the paralysis will of course depend on the seat of the affection. If seated in the cervical portion of the cord, it may give rise to general spinal paralysis. In very rare instances the paralysis may be paraplegia of the upper limbs, the lower limbs remaining unaffected. If the compression be limited to one-half of the cord, which is extremely rare, the result may be spinal hemiplegia with crossed anæsthesia, as described by Brown-Séquard.

Much more frequently the paralysis has the form of paraplegia affecting the lower limbs. If the spinal affection be above the lumbar region, and if the latter be not involved, reflex action is not diminished, but is often increased. The increase is sometimes so great as to occasion severe contractures and rigidity. Reflex action is diminished or lost if the spinal affection involve the gray matter of the lumbar region. Motor paralysis is often neither accompanied by anæsthesia at the outset nor after a considerable period. The paralysis may or may not involve the bladder or rectum. Sooner or later, if lesions of the cord be progressive, these organs become involved, the paralysis of motion and sensation becomes complete, and death takes place, as when equally destructive lesions occur independently of tumors or the various causes of compression.

Pain in the spine, in parts of the trunk below the level of the spinal affection, and in the paralyzed limbs (paraplegia dolorosa) is especially marked in cases of cancer of the vertebræ extending to the cord. This symptom may be more or less marked, however, in other forms of disease. It is presumptive evidence of an intraspinal cancerous tumor that prior to the affection of the cord cancer existed in some other situation. That an affection of the cord is syphilitic is to be suspected whenever the patient has had syphilis; and with reference to treatment it is very important to seek for evidence of syphilis, although the existence of this disease be denied by the patient. It is to be considered that women may have been affected with syphilis without any suspicion on their part of the nature of the disease; and there are instances in which the physician must derive his knowledge on this point wholly from what he is able to observe without exciting by his questions any suspicion in the mind of the patient.

It is an important practical fact that the spinal cord may tolerate compression sufficient to cause paralysis for a long period without disorganizing changes. This is shown by the immediate disappearance of paralysis from displacement of vertebræ, in some cases after it had existed for years.

The PROGNOSIS, of course, varies in individual cases according to the nature of the tumor and the different causes of compression; but inasmuch as the nature of intraspinal tumors is rarely determinable with anything like precision, an opinion in cases of tumor concerning the probable course and termination must have another basis. In general, the prognosis is unfavorable in proportion as motor paralysis is progressive, as sensation is impaired, reflex excitability diminished, and as atrophy of the muscles takes place; in other words, in proportion as the symptoms denote destructive lesions of the cord.

In cases of pressure from displacement of vertebræ, recovery from paralysis or more or less improvement may be expected from measures to remove the cause of compression. In another work I have cited a case in which a patient who had been confined to the bed with paraplegia for three years, having suffered greatly during this period from pain and reflex muscular action, immediately recovered the use of his limbs under the treatment by Prof. Sayre according to his method of suspension and encasing the body in a plaster-of-Paris jacket.[1] This method secures extension of the spine and renders the spinal column immovable—objects of prime importance in cases of existing displacement and disease of the vertebræ leading thereto. As a means of securing rest it may be found to have a wider extent of useful application than has as yet been demonstrated.

The syphilitic alone, of the different tumors, offer encouragement in the way of medicinal treatment. Antisyphilitic medication sometimes accomplishes much, and it should be employed whenever there is ground for the suspicion that the spinal affection may be due to syphilis.

In other respects the treatment indicated is essentially the same as in cases of chronic primary spinal meningitis and myelitis.

Pseudo-hypertrophic (or Myo-sclerotic) Paralysis.

The characteristics of this affection were first pointed out by Meryon in 1858. It was, however, more fully described and its nosological individuality established subsequently by Duchenne. The affection is peculiar to infancy and childhood. It has been known, however, to occur at the age of twenty-six years. In a number of instances the disease has affected several members of the same family, the males oftener than the females.

The distinctive features are increase of the volume of certain muscles and diminished motor power. The muscles of the lower limbs are generally first affected, especially the gastrocnemii or the glutei. Subsequently the affection may extend to the muscles of the trunk and of the upper extremities. Corresponding muscles on the two sides may be affected, but there are many exceptions to this rule of symmetry. Not infrequently the affected muscles are irregularly distributed and the affection may be limited to portions of muscles. The increase of volume may be considerable, and the enlarged muscles are in striking contrast with those not affected.

The anatomical changes in the affected muscles are—hyperplasia of the areolar tissue, an accumulation of adipose tissue between the muscular fibres, and atrophy of the latter. As regards its pathological character the disease belongs in the category of sclerotic or cirrhotic affections. The enlargement of the muscles denotes "false hypertrophy."

The structural changes involve diminution of motor power proportionate to the compression and consequent atrophy of the muscular fibres. Seated in the muscles of the lower limbs, the affection causes a peculiar waddling gait, the limbs in walking being widely separated. If the extensor spinal muscles be affected, a lumbo-sacral anterior curvature takes place when the posture is upright. Frequently, the affection predominating in the flexor muscles of the foot, talipes equinus is a result of the greater action of the extensors, and with this is a claw-like deformity of the toes. After having reached a certain degree and extent, the affection may remain stationary for several years; but at successive epochs it is likely to increase and to become more and more diffused, until patients are reduced to a helpless condition. The progress of the affection is unattended by pain. There is neither hyperæsthesia nor anæsthesia. The functions of the bladder and rectum are unaffected. In an advanced stage the electro-contractility of the affected muscles is notably diminished or lost, but there is no reaction of degeneration. In a case reported by Dr. Balthazar Foster, after the lapse of five years the volume of the affected muscles became as much reduced as it had previously been increased.

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[1] Vide *Clinical Medicine*, 1879, p. 560.

It has been a pathological question whether this disease be a purely myopathic affection or whether it be primarily neuropathic. In a number of typical cases no lesion has been found in the spinal cord, so that the weight of evidence is in favor of the view that pseudo-hypertrophic muscular paralysis is due to a primary affection of the muscles of the nature described.

As regards the PROGNOSIS, a case reported by Benedikt is cited by Jaccoud in which a cure was effected by galvanism. Experience, however, thus far affords little encouragement to expect a cure from any measure of treatment. Electricity should be faithfully tried. By measures having reference to the general condition of the patient the affection may perhaps be prevented from progressing, and there may be more or less improvement. The disease rarely destroys life *per se*, death being due to some intercurrent affection. The duration in 13 cases in which the termination was ascertained, out of 85 cases analyzed by Dr. C. T. Poore, varied between two and thirty years.[1]

Scleroderma, or Sclerema.

The affection called scleroderma or sclerema, also sclerodermia and scleremia, may be here noticed, as it is analogous, in its pathological character and in certain of its effects, to the disease just considered. Other terms than those already given have been applied to it—to wit, scleremus, scleria, sclerosis, scleroma, and sclerymus, all derived from the Greek word *σκληρος* (hard). Its anatomical characteristic is the production, in the lower portion of and beneath the skin, of dense fibrous tissue in great abundance. This renders the integument notably rigid and resisting to pressure. It is closely adherent to the muscles. It cannot be moved over the muscle or pinched up in folds. The muscles are restrained in their movements by the density of the structures above them, as if a firm bandage were closely applied. More or less wasting of the muscles is caused by the pressure. Pigmentary matter is in most cases deposited in the cells of the rete mucosum, producing a discoloration which is yellowish, yellowish-gray, or dark-brown, and more marked in some situations than in others. These changes may be either limited to certain parts or diffused over the whole surface, but they vary in amount in different places.

The affection is extremely rare. A case under my observation was reported by Dr. Walter De F. Day in the *American Journal of Medical Sciences*, April, 1870. Dr. Day was able to collect for analysis reports of only 33 cases, and the case reported by him was the seventh on record in which there was an examination after death. The affection is not to be confounded with that called by some writers acute sclerema, which is peculiar to infants. The latter is a variety of œdema. The affection under consideration occurs generally in middle life. It occurs much oftener in women than in men. It is a chronic affection, usually beginning insidiously and progressing slowly, but exceptionally it may be developed rapidly with a sudden invasion. The upper part of the body is oftenest first affected, but it may appear primarily in any part.

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[1] *New York Journal of Medicine*, June, 1875.

The affection is not necessarily associated with disease of any of the internal organs, and if uncomplicated it is not incompatible with good general health. Existing in a notable degree, it obstructs muscular movements by mechanical pressure and restraint, leading at length to more or less impairment of motor power, by inducing atrophy. The surface is usually cool or cold, but the heat at times may be greater than normal. Sensibility is not impaired, and perspiration continues. The duration is variable. It may continue for months and years. A fatal termination is due to some complication or intercurrent affection. It continues in some cases indefinitely without improvement, and in other cases more or less improvement takes place without recovery, but complete recovery may be hoped for. The number of cases reported is, as yet, too few to determine the ratio of recoveries.

Various measures of TREATMENT were employed in the cases which have been reported. It remains to be ascertained what are the remedies, if there be any, which exert a curative effect. In the case under my observation arsenic was given for some time, and it seemed to be of service, but in this case there occurred grave complications—namely, pleuritis, pericarditis, and peritonitis. An analeptic course and tonic medication are rationally indicated, together with alkaline baths and emollient applications to the skin.

Myxœdema.

This remarkable affection, which was first described in 1873 by Gull, received the name myxœdema from Ord, to whom we owe the most complete study of the disease which has hitherto been made. The disease is characterized by an infiltration of the connective tissue of the body with a gelatinous fluid containing mucin.

Nothing is positively known concerning the CAUSATION. The disease has been observed more frequently in women than in men. It occurs in adult life, rarely at any other period. In a certain number of cases it has followed pregnancy. Mental distress has been thought by some writers to be an element in its causation. The disease seems to be more common in England and in France than elsewhere. In this country only a few cases have been reported.

But little is accurately known of the MORBID ANATOMY. In the few post-mortem examinations which have been made the connective tissue of the skin and the subcutaneous tissue are richly infiltrated with a jelly-like material containing mucin. According to Ord, the fibrillar and cellular elements of connective tissue are increased. This mucous infiltration and new growth of connective tissue are not confined to the skin, but they affect the mucous membranes, the glands, the muscles, the nervous system, and, above all, the outer coat of the arteries. The thyroid gland has been found atrophied, a condition also observed during life in many cases, but not in all. In the late stages of the disease the cortical substance of the kidneys is atrophied—a condition which may be secondary to the changes in the arterial walls.

The peculiar œdema begins generally in the face, producing a characteristic physiognomy. The eyelids, the lips, the alæ nasi, the cheeks—in fact, all of the features—are swollen. The œdema which causes the swelling differs from ordinary œdema in its firmness and resiliency. It does not pit on pressure and does not gravitate. The face acquires a dull, expressionless appearance, with an absence of all the finer lines of the features. The œdematous skin has an anæmic, alabaster-like appearance, with the exception of the cheeks, which present reddish spots or a more diffuse dull pinkish hue.

Next to the face, the hands are most markedly affected with the mucous œdema. They become swollen and shapeless, an appearance which has been described by Gull as "spade-like."

The same condition as that affecting the face and the hands may extend over the whole body, so that the skin everywhere is swollen.

The skin is usually dry and rough in consequence of the absence of perspiration.

As already remarked, in a large proportion of cases the thyroid bodies are atrophied. Ord mentions also the occurrence of tumefaction, with marked resilience of the skin, just above the clavicle.

In the majority of cases there are nervous symptoms no less characteristic than the mucous œdema. For a long time these symptoms consist only in a progressive hebetude affecting all mental and physical actions. The mental processes are slow and labored, but they may be for a long period correct. Speech is slow and conducted with a monotonous, and often hoarse, voice. All muscular movements require a long time in their execution, and they are often attended with a slowness of co-ordination which may suggest ataxia. Sensation is normal, save a certain tardiness in perception.

After a time mental aberration is likely to appear, and this is manifested in various forms of insanity attended with hallucinations.

Ord speaks of the frequent occurrence of two subjective symptoms referable to the special senses—namely, a persistent unpleasant taste and a persistent unpleasant smell. In some cases the occurrence of salivation has been noted.

The hair often falls out, and the teeth frequently decay. The appetite may be normal, but after a time it is generally disturbed. There is usually anæmia, which may be very profound. Albuminuria is common only in the later stages of the disease. The bowels are generally constipated. There is not only a subjective sensation of coldness, but the temperature of the body is actually lowered, ranging from 94° to 98°. Sometimes there is a difference in the surface-temperature of the two sides of the body.

The DURATION of the disease is long, varying between three and twenty years or more. The prognosis is unfavorable, but instances of improvement are not uncommon, and cases of recovery have been reported. Death may be due to gradual exhaustion, to uræmia, or to some complication. Bulbar paralysis has been observed as a complication.

There are various theories as to the nature of the disease, but they are all very hypothetical. A plausible assumption is that myxœdema is due to some trophic nervous disturbance; but attempts to define the nature of the trophoneurosis have not been successful. Some have surmised disease of the sympathetic system, others disease of the medulla oblongata.

Of considerable interest is the possible relation between myxœdema and atrophy of the thyroid gland. The facts bearing upon this relation are as follows: Extirpation of the entire thyroid gland in human beings for goitre has in some cases been followed by a peculiar cachexia, with mental disturbance, and even with firm œdema. This cachexia has been called cachexia strumipriva. The extirpation of the entire thyroid gland in dogs is nearly always fatal. Horsley has produced by the extirpation of the thyroid gland in monkeys profound anæmia and an œdema similar to myxœdema. The affection known as cretinism bears some resemblance, particularly in the mental symptoms, to myxœdema, and cretinism seems to be in some way connected with disease of the thyroid gland. Finally, myxœdema is often associated with atrophy of the thyroid gland. These facts lend some support to the hypothesis that there may be some causative connection between myxœdema and atrophy of the thyroid gland, but in what this connection consists we are wholly ignorant.

In the TREATMENT of myxœdema the prolonged use of jaborandi has been found in a few cases to have a favorable influence over the disease. From ten to sixty minims may be given four times daily. Arsenic, nitro-glycerin, and chalybeate preparations are recommended. Patients should be protected against cold, and the diet should be ample and nutritious. Baths and friction should enter into the treatment.

CHAPTER V.

FUNCTIONAL DISEASES OF THE BRAIN AND SPINAL CORD.

Coma.—Saturnine Encephalopathy.—Vertigo and Ménière's Disease.—Paralysis, Remarks on.—Treatment of Functional Paralysis.—General Paralysis.—Hemiplegia.—Paraplegia.—Acute Ascending Paralysis.—Nervous Asthenia, or Neurasthenia.—Spinal Irritation.

FOLLOWING the inflammatory and structural diseases of the brain and spinal cord, certain functional affections referable to these central parts of the nervous system may be more advantageously considered as forming a separate group than as included among the neuroses, which will occupy the concluding chapter of this section. The group will embrace the following affections: coma, saturnine encephalopathy, vertigo, general paralysis, hemiplegia, paraplegia, acute ascending paralysis, nervous exhaustion or neurasthenia, and spinal irritation.

It is to be understood, here and elsewhere, that the term functional is used provisionally, in a conventional sense, to distinguish diseases without any established anatomical characters. In a literal sense a purely functional disease probably has no existence. In other words, disturbance of function always involves some anatomical change; but in the diseases which, for the sake of convenience, are distinguished as functional either anatomical changes have not been ascertained or their pathological connection with these diseases is doubtful.

Mental disorders are properly embraced among the functional affections of the brain. They do not usually enter into the course of instruction in the Principles and Practice of Medicine. They are, however, to be included within the range of the studies of the physician. Lectures and treatises are devoted specially to these disorders, and their management constitutes one of the specialties of medical practice; but all practitioners are called upon to treat affections of the mind falling short of well-marked insanity. Melancholia and hypochondriasis in their lighter grades—that is, not amounting to insanity—come constantly under the notice of the physician in general practice. Want of energy, lack of buoyancy, mental apathy, and inertia often exist without any obvious disease. The physician should consider these as generally denoting corporeal ailment; and this should be impressed upon the minds of patients, since they are thereby led to reformations and measures of management which will be likely to afford relief, and are better enabled to strive against the depressing influence of mental disorder. A vast amount of unhappiness is due to causes which under intelligent medical direction may be removed, or to functional morbid conditions which judicious treatment may relieve. It is a grave error to attribute disorders of the mind exclusively to mental causes. These few remarks open up a subject of great importance. Insanity is to be prevented by the general practitioner. They who devote themselves to the treatment of the insane have not the opportunity of preventing the development of insanity.

The study of mental disorders is important to the physician as involving questions of medical jurisprudence. The existence or otherwise of insanity sufficient to require or warrant confinement in institutions for the insane, the exemption of responsibility for criminal acts, incompetency to perform legal obligations, to take care or dispose of property, etc., are practical questions concerning which physicians are called upon to give testimony; but for information bearing on these questions the student and practitioner are referred to works treating of disorders of the mind and of medical jurisprudence.

Coma.

The term coma expresses a state of unconsciousness from which the patient either cannot be aroused or is aroused with more or less difficulty. It is customary to apply this term to extreme degrees of unconsciousness, more correctly expressed by the terms lethargia and carus. The latter term denotes a degree in which the patient cannot by any efforts be aroused. This degree is generally called profound coma. Sopor expresses an abnormal degree of somnolence, or moderate coma. A patient in this state is often said to be semi-comatose. Coma differs from normal sleep qualitatively as well as quantitatively. Sleep is a physiological event of essential importance to the welfare of the economy. Coma is a pathological condition from which the system derives no benefit. It follows that coma is never a substitute for sleep.

Coma is a symptom occurring in most of those diseases of the brain which have been considered—namely, hyperæmia and anæmia of the brain, thrombosis and embolism, cerebral and meningeal hemorrhage, meningitis, encephalitis, and tumors. In all these affections, except perhaps active hyperæmia, the rationale of the coma is a diminution of the supply of oxygenated blood to the brain-substance. Coma may be an effect of cerebral anæmia (ischæmia) due to impoverishment of the blood and enfeebled circulation, as in the hydrencephaloid affection first described by Marshall Hall. (Vide p. 649.) Coma is a pathological element in certain of the neuroses which remain to be considered—namely, epilepsy, hysteria, and catalepsy. It is an incidental event in various diseases exclusive of those of the nervous system; examples of which are acute and chronic affections of the kidneys, acute yellow atrophy of the liver, pernicious intermittent fever, scarlet fever, smallpox, typhus and typhoid fever, etc. It may be produced by such toxic agents as opium and other narcotics, alcohol, ethers, chloroform, etc. The sudden loss of consciousness at the instant of the attack in epilepsy is attributed to cerebral anæmia caused by spasm of the arteries within the brain. Of the other instances, the causation of the coma involves a toxical agency certainly in some, and probably in most if not in all cases. The nature of these agents in the general diseases just named and their *modus operandi* are not understood.

There are cases of coma not connected with any of the foregoing diseases, and not clearly referable to any known morbid condition of the brain. The coma is then to be considered and treated as an individual affection. It is, of course, in these as in other cases merely a symptom, but the pathology, and perhaps the etiology, are unknown or conjectural. The coma may be greater or less in degree, sometimes not exceeding stupor, and sometimes so profound as to constitute carus. It may occur suddenly, constituting a veritable apoplectic attack, or it may be developed more or less slowly. It may last for a few hours or it may continue for several days. It may end, sooner or later, either in death or recovery.

In fatal cases morbid conditions may be found after death which were not determinable during life; examples are—thrombosis of the basilar artery or of the longitudinal sinus and meningeal hemorrhage. The two former of these conditions not many years since would have escaped recognition as causes of the coma; and so further developments in pathological anatomy may furnish an explanation, post-mortem, of cases in which adequate morbid appearances are not now discoverable. In the progress of etiology, too, toxical agents causing coma may be discovered, of which now nothing is known. This is not an unreasonable expectation, in view of the fact that urea and cholesterin have only within a few years been recognized as coma-producing principles.

Coma in some instances seems attributable to cerebral exhaustion; that is, it follows prolonged or excessive mental activity in conjunction with impaired nutrition and general debility. It must be confessed that the term cerebral exhaustion does not embody any clear idea of the pathological condition which the coma represents, but it has a practical significance in relation to the causation and treatment. Of course, in deciding that coma is to be regarded as a functional affection the various diseases and determinable morbid conditions with which it is known to have pathological connections are to be excluded. Recognizable internal toxical agents, *e. g.* urea, and those which are external, *e. g.* opium, alcohol, etc., are also to be excluded. The diagnosis involves a careful investigation of the causation in these different directions.

Assuming a case in which the pathology and etiology are not determinable, the prognosis and treatment must be based upon circumstances pertaining to the coma. Other things being equal, the prognosis is unfavorable in proportion to the degree and duration of the coma. If the patient cannot be aroused sufficiently to open the eyes or protrude the tongue, the danger is great, and it increases with the continuance of the comatose state. When the ability to swallow is lost a fatal ending is to be expected. *Per contra*, if the coma be moderate in degree the prognosis is favorable. Absence of paralysis, the temperature of the body not increased, mobility of the iris, the pupils neither contracted nor dilated and equal in size, the ophthalmoscopic appearances normal, the respirations quiet and of regular rhythm,—these are points of importance in the exclusion of grave cerebral diseases and in their bearing upon a favorable prognosis.

The TREATMENT is to be governed by the symptomatic indications in individual cases. These rarely, if ever, point to depletion by bloodletting or other depressing measures. The opposite treatment is usually indicated by feebleness of the circulation and general debility. Supporting treatment is especially indicated in feeble subjects and when cerebral exhaustion is an etiological factor. It might prove a serious error, under these circumstances, to pursue therapeutical measures on the supposition of the existence of cerebral meningitis or hyperæmia.

The propriety of counter-irritation is doubtful. Rest, alimentation, nerve-tonics, and the judicious employment of wine or other alcoholics are the measures which generally meet the indications present.

Saturnine Encephalopathy.

A rare form of poisoning by lead is characterized by cerebral disturbances of a grave character—namely, coma, convulsions, and delirium. These three events are combined in a considerable proportion of cases. An attack may occur either abruptly or after certain premonitory symptoms. These are—headache, vertigo, ringing in the ears, and in some instances amaurosis. Other manifestations of lead-poisoning—paralysis, arthralgia, colic—may or may not precede the encephalopathic attack. The coma is more or less profound. In favorable cases it disappears after having continued for one or two days. In unfavorable cases it increases in degree and continues until death. The convulsions are epileptiform in character (eclampsia vel epilepsia saturnina). Paroxysms lasting from five to twenty minutes occur after variable intervals. The delirium is in some cases active and violent, and in other cases hilarious. It is sometimes characterized by hallucinations of sight and hearing. Either of the three events may be predominant. Coma and convulsions in some instances occur without delirium, and coma between the paroxysms of convulsions is sometimes wanting. Amaurosis, if it do not precede, is likely to accompany, the attack. The amaurosis is not caused by retinal changes, and the ophthalmoscope shows the normal appearances.

A satisfactory pathological explanation of these effects of lead-poisoning cannot be given. They have been attributed to anæmia of the brain from changes in the minute cerebral arteries, to œdema of the brain from increased arterial pressure, and to the direct irritation caused by the presence of lead. Uræmia is probably a pathological element in some cases. The urine is diminished in quantity, and has been found to contain albumen. The effects, however, are not in all instances referable to uræmia, inasmuch as the evidence of diminished elimination of urea is not always present. Heubel has produced similar effects in dogs by poisoning them with lead, and this is one of the recognized causes of the so-called gouty or small, granular kidney.

The PROGNOSIS is grave in proportion to the frequency and violence of the convulsions, together with the degree and continuance of the coma. The rate of mortality has not been definitely ascertained, but the proportion of fatal cases is large.

There is little to be said with reference to the TREATMENT. Depletory or debilitating measures are contraindicated. It should be ascertained, by examinations of the urine, how far uræmia may be a factor in causing the coma and convulsions. Hydragogues and sudorific measures are indicated if there be ground for supposing that there are symptoms of the accumulation of urea in the blood. The inhalation of chloroform or the administration of the chloral hydrate may be resorted to in order to control the convulsions. The iodide of potassium should be given to the extreme of toleration.

Vertigo—Ménière's Disease.

Vertigo is a symptom occurring in various cerebral diseases, and also when it is to be regarded as an individual functional affection. In this article it is considered as a functional affection and as a symptom of Ménière's disease. It occurs as a paroxysmal affection. A person seemingly, perhaps, in good health suddenly becomes giddy, surrounding objects appear to be revolving, or the person seems to himself to be performing movements of gyration, which, according to Charcot, always appear to be from the left to the right. The sensation is like that caused by long-continued rotatory movements of the body. If the vertigo be moderate or slight, it simply renders the person unable to walk steadily. He reels like a drunken man. In a greater degree walking is impossible, and there may be inability to stand without support. In some instances the patient falls, but without loss of consciousness and without muscular spasm of either the facial or other muscles. The attack is accompanied by a sense of prostration, which, however, is due in no small degree to apprehension. Nausea is a frequent symptom, and vomiting sometimes occurs. The paroxysm varies much in duration in different cases. It may last for a few moments or the vertigo may continue for hours and days.

Successive attacks recur after variable intervals. Days, weeks, or months may intervene. In some cases vertigo for a certain period may be produced by slight exciting causes, such as any intellectual effort, sudden muscular exertion, looking upon a bright light, etc. A patient may be exempt while in the recumbent posture, but he may experience vertigo whenever the attempt is made to sit up or stand. For this reason patients sometimes keep the bed for a long period.

Attacks of vertigo generally give rise to great apprehension. The patient fears that apoplexy, paralysis, or a fit of some kind is impending. If the apprehension be not removed by positive assurances from the physician of the absence of danger, it may lead to a distressing state of despondency. The patient is afraid to walk or to be left alone, and all public assemblages are avoided, lest something may happen to him to excite disturbance. In a case which has been under my observation the patient, a gentleman in middle life, notwithstanding assurances of the absence of danger, has for many years been constantly attended by either a relative or a servant, although a long period has elapsed since an attack occurred and his health is excellent. This state of anxiety has repeatedly, in cases under my observation, continued for months and years. The physician is fully warranted in giving positive assurances that these attacks of vertigo are devoid of danger. Of a large number of cases which I have observed, in no instance has apoplexy, paralysis, epilepsy, or any serious affection followed. It is an unfortunate error to state, as is done by some writers, that vertigo is premonitory of the cerebral affections just named. The needless apprehension of patients is sometimes encouraged by the fact that it is shared by the physician. I have met with cases in which danger of apoplexy and paralysis has been represented to the patient. Charcot relates a case in which the attacks were confounded with epilepsy, the patient having for a long time been under treatment for that disease. Assurances of the absence of danger may do much good, not only by removing needless apprehension, but in the way of effecting a cure; for the mental anxiety occasioned by the attacks increases the liability to their recurrence.

Of the PATHOLOGY of vertigo more cannot be said than that it represents a functional cerebral condition. Its etiological relations are better understood. A French writer, Ménière, in 1861 pointed out the supposed connection of a disease characterized by deafness and vertigo with an affection of the semicircular canals of the internal ear. Subsequent clinical observations have established this causation in a certain proportion of cases. The vertigo is not infrequently preceded and accompanied by tinnitus aurium, sometimes referred to one ear and sometimes to both ears. Sudden deafness in one ear or in both ears has been observed to be coincident with the attacks of vertigo. The attacks have been found to cease on the cure of suppurative otitis, and even on the removal of an accumulation of wax or a foreign body from the meatus.[1] The most rational explanation is that a reflex influence exerted upon the brain emanates from the aural organs. The affection in these cases is now known as *Ménière's disease.*

All cases of functional vertigo are not cases of *vertigo ab aure læsa.* In perhaps the larger proportion there is no evidence of aural disturbance. Dyspeptic ailments appear to be causative in some cases, but not constantly as contended by Trousseau, who called the affection stomachal vertigo (*vertigo a stomacho læso*). It is sometimes fairly attributable to prolonged overtasking of the mental faculties and anxiety of mind. Excessive venery, the immoderate use of tobacco, and intemperate indulgence in coffee or tea have a causative influence. It may be incident to the debility which remains after convalescence from a severe disease; for example, typhoid fever. DaCosta attributes it in some cases to lithæmia (uricæmia).[2]

The TREATMENT involves, as the first and most important measure, removal of the supposed cause or causes. The ears should be carefully examined with reference to removable morbid conditions. Measures addressed to cerebral congestion—namely, bloodletting, counter-irritation, purgation, and reduced diet—are hurtful. On the contrary, tonic remedies, a nutritious diet, and hygienic means of invigorating the general health are generally indicated. The practioner should bear in mind the importance of assuring the patient positively of the absence of all danger. This will sometimes do much toward the cure. A cure is often soon effected, but in some cases the disorder persists for a long time. Charcot extols the efficacy, in cases of aural vertigo, of quinia given in full doses (20 grains) for several weeks.

Paralysis—General Remarks.

Paralysis is an important symptomatic event in the clinical history of different cerebral and spinal diseases which have been already considered. It is not always referable to either an inflammatory or a structural disease, and it is then to be distinguished as functional.

By the term paralysis is understood either the loss or an abnormal diminution of voluntary power over muscles. The term is also applied to the loss or a diminution of sensibility. The latter is sensory and the former motor paralysis. Sensory paralysis, however, is expressed by the terms *anæsthesia* and *analgesia;* the first relating to the sensibility to touch, and the second to the sensibility to pain. Analgesia may exist without anæsthesia. A patient, for example, partly under the influence of chloroform or ether, may feel the cutting of the knife in a surgical operation, but without any sensation of pain. On the other hand, the loss of sensibility to touch (anæsthesia) carries with it the loss of sensibility to pain (analgesia). Cutaneous analgesia is ascertained, clinically, by pinching or pricking the skin. Anæsthesia is ascertained by measuring the distance from each other at which two points in contact with the skin are recognized. An instrument devised for this purpose, called the æsthesiometer, may be used, but the common compasses used by the draughtsman will answer as well, and, if these be not at hand, two pins will suffice. The farther the two points of contact must be removed from each other in order that each may be separately felt, the less the sensibility to touch. The sensibility is undiminished if the patient be able to feel two points of contact approximated as closely as they are when appreciable in health. The sensibility to touch differs in different parts of the body considerably in health. It is most acute on the prolabia, the tip of the tongue, and the ends of the fingers. These normal differences are to be taken into account in judging of anæsthesia, and also variations within normal limits in different healthy persons and in corresponding situations on the two sides of the body. Moderate deviations from the average degree of sensibility, as thus tested, or from an exact uniformity of the two sides, are not to be considered as abnormal without corroborative evidence of sensory paralysis.

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[1] Vide "A Clinical Analysis of the Inflammatory Affections of the Inner Ear," by H. Knapp, in *Archives of Ophthalmology,* 1871, vol. ii. No. 1.

[2] Vide *Am. Journ. of Med. Sciences,* Oct., 1881.

Motor paralysis is expressed by the term *akinesia* or *akinesis*. The term paralysis, however, is generally used instead of this term, and if not designated as either sensory or motor the latter is understood. Paralysis may affect either the voluntary or the involuntary muscles. The heart, œsophagus, stomach, intestines, bladder, and arteries may be the seat of akinesia. This pathological condition enters into the consideration of these organs in other sections of this work.

Under the name *erythromelalgia* Dr. S. Weir Mitchell[1] first called attention to an affection characterized by vaso-motor paralysis of the vessels of the extremities. Males more than thirty-five years of age are most frequently affected. The symptoms are burning pain in the feet, rarely in the hands, accompanied by flushing and congestion of the part, which may be somewhat swollen. The temperature of the part is increased. The sensation is normal or it may be sometimes increased. There are no trophic troubles. Erythromelalgia is an interesting counterpart to the disease known as local asphyxia or symmetrical gangrene, which is caused by vaso-motor spasm.

Paralysis is complete when there is total loss of voluntary power over the paralyzed muscles. It is incomplete when this power is not entirely lost, but is more or less diminished. The term *paresis* is often used in a sense equivalent to that of incomplete paralysis. Incomplete paralysis or paresis differs, in different cases, between the slightest appreciable impairment of the normal motor power and a degree approaching as closely as possible completeness. Paralysis is general when it embraces the muscles of the four extremities, either with or without the muscles of the face. It is partial when a portion of the muscular system is involved, as in hemiplegia and paraplegia. It is local when a very few muscles, or perhaps a single muscle only, are paralyzed.

Paralysis may be *neuropathic* or *myopathic*. A myopathic paralysis depends on a primary morbid condition of the muscles paralyzed. A neuropathic paralysis depends on some cause which either prevents the action of volition upon the cerebral fibres which are continued into the nerves going to the paralyzed muscles, or obstructs the transmission of volition at some point between the muscles and the part of the brain in which the voluntary act originates. For the performance of voluntary motion there are three requisites—namely, the communication of the voluntary influence to certain parts of the brain, the conduction of this influence from the brain through motor nerves, and the ability of muscles to contract in obedience to the will. Paralysis may depend on causes involving primarily either of these three requirements.

An important division of neuropathic paralyses is into central and peripheral. Central paralyses depend on morbid conditions seated in either the brain or spinal cord, and they may be further divided into cerebral and spinal. Peripheral paralyses depend on morbid conditions affecting nerves at any point between their terminations and their central connections with either the brain or the spinal cord. Paralyses are peripheral when they depend on affections of cranial or spinal nerves within the skull or spinal cord.

There are certain points of difference in the effects of cerebral, spinal, and peripheral paralysis, respectively, which are of practical importance as regards localization of causative conditions and differential diagnosis. In cerebral paralyses the reflex excitability of the affected muscles is retained and may be increased. The electrical excitability is also the same as in health, or it may be increased. The muscles do not speedily become atrophied. The cutaneous sensibility is often undiminished, or it is not diminished in proportion to the motor paralysis, and is regained in a short time, although the loss or impairment of the voluntary power over the muscles remains. In spinal paralysis the paralyzed muscles may or may not retain their reflex and electrical excitability, the differences in these respects being dependent on the situation of the affection of the cord, together with the degree and the extent of lesions. The reflex excitability is sometimes notably increased. Sensibility may be either retained, diminished, or lost. There may or may not be trophic disturbances. These points have entered into the clinical history of the different spinal affections which have been considered.

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[1] *Am. Journ. Med. Sciences*, July, 1878.

The characteristics of peripheral paralysis will be considered in the next chapter. The reaction of degeneration, which is of much value in the diagnosis of the different kinds of paralysis, has already been described (p. 733).

The foregoing remarks relate to paralysis dependent on different pathological conditions. Paralyses which are dependent on molecular changes not appreciable, or on histological lesions not yet ascertained, and which are therefore to be considered at present as functional, generally end in recovery. Their duration is variable. It may be but a few hours, and even minutes, or, on the other hand, days, weeks, and months. The paralysis is in some cases intermittent. The recovery is sometimes abrupt, and sometimes it takes place after a more or less gradual improvement. The paralyzed muscles do not waste, except as an effect purely of disease. There may be anæsthesia or the cutaneous sensibility may remain unaffected. In functional paralyses the electrical excitability is retained, and the phenomena of the "reaction of degeneration" are wanting. Reflex excitability is sometimes retained and sometimes diminished or lost. The circumstances under which the paralyses occur, and their pathological relations, are of importance in the discrimination. The paralyses which often follow diphtheria, and sometimes other acute febrile diseases, are generally to be considered as functional, although these diseases may be followed by organic paralyses, usually of spinal or of peripheral origin. The so-called hysterical paralyses are examples of typical functional paralysis. The association of paralysis with these affections is often of significance with regard to diagnosis. Causative circumstances are to be taken into account. Functional paralyses may be referable to exhaustion from prolonged muscular exertions and to the action of cold. There is no pathological propriety in calling the paralysis attributed to the latter cause rheumatic; and clinical experience affords no proof of any relationship between paralysis *a frigore* and rheumatism.[1] The so-called reflex paralysis will be presently noticed in connection with paraplegia. Finally, the functional character of paralyses may be inferred from the probable causation by certain vegetable poisons, such as ergot, tobacco, lathyrus sativus, absinthe, camphor, etc. Saturnine paralyses may be regarded as peripheral, and these will be considered in the next chapter. Other differential points relate to the particular varieties of functional, cerebral, and spinal paralysis—namely, general paralysis, hemiplegia, and paraplegia—and will be noticed in connection with these disorders.

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[1] The usage of German writers in designating as rheumatic all affections supposed to be produced by exposure to cold has been adopted by some English writers, but it is not to be recommended. Under the name "paralysis from commotion" German writers embrace cases in which the affection is due to injury, but without appreciable lesions. (Vide Eichhorst, *Handb. d. Spec. Path. u Therap.*) Cases in which paralysis occurs after a variable period dating from railway accidents have been described, especially by Erichsen. These cases have been considered as important in a medico-legal point of view.

Treatment of Functional Paralysis.

In the treatment of functional paralysis a primary object is the removal of supposed direct or indirect causative agencies. Exhaustion from over-exertion of body or mind is an indication for rest. Toxical agents, tobacco, etc. should not continue in operation. Anæmia claims appropriate therapeutic and hygienic measures. Cystitis, phimosis, or other local diseases which may be suspected of standing in the relation of causes by means of a reflex influence or in other ways should receive proper attention.

Of remedies having direct reference to the paralysis, electricity holds the first rank. Experience shows the usefulness of both the faradic and the galvanic current, individual cases differing as regards the superiority of the one over the other. There need not be that delay in resorting to electricity which is an important precaution in cases of paralysis dependent on diseases involving inflammation. Care, however, is to be taken not to employ too strong currents, or to prolong or repeat unduly their applications. For practical details the reader is referred to works which treat specially of electricity as a curative agent. Its curative efficacy must consist in its effects upon the functional activity of the nerves and muscles involved in the paralysis, on the circulation in these parts, and on their nutrition. Its action is that of an excitant, and it is evident that if carried too far it may do harm rather than good. The success of electrical treatment in all paralytic affections will depend on the exercise of judgment in determining the indications in particular cases, and on skill in its employment. The advantage of experience in its practical application, together with the time and appliances which it requires, renders it desirable that some physicians should give to it special attention. Hitherto, the employment of electro-therapeutics in this country has been too much in the hands of uneducated practitioners who know but little of the agent which they employ, and still less of the human organism.

Friction and massage, or kneading with deep pressure, are important measures of treatment. Stimulating liniments facilitate the rubbing, and are to a certain extent in themselves useful. These measures are serviceable by promoting the circulation and maintaining nutrition. They should be systematically carried out and persisted in. If the paralysis be complete, passive movements are important. They should be resorted to daily, and continued as long as they are tolerated without fatigue. If the paralysis be incomplete, daily exercise of the paralyzed muscles by means of the will is of more importance than any remedial measure. As much exercise of the paralyzed muscles as can be taken without fatigue is desirable. The physician should impress the importance of this measure, the more because patients are slow to appreciate it.

Douches of alternately cold and warm water are useful. They should not be continued long enough to be followed by uncomfortable sensations; on the contrary, it is desirable that they should give rise to agreeable effects. Benefit is also often derived from the use of the sponge-bath followed by brisk friction.

Of drugs, strychnia or nux vomica and phosphorus are to be recommended. Whether or not they have any specially curative effect, clinical experience affords evidence of their utility.

General Paralysis.

The disease known as general cerebral paralysis, general paresis, or the paralysis of the insane (dementia paralytica) is fully considered in works devoted to the subject of insanity. Exclusive of this disease, general paralysis referable to intracranial morbid conditions is rare. It may be a result of two attacks of hemiplegia, the first attack affecting one, and the second the other, side. Hemorrhage in the central portions of the pons varolii or in the medulla oblongata may give rise to double hemiplegia, or, in other words, general paralysis. Instances of general paralysis involving muscles supplied by cranial nerves are among the curiosities of clinical experience. In a case under the observation of the late Prof. Coventry power was lost over all the voluntary muscles except the orbicularis palpebrarum and the respiratory muscles. Paralysis following diphtheria has been known to involve not only the extremities, together with the neck and trunk, but the muscles of the face, throat, and mouth. The intellect in these cases may be intact; excito-motory or reflex movements perhaps remaining unaffected, respiration, deglutition, digestion, and all the functions necessary to existence going on, but, as regards voluntary manifestations, the patient being in a state of living death. General paralysis involving cranial nerves has also been known to occur in connection with hysteria. In a case for many months under observation complete paralysis of the four extremities coexisted with incomplete bilateral paralysis of the face. The paralysis in this case was attributed to lead.

In the great majority of cases general paralysis is spinal. The seat in the spinal cord is to be inferred whenever cranial nerves are not involved and there are no symptoms denoting cerebral disease. General spinal paralysis, as has been seen (vide Chapters II. and IV.), is incident to inflammatory and structural affections of the spinal cord and its meninges. These are to be excluded in arriving at the conclusion that the paralysis is functional. The paralysis may be complete, or there may be a greater or less degree of paresis, and it may or may not be associated with anæsthesia. It follows diphtheria in rare instances, and is sometimes connected with hysteria. It was produced by woorara in a case of rabies, to which reference will be made in treating of that disease. It may be caused by exhaustion and exposure to cold. This causation was exemplified in the case of a man forty-five years of age, who during a severe snowstorm rode eight miles in a horse-car, standing on the rear platform, his back exposed to the storm. After reaching home he became unconscious for a short time, and on recovering his consciousness there was complete loss of motion and sensation below the head. He was disposed to somnolency, but was easily aroused, and his mental faculties were intact. I saw him on the fifth day after the date of the attack. Under a treatment consisting of stimulants, frictions, and nutritious food he had, in a measure, recovered from the paralysis. This treatment was continued, and in a short time the recovery was complete.

In functional spinal paralysis the paralytic condition may be developed rapidly or more or less gradually. The muscles of all the limbs may be simultaneously affected, or the paralysis may begin in one limb, the other limbs becoming successively affected. The bladder and rectum are rarely involved. The respiratory movements, as a rule, are not affected. Exceptionally there is much suffering from dyspnœa. I have known paralysis of the diaphragm to occur, as shown by the purely costal breathing and by inability to sneeze, although an intense desire and grimaces were produced by the introduction of snuff into the nostrils.

Recovery may take place after a few days or it may be delayed for many months.

Strümpell has reported a remarkable case of complete anæsthesia of the whole surface of the body, and in a nearly equal degree of the accessible mucous membranes. All kinds of sensation were lost. There was no sense of smell or of taste. There was complete amaurosis of the left eye and partial deafness of the right ear. The muscular sense was gone. The patient was not conscious of muscular contractions or of fatigue. Some reflex action was preserved. There was motor paralysis of the right arm and leg, but none on the left side. The movements of the non-paralyzed limbs were normal when controlled by the eye. If the eye in which the sight was preserved were closed, the movements were uncertain, although not ataxic. The patient at once fell asleep when the eye and the ear in which the perceptions were preserved were closed.

Hemiplegia.

Except when due to cerebral affections which have already been considered—namely, hemorrhage, softening from thrombosis or embolism, encephalitis, and tumors—hemiplegic paralysis is of infrequent occurrence. Examples of functional hemiplegia are as follows: Cases have been reported in which it took place suddenly during strong mental excitement, as in a fit of violent anger, recovery being sometimes speedy and sometimes slow. Todd designated the affection, in these cases, *emotional hemiplegia.* It sometimes follows epilepsy. In a case under my observation in which epileptic paroxysms recurred at short intervals for several days, hemiplegia occurred before the series of paroxysms had ceased, the face as well as the limbs on one side being paralyzed. The paralysis disappeared completely after a few days. Hysterical paralysis is sometimes hemiplegic. The face and tongue are not involved, and as a rule the left side is paralyzed. Hemiplegia may follow chorea, the limbs paralyzed being those of the side more affected with the choreic movements. The paralysis in these cases does not extend to the face or tongue. The paralysis which follows diphtheria is sometimes hemiplegic. I have known transient hemiplegia to follow insolation.

The following cases, stated as concisely as possible, will serve to illustrate hemiplegia from either embolism or thrombosis not giving rise to softening and hysterical hemiplegia:

A merchant, aged seventy-five, of active habits, attending daily to business, left his home in the morning in his usual health. He was observed to act strangely. He walked with difficulty and had trouble in moving his left hand. He was prevailed upon to return home in a carriage, although he insisted that nothing was the matter with him. In an hour after his return I saw him. He was somnolent, but easily aroused. There was paresis of the left upper and lower limbs and of the left side of the face. The tongue was deviated to the left side. On the following day there was marked improvement. The improvement continued, and on the sixth day recovery was complete, and he insisted on attending to his business as usual. There were no signs of cardiac disease. A little laurel-water and aconite were the drugs prescribed. He was allowed at once a nutritious diet. Six years afterward there had been no return of the paralysis and the patient was in good health. Thrombosis was the probable causative condition in this case.

A man, aged about seventy, after a long walk in the afternoon retired at night feeling well, and slept soundly until morning. On awakening there was paresis of the upper and the lower limb on the right side. The face and tongue were not involved, but there was aphasia, the patient being able to speak but a few words. Aside from these symptoms he had no ailment. His appetite was unaffected. The paralysis and aphasia disappeared in the course of a few days, no treatment being employed. Three years afterward he had had no return of paralysis and was in good health. The diagnosis in this case lies between thrombosis and embolism.

A case at my hospital clinic exemplified the occurrence, first of embolism, and afterward of thrombosis. The first attack was sudden, without loss of consciousness, and the paralysis was on the right side. The paralysis was complete, but after an hour it had entirely disappeared. Several months afterward hemiplegia again occurred. In this attack the paralysis was developed gradually, becoming nearly complete in the course of a week. In a few weeks there remained only a slight diminution of motor power.

Intermittent hemiplegia of very brief duration was exemplified in the case of a clerk, aged forty years, who at breakfast, while apparently in perfect health, had a sudden attack of complete hemiplegia of the right side, without loss of consciousness or any confusion of ideas. The paralysis disappeared completely in the course of an hour. During the following two weeks he had ten or twelve recurrences of the hemiplegia, the paralysis lasting for a few moments only, his health otherwise being perfectly good. The attacks then ceased to recur, and I learned several months afterward that he was well. This patient had had rheumatism, but there were no signs of cardiac disease.

A dressmaker, aged eighteen, a patient in Bellevue Hospital, exemplified hysterical hemiplegia. Three weeks before her admission, having retired perfectly well the previous night, in the morning she was unable to move the left lower limb. Sensibility was also lost. Toward evening she suddenly lost sensibility and the power of motion in the left upper limb. After a week she recovered power over the upper limb. The paralysis of the lower limb persisted. In walking with the aid of a crutch she dragged the paralyzed limb. There was no facial paralysis and the tongue was not deflected from a right line. There were no symptoms of cerebral disease. She had retention of urine, requiring the catheter, but after a time she succeeded in urinating, the physician in charge refusing to continue the use of the catheter. She was notably hysterical, and the globus hystericus was well marked. A month after her admission she was much frightened by a thunderstorm, and had hysterical convulsions. The cold douche promptly arrested the convulsions, and on recovering full consciousness she at once regained, almost completely, power over the paralyzed limb. The next day she walked without difficulty, and the following day went out of the hospital on a permit. Suddenly, after the lapse of a week, she again became hemiplegic on the same side as before. She recovered in a short time under the use of the shower-bath. She subsequently had paraplegia, with analgesia and anæsthesia. She recovered, and was discharged from the hospital well. This case illustrates the occurrence of *hemianæsthesia*, which in this instance and in the larger number of cases is of hysterical origin. Hemianæsthesia may be produced also by cerebral lesions. (See p. 674.) The loss of sensation may affect all forms of sensation, including the special senses.

Spinal hemiplegia, with motor paralysis on one side and anæsthesia on the opposite side, probably always implies a lesion limited to the half of the cervical portion of the cord on the side of the akinesia.

Instances have been observed of paralysis affecting the upper limb of one side and the lower limb of the other side. This form of paralysis has been called *hemiplegia cruciata* (not to be confounded with alternating paralysis, p. 674), and may be produced by lesions situated at the crossing of the pyramids in the medulla oblongata.

Malingerers sometimes feign hemiplegia. They are likely to pretend that there is loss of sensation as well as of motion; and this should excite suspicion. Hughlings-Jackson has pointed out a method of discovering this deception. The malingerer keeps the arm close to the body when asked to stoop forward. In doing this he exerts a voluntary power over the limb. In hemiplegia when the patient stoops the palsied arm falls forward.

Paraplegia.

Paraplegia is to be considered as functional when the absence of any anatomical change is to be inferred from the symptoms and course of the affection. As a functional affection it is much more frequent in its occurrence than general paralysis or hemiplegia. It is the most frequent form of hysterical paralysis. I have met with a case in which repeated attacks had occurred between the ages of seven and ten years, the patient being subject to other hysterical manifestations. It is a not infrequent sequel of diphtheria. It may be referable to exhaustion and exposure to cold. The following case is probably an example of sexual excess as a causative agent: A young woman admitted into Bellevue Hospital stated that she found both lower limbs completely paralyzed on awakening in the morning. She was subject to nocturnal attacks of epilepsy, and a paroxysm occurred during the night when the paralysis took place. She was a prostitute, and had had sexual intercourse with several persons on that night. There was complete loss of sensibility. Slight reflex movement could be excited. On the ninth day after her admission recovery of motion and sensation was complete. and she was well enough to be discharged on the thirteenth day, having had no treatment directed specially to the paralysis.

The subject of *reflex paralysis* has relation chiefly to paraplegia. Many clinical observers within late years have reported cases of paraplegia associated with various affections of different organs—the kidneys, bladder, uterus, ovaries, intestines, etc.; and a causative connection has been inferred from the fact that patients recover from the paralysis after recovery from their local affections. The latter, it is supposed, induce paralysis by a morbid influence transmitted through the reflex system of nerves; and hence the significance of the name *reflex paraplegia.* In order to establish a causative connection between different local diseases and paralysis it is necessary to show that the former precede the occurrence of the latter in a number of cases too large to be explained by mere coincidence. That facts sufficient to show this have been accumulated may fairly be doubted; and when, on the other hand, it is considered that the various local diseases supposed to have a causative connection are not associated with paralysis in the vast majority of cases, such a connection, when the association exists, is conjectural.

The pathological explanations of reflex paralysis are far from satisfactory. The theory of Brown-Séquard is that a reflex influence causes spasm of the arteries of the spinal cord, and that the paralysis is due to anæmia and defective nutrition thus induced. Assuming spasm of the vessels limited to a segment of the cord to be caused by a reflex influence, that this condition is persistent enough to produce the pathological effects attributed to it is highly improbable. Another explanation is that the supposed reflex influence has a paralyzing effect exerted through the vaso-motor nerves upon the arteries, and causes paraplegia by inducing congestion of the cord; but paraplegia does not take place in connection with diseases which must induce spinal congestion, such as the cold stages of intermittent fever, dilatation of the right side of the heart, etc.; and it is, to say the least, conjectural to include congestion of the cord among the causative conditions giving rise to paraplegia.

In some reported cases of supposed reflex paraplegia lesions of the spinal cord have been found after death. Leyden, Duménil, Weir Mitchell, and others think that the extension of neuritis centripetally sometimes to the spinal cord will explain the occurrence of paralysis in the cases in which it is supposed to be due to a reflex influence.

In view of these considerations the theory of reflex paralysis lacks a solid foundation.

Paraplegia may be an imagined disease. I have met with an instance in which this self-deception was diagnosticated, the case being analogous to one reported by J. Russell Reynolds, and called by him paralysis "dependent on idea." These cases are to be distinguished from cases of simulated paraplegia. I have met with the latter in connection with hysteria. Kunze relates the case of a woman who, having kept the bed for a considerable time under the conviction that she had paraplegia, on the occasion of the house taking fire resumed instantly the use of her limbs, and on reaching the house of a relative at once became again a paraplegic. It is not easy to discriminate imagined paraplegia, inasmuch as hysterical paralysis may instantly be removed by a mental impression. This is seen in cases of aphonia from laryngeal paralysis. I have given the synopsis of a case in which paraplegia disappeared under the influence of fright during a thunderstorm. Caution is to be observed in the diagnosis; and if made the physician is to use discretion either in asserting that the supposed paralysis is imagined, or in employing electricity or some other method of treatment with reference to a physical influence, conjoined with a confident assurance that the treatment will be successful.

Acute Ascending Paralysis.

Under this name Landry in 1859 described a disease characterized by rapidly-developed paralysis, beginning in the lower extremities and extending to the upper extremities, and often to muscles supplied by nerves originating in the medulla oblongata. Sensation and the functions of the bladder and rectum generally remain normal. No anatomical lesions explaining the paralysis have been discovered. The disease is sometimes called *Landry's paralysis*.

After prodromic symptoms of an indefinite character, with slight fever, lasting for a few days and sometimes for several weeks, paresis of the lower extremities occurs. The paresis begins in the feet, and in the course of a few days extends over the limbs, the paralysis increasing in degree and becoming complete. The paralysis extends to the trunk, and thence to the upper extremities. The respiratory muscles are affected, and dyspnœa becomes a prominent symptom. Subsequently the muscles concerned in speech, mastication, and deglutition are involved. Death takes place by apnœa and by exhaustion from innutrition. The duration varies between a few days and several weeks, the average being from eight to twelve days.

The intellect remains intact, and those cranial nerves only are implicated which arise from the medulla oblongata. Sensibility of the skin and muscles is but little, if at all, affected. There is sometimes hyperæsthesia. The paralyzed muscles do not become atrophied; their electrical excitability is generally unaffected; and reflex excitability is not diminished until the latter part of the disease. The rectum and the bladder are not affected. There is usually fever, but not of a high grade.

In a small proportion of cases the disease ceases its progress before grave symptoms are manifested, and recovery shortly takes place.

The disease affects men oftener than women, and the majority of cases are in persons between twenty and forty years of age. Beyond these facts nothing is known of the etiology. The most plausible supposition concerning the nature of Landry's paralysis is that the disease is of an acute infectious character, with localization in the motor tracts of the spinal cord, perhaps in some cases in peripheral nerves. This view is supported by the occurrence of prodromic constitutional symptoms, by the existence of fever, the development of more or less enlargement of the spleen, and the frequent presence of albuminuria.

Clinical experience has thus far afforded no evidence of the success of any particular method of treatment. The iodide of potassium, nux vomica, the wet pack, and the galvanic current are recommended. Alcoholics, ammonia, and stimulating tonics, together with sustaining nourishment, are indicated with a view to obviate the tendency to death.

Nervous Asthenia—Neurasthenia.

The term nervous asthenia or nervous exhaustion may be used to denote a morbid condition common in this country, especially in the larger cities. As the name signifies, debility, prostration, or exhaustion, affecting especially the nervous system, constitutes the affection. It occurs without anæmia or any notable disorder of the vital functions. It proceeds from undue functional activity of the nervous system connected with the exercise of the intellectual and emotional faculties. This morbid condition was considered as a distinct functional affection in the first edition of this work (1866). So far as I know, it had not been so considered prior to that date. Under the name neurasthenia, introduced by Beard, writers in this and in other countries have accorded to it a place among the functional disorders of the nervous system. Popularly, and to some extent among physicians, it is known by the name "nervous prostration."

Physicians are often consulted by patients who, although far from being well, have no well-defined malady. They complain of languor, lassitude, want of buoyancy, aching of the limbs, and mental depression. They are wakeful during the night and enter upon their daily pursuits with a sense of fatigue. Under the pressure of mental excitement they may be able to exert themselves with their usual energy, but when the excitement subsides they are jaded and worn out. They become apprehensive that their powers are giving way, and they often fancy the existence of some serious malady. An investigation of the different organs of the body reveals no evidence of disease. The lungs, heart, kidneys, etc. are sound. None of the affections embraced in the nosological catalogue may be discovered, yet the morbid condition is real. This brief sketch represents a class of cases with which every physician is familiar.

An inquiry into the habits and circumstances connected with the daily life in the cases now referred to will show that the mind has been for a long period unduly taxed by the cares and responsibilities of business, over-exertion in the pursuit of wealth or other objects of ambition, long-continued anxiety or inquietude; in short, the patient is suffering from wear and tear of the nervous system. The action of mental conditions is increased by other causes which tend to diminish the vigor of the body, such as irregular habits with respect to diet, the hours devoted to sleep and rest, etc. The causes which act directly through the mind are especially operative in this country, owing to the early age at which persons engage in the active pursuits of life, the great inducements to excessive exertions, and the emulation excited by the activity of others. It is not uncommon to meet with persons who have overtasked the nervous system without any rest or recreation for many consecutive years.

The condition of nervous asthenia is in itself not a small evil, causing much discomfort and unhappiness to the patient, and rendering him uncomfortable to others by inducing depression, irritability, or moroseness. That this condition favors the development of diseases to which there may be a predisposition, or co-operates with the various causes of disease, is probable. It often engenders a desire for stimulants, and thus leads to intemperance. It may eventuate in confirmed melancholia, hypochondriasis, or other forms of mental derangement.

An essential part of the TREATMENT relates to the causation. Frequently it is desirable to endeavor to procure, temporarily, complete relief from exertion and care. It is, however, extremely injudicious to advise permanent relinquishment of active occupations. After rest and recreation for a season sufficient for recuperation of the nervous system a return to accustomed pursuits is advisable, with injunctions to observe proper moderation and to comply with the laws of health as regards a fair apportionment of time to recreation and a proportionate exercise of all the faculties of the mind. It is of course desirable that causes of mental uneasiness and anxiety should be removed, but this part of the treatment often is not within the range of medical control. Habits tending to impair bodily vigor are to be corrected, such as dietetic imprudences, late hours, sexual errors or vices, the immoderate use of tobacco, etc. Hygienic measures to invigorate the body are important, such as nutritious alimentation, out-of-door life, sea-bathing, or the sponge-bath, etc. Tonic remedies, varied from time to time and long continued, are serviceable both morally and medicinally.

At the present time there is perhaps a tendency to exaggerate the pathological relations of the neurasthenic morbid condition. No anatomical system in the body tolerates functional activity better than the nervous system, provided the organism be sound and the requirements of health in other respects be observed. Patients are sometimes disposed to attribute to over-exertion of the mental faculties what is really due to other violations of the laws of health. The opinion that the symptoms denote excessive brain-work is often received with satisfaction. In some cases, considering the small amount of intellectual work accomplished and the limited capabilities of the brain, this opinion seems almost ludicrous.

Spinal Irritation.

The name spinal irritation dates from the publication of a paper by Brown of Scotland in 1828. Shortly afterward the work of Teale on neuralgia and that of Tate on hysteria appeared, these authors referring the phenomena of these diseases to irritation of the spinal cord. In 1834 the published observations of W. and D. Griffin claimed, in behalf of the supposed morbid condition thus named, a larger scope of application, and this condition soon came to be generally recognized as an individual functional affection. In an article contained in the *American Journal of the Medical Sciences* for April, 1844, I gave the results of an analysis of 58 cases which I had observed and recorded. For several years I was accustomed, in medical lectures, to include spinal irritation among the functional diseases of the nervous system. I was at length satisfied that the phenomena grouped under this name had other pathological connections, and that the claim of spinal irritation to a place in the nosology was not well grounded. Others held this opinion, and the name had nearly passed out of use; but within the past few years it has again come into vogue, and it is now recognized by many as denoting a neuropathic affection.

In so-called spinal irritation more or less pain is referred to the spine, and pressure over certain parts of the column, these parts differing in different cases in number and extent, shows greater or less tenderness. This tenderness is also shown by the application of ice, a hot sponge, and electricity. With the spinal tenderness are associated, in different cases, a variety of symptomatic phenomena, and these, in their situations, are in correspondence with the relations of the latter to the spinal cord. Thus, intercostal and cervico-occipital pains, nausea, vomiting, singultus, palpitations, and nervous cough are associated with tenderness over the cervical and dorsal vertebræ, whereas pain in the lower parts of the body, tympanites, colic, and dysuria are associated with lumbar tenderness. Patients generally are mentally depressed, the power of physical or mental endurance is diminished, sleeplessness is common, general hyperæsthesia of the surface of the body is sometimes marked, the extremities are cold, and different cases offer a great variety of subjective symptoms. The great majority of patients are women.

That there are cases which exemplify the clinical history of spinal irritation, as described formerly and at the present time, cannot be denied; but that the phenomena are caused by irritation of the spinal cord, or that they are attributable to a morbid condition (either to hyperæmia or to ischæmia) in this part of the nervous system, is by no means certain. In a large proportion of cases the phenomena are effects of general anæmia. In some instances they may be due to toxical blood-conditions, and they may be caused by reflex influences originating in local disorders of different organs.

Clinical observations show that when there is tenderness over the spine in connection with disorders of various kinds, soothing applications or mild counter-irritants over the tender portion or portions are often serviceable as palliative measures of treatment. Curative measures must be employed in other directions.

It is important not to consider the pains and tenderness referred to the spine in cases of so-called spinal irritation as symptoms of inflammation of the cord or of its meninges. The absence of fever, the sex of the patient, the existence of anæmia and often of hysteria, point to a purely functional disorder and render the exclusion of spinal meningitis and myelitis easy.

The name spinal irritation was introduced at a time when local pain and tenderness were regarded as evidence of inflammation, and, as the name denotes a functional condition, its introduction had a favorable influence on the treatment, inasmuch as at that time antiphlogistic measures—namely, bleeding, cathartics, mercury, and severe counter-irritation—were considered indispensable in treating inflammatory affections.

CHAPTER VI.

DISEASES OF THE NERVES.

Neuritis.—Multiple Neuritis.—Peripheral Paralysis.—Paralysis of the Third Cranial Nerve.—Paralysis of the Fourth Cranial Nerve.—Paralysis of the Fifth Cranial Nerve.—Paralysis of the Sixth Cranial Nerve.—Paralysis of the Motor Portion of the Seventh Cranial Nerve.—Paralysis of the Eighth Cranial Nerves.—Paralysis of the Ninth Cranial Nerve.—Local Paralysis of other than Cranial Nerves.—Paralysis of the Cervical Sympathetic Nerve.—Paralysis from Lead.—Paralysis from Arsenic, Copper, Mercury, and Phosphorus.—Neuralgia.—Trifacial Neuralgia.—Cervico-occipital Neuralgia.—Cervico-brachial Neuralgia.—Dorso-intercostal Neuralgia.—Lumbo-abdominal Neuralgia.—Crural Neuralgia.—Sciatic Neuralgia.—Dermalgia.—Myalgia.—Cephalalgia.

THE diseases to be considered in this chapter are those affecting either nervous trunks, their branches, or their terminal distributions; in other words, diseases of the peripheral nervous system. In this division are embraced neuritis, paralytic affections, and neuralgia referable to nervous trunks or branches. Paralysis from lead will be included among the peripheral paralyses.

Neuritis.

Within recent years it has been shown that neuritis is a much more common and important affection than was formerly supposed. The disease known as multiple neuritis possesses sufficient clinical peculiarities to merit consideration in a separate article.

In acute neuritis the affected nerve is swollen, reddened, and more succulent than normal. Microscopical examination shows hyperæmia, and often ecchymoses, serum, and emigrated white blood-corpuscles, frequently containing oil-globules, in the interstitial tissue of the nerve-trunk. The nerve-fibres may be little changed, or their medullary sheaths may be broken up and the fibres undergo degenerative atrophy. Acute neuritis may terminate either in resolution with regeneration of the nerve-fibres or in chronic neuritis. Suppurative neuritis is rare, and is due to some special infection.

Chronic neuritis is characterized by increase of the interstitial tissue and degeneration of the nerve-fibres. The nerve is firmer than normal and of a grayish color. It may be either increased or diminished in size.

Sometimes purely degenerative changes are found in the nerves. These are identical with those which follow section of a nerve-trunk, and they are often designated, but with doubtful propriety, as parenchymatous neuritis. (See Multiple Neuritis.)

Neuritis may affect only a small portion of a nerve in its course, or it may extend from a primary focus of inflammation either in a central or a peripheral direction. We thus distinguish *circumscribed neuritis* and *ascending or descending neuritis*. It is also held that a neuritis may extend irregularly along the course of a nerve, so as to attack certain spots and leave the intervening portions unaffected. This variety is called *neuritis disseminata*, *neuritis migrans*, or *segmental neuritis*. When the inflammation is confined chiefly to the nerve-sheath, it is called *perineuritis*.

The most important causes of neuritis are direct injuries to the nerves, the extension of inflammation from adjoining parts, exposure to cold, certain poisons (especially lead), and the poison of certain infectious diseases. Many cases of so-called reflex paralysis are referred by Leyden to an ascending or migrating neuritis extending from the region of irritation (for example, an inflamed bladder) to the spinal cord, where an acute or chronic myelitis may be induced. The changes which occur in nerves suffering from compression, as well as those caused by indirect injury, such as blows, and by severance of their continuity, although often included under the head of neuritis, are in reality degenerative.

Neuritis is attended, especially in the early stages, with severe pain, both in the region of distribution and in the course of the affected nerve. There is tenderness on pressure over the inflamed nerve, the swelling of which can occasionally be appreciated by palpation. Subsequently there may be anæsthesia, either with or without analgesia, but the anæsthesia does not usually become extreme. Neuritis seated in motor nerves or in those containing motor fibres gives rise to paralysis of motion, either partial or complete. This paralysis is followed by muscular atrophy and the reaction of degeneration. Trophic disturbances, particularly herpetic eruption and moderate œdema, occasionally occur in the region supplied by the inflamed nerve.

The onset of the disease may be acute or the symptoms may develop gradually. As the nerves are endowed with a remarkable power of regeneration, recovery may take place if the cause be removed and the nerve be not too much damaged. Probably, slight degrees of neuritis without symptoms as severe as those described are not uncommon. Inflammation of the nerves, as well as affections of the intervertebral ganglia, has been repeatedly discovered in cases of herpes zoster intercostalis.

The DIFFERENTIAL DIAGNOSIS of neuritis from neuralgia is not always easy. In fact, some cases of neuralgia are probably the result of a neuritis. The main points in the diagnosis are the paroxysmal character of the pain in neuralgia, as well as the existence of isolated points of tenderness (Valleix's points), and the absence of paralysis.

The TREATMENT has reference especially to causative circumstances. Beyond the indications relating to causation, revulsive and soothing local applications are indicated. Subcutaneous injections may be required to relieve the intensity of the pain. The employment of electricity is useful in the form of the weak galvanic current, although some recommend strong currents. In chronic neuritis cauterization has been employed with benefit in some cases.

Multiple Neuritis.

The ANATOMICAL LESIONS in multiple neuritis are in many cases degenerative, resembling those in divided nerves; but in some, particularly in acute, cases they are inflammatory. In the first class of cases the medullary sheaths of the nerve-fibres are broken up into fragments; the cells in the sheath of Schwann are enlarged and increased in number; fatty granular corpuscles accumulate in the situation of the medullary sheath as well as between the nerve-fibres; and the axis-cylinder, which may remain for a long time intact, finally disintegrates. In inflammatory cases an inflammatory exudate, consisting of serum, pus-cells, and red blood-corpuscles, also accumulates in the interstitial tissue of the nerve-trunk. Microscopical examination is requisite to determine the changes, as often no distinct alteration is visible to the naked eye. If the case terminate in recovery, the nerve-fibres are regenerated in the same manner as after section of nerve-trunks. In chronic cases a new growth of connective tissue appears in the nerve-trunks.

The affection involves many, sometimes most, of the spinal nerves. The cranial nerves may also be affected, but less commonly than are the spinal nerves. The lesions are not distributed uniformly over the nerves, but are more intense in some places than in others. Usually, a nerve-trunk is more or less affected throughout its entire length. If the spinal nerve-roots be also involved, there are, usually, slight atrophic or degenerative changes in the ganglion-cells of the anterior horns of the spinal cord. From the symptoms it may be inferred that the motor nerve-fibres are by preference the seat of the disease.

The CAUSATION of primary multiple neuritis is not known, but the symptoms support the usually-accepted view that the disease is due to some special infection. Certain cases of paralysis after acute infectious diseases, particularly after diphtheria, are referable to a multiple neuritis.[1] The affection is most common in early adult life.

SYMPTOMS.—Multiple neuritis may pursue an acute or a chronic course. The acute cases begin generally with fever and constitutional symptoms, often of considerable severity. Swelling of the spleen and albuminuria are sometimes present. These symptoms indicate the infectious nature of the disease. At the onset there is pain, usually severe, and felt generally in the loins and in the extremities. Moderate swelling of the joints has also been observed. Paralysis soon makes its appearance, as a rule first in the form of paresis of the lower extremities. The paralysis increases in intensity and in extent, involving the upper extremities, and, it may be, the muscles of the trunk, especially the muscles of respiration. Reflexes, particularly the tendon reflexes, are diminished. As may be inferred from what has been said on p. 733, the paralyzed muscles rapidly atrophy, and the nerves and muscles soon present the reaction of degeneration. In the mean time the pain has usually lessened, although some remains, especially upon pressure over the affected nerves and muscles. Abnormal sensations, called paræsthesiæ, such as numbness and formication, are often experienced. There usually is some loss of sensation, but marked anæsthesia is not generally produced. On the other hand, in acute cases there often is pronounced hyperæsthesia. The functions of the bladder and of the rectum remain intact. Herpetic eruptions, œdema, and less frequently other trophic disturbances may occur. There is often observed great frequency of the pulse, probably due to involvement of the vagus.

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[1] According to Scheube, the disease which is known as beriberi or kak-ke, and which prevails in Japan and India, is a multiple neuritis.

Acute multiple neuritis may terminate within a few days or two weeks in death. The most frequent cause of death is paralysis of the respiratory muscles. In some cases recovery takes place, which is usually gradual, as considerable time is required for the regeneration of the nerve-fibres. In other cases the acute symptoms subside and more or less paralysis remains permanently, or the disease may pursue a chronic course.

Multiple neuritis may begin insidiously, and without acute symptoms lead to atrophic paralysis. In these chronic cases recovery is also possible, or the disease may come to a standstill with more or less permanent paralysis.

The main difficulty in DIAGNOSIS is to distinguish multiple neuritis from anterior poliomyelitis, with which it has doubtless often been confounded. The main points in this diagnosis relate to the presence of pain and other sensory disturbances in neuritis which are absent in uncomplicated anterior poliomyelitis. The distinction between acute ascending paralysis and multiple neuritis may also occasion some difficulty, but careful attention to the symptoms which have been described will generally lead to a correct diagnosis.

The PROGNOSIS, as is evident from what has been said, is grave, but not extremely so. In distinction from the spinal cord, the peripheral nerves are capable of extensive regeneration, so that recovery may take place after the paralysis has become complete and has lasted for a number of weeks or even months.

In view of the probably infectious nature of the disease, it is recommended at the onset of acute cases to administer large doses of salicylic acid. The intensity of the pain will usually require the use of opium in some form. Counter-irritation in the form of stimulating liniments may be applied over the affected parts. The systematic and long-continued use of electricity, beginning with not too strong galvanic currents, is important. Cutaneous faradization is sometimes employed as a means of counter-irritation. In chronic cases especially massage may be beneficially employed.

Peripheral Paralysis.

In preceding articles various causes of peripheral paralysis have already been mentioned. These causes will here be enumerated. Peripheral paralysis may be caused by inflammation, degeneration, compression, and injury of a nerve. It may be caused by toxic substances, of which the most important is lead, and less common causes are copper, arsenic, certain vegetable alkaloids, and some other substances. Peripheral paralysis is common after diphtheria, and is less frequent after other acute infectious diseases, such as smallpox, typhoid fever, measles, etc. It may be the result of syphilis, but rarely of other chronic infectious diseases. An important cause of peripheral paralysis, particularly of facial paralysis, is exposure to cold. No satisfactory explanation of reflex peripheral paralysis has been given. Finally, peripheral paralysis may occur without known cause.

Peripheral paralysis may be the result of some demonstrable change in the nerve, such as inflammation, degeneration, or tumor, or it may be purely functional. As our knowledge extends, the instances of purely functional paralysis become fewer.

The following are the main points to be considered in the DIAGNOSIS of peripheral paralysis: The paralysis affects a muscle or a group of muscles supplied by a nerve-trunk or a nerve-branch. In certain forms of spinal paralysis (anterior poliomyelitis) the paralysis may be confined to a group of muscles, but this is a group of muscles functionally associated, and not necessarily supplied by a single nerve-trunk. In severe peripheral paralysis the affected muscles undergo rapid atrophy, and the nerves and muscles take on the reaction of degeneration according to the principles already laid down (p. 733). The reflexes are generally diminished in peripheral paralysis. Cases vary as to the existence of sensory disturbances, but a certain degree of these is not uncommon, and marked anæsthesia may accompany the motor paralysis.

The TREATMENT in cases of peripheral paralysis has relation to local causes whenever these can be ascertained and reached. Their continued operation is, if possible, to be prevented. Lacerations and divisions of nerves are to receive proper surgical treatment with reference to the restoration and reunion of separated or injured parts. Tumors pressing upon nerves are to be removed if practicable, abscesses opened, and inflammation treated according to the indications. It is obvious that the probability of improvement and recovery must depend on the nature of the causes and the extent to which they can be successfully treated. Cases in which the paralysis is due to cold or to transient external pressure, and those in which there is no discoverable causation or appreciable lesion, generally tend to a favorable termination; and this may be hastened by therapeutic measures.

The chief therapeutic agent is electricity. The employment of this agent is to be governed by rules the consideration of which is not embraced within the scope of this work. Local frictions, the use of stimulating liniments, the cold and warm douche, the topical application of strychnia, and voluntary exercise of the paralyzed muscles whenever the conduction of volition is not wholly obstructed, are measures of utility in peripheral as in central paralysis. In cases of peripheral as in cases of central paralysis the probability or the possibility of a syphilitic connection is to be considered with reference to the treatment. Antisyphilitic medication should be employed whenever there is ground for a suspicion of this connection.

Paralysis of the nerves of special sense—namely, the olfactory, the optic, or the auditory nerve—is frequently dependent on abnormal conditions relating to the accessory organs of smell, sight, and hearing. This remark applies more especially to the eye and ear. Affections of the eye and ear involving loss or impairment of sight and hearing belong in the province of surgery, and treatises are devoted specially to them. It is important, however, for the physician to take cognizance of paralysis affecting these senses, as denoting intracranial disease. In most cases paralysis of the nerves of special sense, if due to intracranial disease, is associated with paralysis of other parts. Paralyses of the other cranial nerves severally claim brief consideration. It is more correct to say paralysis of the parts to which the branches of a nerve are distributed than to apply the term to the nerve, but the latter is a convenient mode of expression and is sanctioned by usage.

Paralysis of the Third Cranial Nerve.

Paralysis of this nerve, the motor oculi communis, gives rise to dropping of the upper eyelid or ptosis, diverging strabismus, protrusion of the eyeball, and dilatation of the pupil. These effects correspond with the distribution of the several branches of the third pair of nerves. The internus rectus muscle being paralyzed, the eyeball is turned outward by the externus rectus and the superior oblique muscle; and dilatation of the pupil is caused by paralysis of the circular fibres of the iris. The effects are so obvious and characteristic as at once to be referable to paralysis of this nerve. Paralysis of this nerve may be produced by an injury in the neighborhood of the eye, by the action of cold, and by over-exertion in occupations which require especially the use of the eyes. If produced by cold or over-use, the paralysis is functional and will not be permanent. Double vision and imperfect accommodation accompany paralysis of this nerve. The paralysis is rarely limited to this nerve if it proceed from intracranial lesions, but it is associated with paralysis affecting other nerves or with other marked effects of cerebral disease. It is rare in cases of hemiplegia. Occasionally it is the first event pointing to grave cerebral lesions. It may be a forerunner of hemiplegia, convulsions, or coma. In a case under my observation paralysis affecting this nerve on the right side occurred without other central symptoms three weeks before an attack of apoplexy and left hemiplegia, the latter proving fatal two hours after the attack. If ptosis exist without strabismus and dilatation of the pupil, it may be inferred that the paralysis is not central but peripheral. The paralysis is then limited to the upper branch of the nerve. So also, if the inferior branch be alone affected, causing external strabismus and dilatation of the pupil without ptosis, the paralysis is probably peripheral.

Paralysis of the Fourth Cranial Nerve.

Paralysis of this nerve, the patheticus, produces effects less marked than those of paralysis of the third nerve. Movement of the eye at the same time downward and inward is impaired. Double vision exists of objects occupying the lower part of the field of vision, and is therefore manifested especially in looking downward, as in going down stairs. Paralysis of this nerve is an isolated affection in some cases of syphilis.

Paralysis of the Fifth Cranial Nerve.

The effects of complete paralysis of this nerve are shown by its division within the skull in inferior animals. Loss of sensibility (anæsthesia and analgesia) on one side of the face, of the mucous membrane within the mouth, the conjunctival membrane on that side, and the anterior portion of the tongue, and paralysis of the masticatory muscles on the same side, immediately follow. The intracranial division of this nerve is followed within a short time by ulceration of the cornea, and sometimes by suppurative inflammation of the whole eye. Complete paralysis of the trigeminus from disease is rare. The motor portion may be affected either separately or in conjunction with the sensory divisions. Impairment or loss of the power of mastication denotes paralysis of the motor or third division. Bilateral paralysis of the motor portion occurs rarely in cases of bulbar paralysis. The different sensory divisions may be affected separately—namely, the ophthalmic, the superior and the inferior maxillary. The loss or impairment of sensibility is then confined to the portions of skin and mucous membrane supplied by the branches of the paralyzed division.

Ulceration of the cornea, absence of reflex conduction, redness and swelling of the face and of the gums, constitute evidence that the paralysis is peripheral, the local cause being perhaps intracranial, but extracerebral. When the paralysis is central, it is accompanied by symptoms denoting cerebral disease, and other cranial nerves are generally involved.

Pain is sometimes referred to parts supplied by the branches of the nerve, notwithstanding anæsthesia and analgesia in these parts are complete (anæsthesia dolorosa). The cause of the pain is situated in the portion of the nerve posterior to the seat of the affection which causes the paralysis. The explanation is the same as when, after an amputation, pain is felt to be in the amputated limb ("eccentric projection of perception").

Bilateral paralysis of this nerve is sometimes observed. In a case reported by Althaus the common sensibility of the face and scalp was lost, together with the sense of temperature and of tact; the mucous membrane of the eyes, nose, and mouth was completely anæsthetic; the sense of taste was preserved, although the exercise of it seemed less prompt than in health; and the muscles of mastication were completely paralyzed. The tongue presented a frightful spectacle, having been bitten and lacerated in every direction during the act of taking food. The patient experienced no pain when a tooth was drawn. The lips were covered with froth. The sense of smell and of sight were not affected, except that the latter was obstructed by thick leucoma of both corneæ. The patient suffered much from photophobia. There was an abundant secretion of mucus from the Schneiderian membrane. Hearing was intact, but the patient was greatly annoyed by tinnitus aurium. The paralysis was produced by long exposure of the face to a keen wind, the morbid condition being supposed to be inflammation of the neurilemma at the point where the nerve emerges from between the transverse fibres of the pons varolii at the base of the brain. The brain was unaffected, and the paralysis was limited strictly to the distributions of the fifth pair. At the end of three months, under treatment consisting of the systematic application of the continuous galvanic current to the several branches of the affected nerve, the recovery was nearly complete.[1] An important indication for treatment is to protect the anæsthetic parts, particularly the cornea, from external injury.

Paralysis of the Sixth Cranial Nerve.

Paralysis of this nerve, the motor externus or abducens, leads to converging strabismus, the eyeball being turned inward by the action of the internal rectus or adductor muscle, from the loss of the antagonism of the external rectus or abductor muscle. The patient is unable to turn the eyeball outward, whereas in ordinary cases of strabismus this can be done if the other eye be closed. Paralysis of this nerve sometimes occurs in cases of cerebral meningitis and in connection with lesions giving rise to hemiplegia. This nerve, however, escapes in the majority of the cases in which paralysis of other parts occurs from intracranial affections. When the nerve is paralyzed, paralysis of the third cranial nerve is frequently associated. The effect of paralysis of both these nerves is immobility of the eyeball as regards lateral movements, which can be turned neither inward nor outward. Double vision is likely to exist when the paralysis is limited to the sixth nerve. Functional paralysis of this nerve is not uncommon. It may be caused by neuritis, and it is sometimes attributable to syphilis.

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[1] *Transactions of the Royal Medical and Chirurgical Society of London*, 1869, vol. lii.

Paralysis of the Motor Portion of the Seventh Cranial Nerve—Bell's Paralysis—Mimetic Paralysis.

Paralysis of this nerve, the portio dura, or the motor facial nerve *par excellence*, is much less infrequent than paralysis of the sensitive portion of the fifth nerve. Complete paralysis of this nerve produces characteristic appearances, by means of which the affection is at once recognized. The expression of the physiognomy peculiar to the person is abolished on the affected side. The naso-labial fold is flattened on this side. In speaking or smiling the mouth and sometimes the extremity of the nose are drawn to the opposite side. This appearance is so striking and distinctive that the existence of the paralysis is immediately apparent. The power over the orbicularis palpebrarum being lost, the patient is unable to wink or to close the eye. When the attempt to close the eye is made, the eyeball is turned upward, so that the cornea is concealed by the upper lid. The upper lid is abnormally elevated and the lower lid is depressed, so that the eye appears to project more than in health. Owing to the loss of power over the orbicular muscle, the tears are not diffused over the conjunctiva as in health, and hence results epiphora. From the constant exposure of the conjunctiva in sleep as well as in waking hours it becomes inflamed, and opacity of the cornea may be a result of the conjunctivitis. The eyebrow is lowered, and the patient is unable to frown or to wrinkle the forehead on the affected side, from loss of power over the corrugator supercilii and the anterior portion of the occipito-frontalis muscle. The ala nasi ceases to dilate, but may contract in inspiration; and dilatation, if it occur, is in expiration. Loss of power over the orbicularis oris on the affected side renders the patient unable to whistle; expuition is imperfect, the saliva dribbles from the angle of the mouth; and the pronunciation of certain letters—namely, *o*, *b*, *p*—is difficult. Loss of power over the buccinator renders the cheek flaccid; it is liable to be wounded in mastication, and the patient is annoyed by the accumulation, between the cheek and gums, of food which he is obliged to remove with the finger. The cheek and lips of the affected side are sometimes puffed outward by the expired breath, as in the act of expelling tobacco-smoke from the mouth. These obvious appearances are readily understood by reference to the anatomical distribution of the branches of this nerve. They are, of course, less marked in proportion as the paralysis is incomplete. The velum pendulum palati on the paralyzed side is sometimes abnormally flaccid, and the uvula is deflected to the opposite side. The auditory sense on the paralyzed side is more acute than in health. After long continuance of the paralysis contracture of the paralyzed muscles may develop, causing deviation of the features to the affected side.

Paralysis of this nerve is not very infrequently an isolated affection, existing independently of cerebral disease. The seat of the affection may be intracranial, but extracerebral; that is, the seat, although within the skull, is between the central connection of the nerve and its entrance into the meatus auditorius. It may be due to the pressure of a tumor on the nerve after it emerges from the stylo-mastoid foramen. The nerve may be affected within the petrous portion of the temporal bone from fracture, or in connection with caries of this bone resulting from median or internal otitis. Facial paralysis occurs not infrequently from the action of cold upon the face. I have met with instances of its occurrence in connection with anæmia. It is sometimes produced in new-born children by the employment of the forceps in labor. Facial paralysis may proceed from the various intracranial affections which have been considered. In these cases it is frequently associated with paralysis affecting other nerves. It is sometimes a forerunner of a grave cerebral affection.

If central, the paralysis is generally preceded or accompanied by symptoms denoting a cerebral affection. If the nerve be affected in a situation involving the chorda tympani, the sense of taste on the lateral half of the anterior portion of the tongue corresponding to the paralyzed side is diminished, as may be shown by comparing the sensations produced by placing successively on the two sides of the anterior extremity of the tongue some bitter substance in powder, such as aloes. The salivary secretion is diminished. These effects are due to the involvement of the chorda tympani in the paralysis. The taste and the salivary secretion are not affected if the cause producing the paralysis act upon the nerve anterior to the situation at which the chorda tympani is given off.[1] Flaccidity of the palatine arch on the affected side and deflection of the uvula to the sound side denote that the cause of the paralysis acts upon the nerve either within the aqueductus Fallopii or before it enters this canal, the branches supplying the levator palati and the azygos uvulæ muscles being here given off. If the paralysis be central, the electro-muscular contractility is preserved; and if it be peripheral, the reaction is rapidly lost. The paralysis is peripheral when the nerve is affected at any point beyond its origin. Paralysis of the sixth nerve accompanying paralysis of the facial nerve denotes a central lesion, these nerves arising near each other in the floor of the fourth ventricle. The absence of reflex movements is evidence that the paralysis is peripheral. If winking be not produced by touching the eyeball, the sensibility of the latter being preserved, the nerve is affected after leaving its central connection; and on the other hand, if the eye be closed involuntarily, following an impression made on the conjunctiva when the voluntary power is lost, the paralysis is central. Abnormal acuteness of hearing is evidence that the nerve is affected behind the point at which the twig going to the stapedius muscle is given off. The paralysis is much oftener complete when it is peripheral than when it is central. Accompanying hemiplegia, the paralysis is generally incomplete and the orbicular muscle of the eye is but little affected. It is rarely wholly unaffected, as stated by Todd. Evident diseases of the ear or of the parotid gland often account for the paralysis as well as its peripheral character, and they point to the seat of the affection.

In cases of facial paralysis dependent on neuritis caused by cold, or when it is functional, recovery may be expected after the lapse of several weeks. If the paralysis continue without improvement for two or three months, it is probably not functional, but if not dependent on central lesions it proceeds from either disease of the nerve or something pressing upon it and occasioning obstruction. Under these circumstances it will be likely to remain a permanent affection. The prognosis is also grave in proportion as the electro-muscular contractility is diminished.

Bilateral paralysis (diplegia facialis) has been repeatedly observed, but examples are extremely rare.[2] It may result from injury or disease of the two nerves or from intracranial lesions. The expression is then abolished on both sides of the face. The face remains immovable, the only changes which are observed relating to the circulation. It is as if the face were covered by a mask. Relaxation of the soft palate may coexist, rendering the voice unnatural. Moreover, articulation is difficult from the immobility of the lips. Mastication is difficult from the same cause. The saliva dribbles from the mouth. The eyes are constantly open and staring (lagophthalmus). Aside from the great inconvenience which bilateral facial paralysis occasions, the prognosis is serious or otherwise according to the situation and nature of the morbid conditions on which the paralysis is dependent. In a case which came under my observation with the double facial paralysis was associated incomplete general paralysis, and the patient died by apnœa from inability to expel, by coughing, matter in the air-passages. Paralysis affecting the alæ nasi may lead to an amount of depression with inspiration sufficient to occasion notable obstruction. The cartilaginous portions of the nostrils on both sides are sucked in with the inspired breath to such an extent as to constitute a serious obstacle to respiration.

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[1] The fibres of the chorda tympani nerve are probably derived ultimately from the glosso-pharyngeal nerve, although some assign them to the trigeminus.

[2] For a report of 28 cases of double facial paralysis, see article by Dr. Pierreson in *Archives générales de Médecine*, Août et Septembre, 1867.

Paralysis of the Eighth Cranial Nerves.

The paralysis affecting the pharynx, which occurs as a sequel of diphtheria in certain cases, and which is sometimes limited to this situation, is to be considered as a paralysis of that division of the eighth pair called the glosso-pharyngeal. This local paralysis will be referred to in treating of diphtheria.

Paralysis of this nerve, together with paralysis of the par vagum, occurs in various cerebral affections which produce deep coma, giving rise to impairment and loss of power of deglutition and diminished frequency of the respirations. This paralysis precedes a fatal termination. It may occur from a small extravasation of blood into or near the medulla oblongata. I have met with an example, the apoplectic attack being characterized by notable disturbance of respiration and inability to swallow, without complete loss of consciousness. Paralysis of these nerves occurs also in bulbar paralysis. The vagus is an inhibitory nerve of the heart. Its irritation causes slowing of the heart-beats, and if it be intense their cessation in diastole. Its paralysis causes increased rapidity of the cardiac pulsations.

Paralysis of the spinal accessory nerve, giving rise to aphonia, has been already referred to in treating of the affections of the larynx. Incomplete aphonia may be a result of paralysis of this nerve from the pressure of an aneurismal or other tumor on the recurrent laryngeal nerve, and it is sometimes produced by the surgeon in operations on the neck.

Paralysis of the Ninth Cranial Nerve.

Paralysis of this nerve, the hypoglossus, on one side, involves loss of power over the genio-hyo-glossus and other muscles of the tongue on that side. The effect is a deviation of the tongue from a right line when protruded, the apex pointing to the side of the paralysis. The direction in which the apex points is readily understood when the action of the muscles which protrude the tongue is considered. These muscles extend from the chin to the base of the tongue; and when from paralysis the protrusion is effected by the muscle on one side, the base is drawn toward that side, and as a result of the base moving in one direction the apex turns in the opposite direction, the latter being toward the paralyzed side. Paralysis of this nerve rarely exists as an isolated affection, and it is generally symptomatic of intracranial disease. It exists frequently in cases of hemiplegia.

In determining the existence of this paralysis it is to be borne in mind that the tongue may deviate from a right line in consequence of absence of teeth on the side to which the apex is turned or of irregular inward projection of teeth on the other side. Dropping of the upper lip on one side produces an appearance as if the tongue deviated to the opposite side; and to obviate liability to error on this score the upper lip should be raised with the finger while the tongue is protruded.

Paralysis of the hypoglossal nerve on both sides is extremely rare, but cases are sometimes observed. In complete paralysis of this nerve on both sides there is inability to protrude the tongue, and the power of speech is lost from inability to articulate. Incomplete paralysis renders protrusion of the tongue and articulation more or less imperfect. Paralysis of this nerve on both sides enters into the paralytic affection which has been considered under the head of Bulbar Paralysis.

Local Paralysis of Other than Cranial Nerves.

Paralysis may be produced by injuries or diseases affecting nervous trunks in any part of the body. The loss or impairment of motion or sensation, or both, extends over the parts supplied with the nervous filaments given off by the nerve below the situation of the injury or disease. Traumatic local paralysis follows division or wounding of nerves and contusions. If a contusion give rise to paralysis by simple shock or stunning the nerve, recovery may be expected; but paralysis due to palpable injury of the structure of a nerve is likely to be permanent, although reunion, with a restoration of function, may take place after the division of a nerve of considerable size. Diseases which give rise to local paralysis by affecting nerves are tumors or morbid growths situated within and without the neurilemma, and inflammation of the neurilemma or of the nerve-substance. The latter generally occurs either by extension of inflammation seated primarily in the structures adjacent to the nerve affected or by primary inflammation of the nerve. Paralysis due to the pressure of a tumor may be recovered from if the pressure be removed before extensive disorganization or atrophy has resulted.

Pressure on a nervous trunk, continued steadily for several hours, may lead to temporary or even permanent paralysis. Paralysis of an upper extremity has repeatedly occurred from remaining in profound sleep or in a state of intoxication with the arm hanging over the back of a chair, the paralysis being due to pressure on the brachial plexus. I have met with many examples of paralysis of the forearm attributable to lying, while intoxicated, with the weight of the body upon the arm. Paralysis of a single muscle or a small group of muscles, when not traumatic, is significant of syphilis as the cause.[1] Among the more common examples of peripheral paralyses of spinal nerves from pressure or injury may be mentioned paralysis of the serratus magnus, of the deltoid, of the muscles supplied by the musculo-spinal nerve, by the ulnar nerve, or by the median nerve, paralysis of the diaphragm, and paralysis of muscles supplied by the anterior crural or by the sciatic nerve.

The following interesting case of local sensory paralysis from injury has fallen under my observation. The injury was received by falling from a considerable height upon the deck of a vessel, and striking upon the nates in a sitting posture. Permanent paralysis of the sensibility of the rectum and anus followed the contusion. Excrescences which at one time existed around the anus were removed without any sensation of pain. The patient has no consciousness of the accumulation of feces in the rectum, and never experiences the desire for defecation. Hence the bowels are habitually constipated. The contraction of the sphincter, due to reflex influence, is intact. Involuntary evacuations occur only when diarrhœa exists and when voluntary power over the muscles is requisite to prevent their occurrence. There is no paraplegia, no difficulty in evacuating the bladder, and no sexual deficiency. The accident occurred more than forty years ago, and in the mean time the person has had good health.

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[1] Vide article by Dr. Keyes in the *New York Med. Journal*, Nov., 1870.

Paralysis of the Cervical Sympathetic Nerve.

The experiment of dividing the sympathetic in the neck, repeated by Bernard in 1851, and its effects studied more fully than by previous observers, is now made familiar to medical students by those teachers in physiology who employ vivisections. The reader is therefore presumed to know that the immediate results of this experiment are congestion of the head on the side corresponding to the section, rise of temperature due to increased rapidity of circulation, and contraction of the pupil on the same side. These effects on the circulation are attributable to paralysis of vaso-motor nervous filaments. Paralysis of this nerve occurs from pressure by a tumor, and it has followed surgical operations and other wounds. Contraction of the pupil is one of the symptoms of aortic aneurism when the tumor is so situated as to press upon this nerve. Dr. S. Weir Mitchell has reported a case in which paralysis of the right sympathetic was caused by a wound from a musket-ball. The right pupil was contracted, and the eye on that side was myopic. On exercise the right side of the face became flushed, while the left side was pale.[1] Dr. William Ogle has reported a case in which, from the local appearance and the phenomena, it was inferred that the right sympathetic had been destroyed by an abscess in the neck. In this case the right pupil was contracted and the eyeball was somewhat retracted. The right side of the head was redder and warmer than the left, especially after exercise. A singular feature in this case was that the patient sweated on the left side of the face only. This was observed repeatedly after active exercise and during exposure to great heat in a Turkish bath. Although this condition had existed for two years, no inflammatory process had followed.[2] Functional paralysis of the sympathetic system in different situations has been supposed to be involved in certain cases of paralysis affecting the upper and the lower limbs, and to enter largely into the pathology of nervous affections accompanied by disturbances of circulation, secretion, and nutrition. These views are founded chiefly on the relations of the sympathetic system to the vaso-motor nerves, as shown by experimental observations on inferior animals.

Paralysis from Lead.

The accumulation of lead within the system may give rise to partial and even to general paralysis, but as a rule the paralysis due to this cause is local. In the great majority of cases the paralysis is limited to the extensor muscles of the upper extremities. The muscles supplied by the musculo-spiral nerve are those chiefly affected. The triceps and the supinators escape. The usual form of lead paralysis is that commonly known as *wrist-drop*. This name expresses the peculiar appearance of the upper extremities. The patient is unable to raise the hand to a line with the forearm. Frequently the first series of the phalangeal bones of the hand cannot be brought on a line with the back of the hand, but the second and third series can generally be extended, as they are acted upon by the interossei muscles. The dropping of the hands is characteristic of this affection. Whenever the peculiar appearance referred to is presented, the physician is justified in assuming that the paralysis is from lead. In lead paralysis of the lower extremities the peroneal muscles are usually first affected, and then the extensors on the leg, the anterior tibial muscle remaining intact. The paralysis is shown by inability to raise the feet with the heels resting on the floor or to raise the toes. The feet drop when the patient walks, and he therefore steps high to avoid tripping. Paralysis of the muscles of the lower limbs is not common. In a case under my observation the extensors of both the feet and hands are paralyzed, and the patient is affected with dropping at the ankle as well as at the wrist.

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[1] Vide *Injuries of Nerves*, by S. Weir Mitchell, M.D., Philadelphia, 1872.

[2] Vide *Trans. Royal Med. and Chirurg. Society of London*, 1869, vol. lii.

The appearance is diagnostic, but other evidence of lead-poisoning may almost always be obtained. As a rule, paralysis succeeds other characteristic effects of lead, especially the affection known as lead colic. (Vide p. 524.) The blue line on the gums referred to in treating of lead colic is always to be sought for. Generally, the occupation of the patient suggests the source of the paralysis, but in some cases the mode in which the lead has been introduced into the system is ascertained only after careful investigation. An examination of the urine for the presence of lead should not be omitted.

Lead paralysis belongs among the symmetrical or bilateral affections. It is very rarely limited to one side, and corresponding muscles on the two sides are usually affected, but in general differently as regards the degree of paralysis. The paralysis may be complete or incomplete, and cases differ much in respect to its degree. The power over the flexor muscles is frequently more or less impaired, but in some cases with almost complete loss of power over the extensors the flexors are but little if at all affected. This fact was illustrated in a striking manner in a case under my observation at Bellevue Hospital. Wishing to group several cases of paralysis for a clinical lecture in the hospital amphitheatre, which is situated at the top of the building, I desired to have a patient affected with complete paraplegia carried up from a ward on the ground-floor. Four flights of stairs were to be mounted. One of the patients, who was to make part of the group, was affected with lead paralysis, the extensors of the hand being nearly powerless. He was a man of great strength, and he volunteered to carry his fellow-patient to the amphitheatre on his back. It was necessary to assist him in obtaining a firm grasp of the patient, but when this was obtained he performed the feat with ease, and after the lecture brought the patient down to the ward.

Lead paralysis belongs to the group of atrophic paralyses. The peripheral nerves and muscles rapidly assume the reaction of degeneration, but complete loss of electrical reaction is not common. These facts make it probable that lead paralysis is due either to an affection of the peripheral nerves or to an affection of the ganglion-cells of the anterior horns of the spinal cord (poliomyelitis anterior). The weight of evidence seems to favor the view that the paralysis is of peripheral origin, but in some instances changes have been found in the ganglion-cells of the anterior horn. Further investigations upon this point are requisite. It is an interesting fact that alterations in the electrical reactions have been found before paralysis appeared, and also in nerves and muscles which did not become paralyzed. Voluntary power over the muscles is restored before normal response to the faradic current appears.

It is to be borne in mind that muscles other than those involved in the common form of lead paralysis, or the wrist-drop, may be paralyzed by this cause. The evidence of lead-poisoning is therefore to be sought after in cases of paralysis in which other adequate causes are not evident. Amaurosis is one of the occasional effects of lead-poisoning. Exceptionally, anæsthesia and analgesia of the surface have also been observed, affecting limited portions of the integument of the body and extremities. The muscles of the larynx concerned in phonation have been known to become affected, leading to aphonia, as well as those of the lips and tongue, rendering articulation difficult.

The DURATION of lead paralysis is very variable. It is sometimes of short duration, but much oftener continues for a considerable period, and improvement, if it take place, is very gradual. If under proper treatment no improvement take place for several weeks, the prognosis is unfavorable as regards recovery, and the paralysis is likely to be permanent. If the paralysis continue long enough to lead to notable atrophy of the affected muscles, and if they do not react to either the faradic or the galvanic current, the condition as regards recovery or improvement is hopeless. The proportion of cases in which this result takes place is not small.

The TREATMENT of paralysis from lead involves, in addition to the measures applicable in general to paralysis, remedies having reference to the elimination of lead from the system. The iodide of potassium is supposed to form with the lead a soluble compound, and in this way to promote its elimination. It must be admitted that the clinical proof of this effect of this remedy is not very conclusive. Warm baths and the hot-air bath are useful. It is doubtful whether sulphur baths, which have been much recommended, possess any advantage derived from the action of sulphur on the body. As regards the treatment addressed to the paralysis, the clinical evidence of the benefit of strychnia, given internally and applied endermically, is conclusive. A most important measure of treatment is the employment of electricity, first in the form of the galvanic current, and then of the faradic current when this begins to produce response. Prophylactic measures are important for those whose occupation involves contact with lead.

Paralysis from Arsenic, Copper, Mercury, and Phosphorus.

Paralysis is an occasional effect of either acute or chronic arsenical poisoning; oftener of the former than of the latter. The paralysis affects most frequently the muscles of the lower extremities (paraplegia). General paralysis is sometimes produced by this metal. There are usually, in addition, sensory disturbances. Recovery takes place as a rule, with some exceptions.

Paralysis from copper is preceded by the symptoms of gastro-duodenitis. Instances of paralysis from this cause are extremely rare.

Mercury sometimes causes paralysis which is always preceded by mercurial tremor. The effects of mercury upon the mouth, conjoined with the occurrence of tremor, point to the cause of the paralysis. The iodide of potassium is supposed to promote the elimination of mercury, as well as of lead, from the system, and is therefore indicated in cases of mercurial poisoning.

Paralysis has been observed after acute and chronic poisoning with phosphorus.

The TREATMENT having direct reference to the nerves and muscles involved in the paralytic affections caused by these metals is the same as in cases of paralysis from other causes.

Neuralgia.

The term neuralgia is used to distinguish functional affections of which the chief characteristic is pain. In a purely neuralgic disease there is neither inflammation nor any appreciable lesion in the painful part. There is a condition of morbid sensibility, and this is all that can be said of its essential pathological character. Visceral neuralgias, as they are called—that is, neuralgias seated in organs belonging to the respiratory, circulatory, digestive, and urinary systems—are considered in the several sections devoted to the diseases of these organs. The neuralgic affections to be here considered relate to nervous trunks or their branches not connected with the organs composing the foregoing physiological systems.

As regards symptomatology, diagnosis, causation, prognosis, and the general principles of treatment, whatever is true of any one of these affections is mainly true of all of them. In order to avoid repetitions, therefore, I shall first consider these neuralgic affections collectively, or neuralgia in general, under the several aspects just named, and shall afterward notice briefly the more important of the affections individually.

Neuralgia may occur abruptly, but in the great majority of cases there are premonitions. The premonitions are—a feeling of weight, a sense of heat, or some uncomfortable sensation in the seat of the affection. The neuralgic pain is either chiefly or exclusively in paroxysms, or there may be more or less continued pain, with exacerbations in which the pain is much increased. The continued pain may be dull or contusive, but during the paroxysms or exacerbations the pain is described as darting, tearing, or lancinating, and it is often excruciating. The paroxysms or exacerbations are of variable duration, lasting in some cases for a few seconds only, and in other cases for several hours or even days. The intermissions or remissions are not less variable in different cases; and their duration may be only a few seconds or moments, and they may extend to days. The lancinating pain shoots in the direction of a nervous trunk and its branches; and patients sometimes delineate the tract and distribution of the nerve affected in defining the directions in which the pain extends. In the great majority of cases neuralgia is unilateral.

Tenderness on pressure, limited to circumscribed points, is an important diagnostic feature of neuralgia. Valleix was the first to show that as regards their relations to the nerves, the tender points are situated as follows: 1st, where nervous trunks or branches emerge from the skull or spinal canal; 2d, over branches which penetrate muscles on their way to the integument; 3d, at the termination of branches which are lost on the surface; and 4th, in places over trunks near the surface. The situation of the tender points in intercostal neuralgia has been stated in treating of that affection. (Vide p. 183.) The diagnostic evidence to be derived from this source in other neuralgic affections will be stated when these are severally noticed. Pain may not be produced by pressure made with the open palm; and, indeed, firm pressure thus made often relieves the pain. The tenderness is shown by pressure made with the ends of the fingers or by percussion. The pain thus produced is often acute, causing the patient to cry out or to try to escape from the exploration. If, however, the pressure be continued, the tenderness is often found to diminish or temporarily to disappear. The tender points are frequently quite limited, the end of the finger being often sufficient to cover them. As a rule, the pain produced by pressure is acute in proportion to the acuteness of the spontaneous pain. The tenderness is most marked during paroxysms or exacerbations of pain. In the intervals between paroxysms, the patient being entirely free from pain, the tenderness on pressure may disappear. Exclusive of the tender points, there may be either hyperæsthesia or anæsthesia of the surface, and the latter may follow the former in the same case.

Paroxysms or exacerbations of pain are excited or increased by sudden or violent movements of parts in which the affected nerves are situated. A concussion of the whole body, as in coughing or sneezing, excites or increases the pain. Cold or hot applications to the surface over the seat of pain in some cases are insupportable.

Fever or any notable constitutional disturbance does not belong to the clinical history of neuralgia. The functions of parts to which the affected nerves are distributed may not be notably disturbed. A neuralgic attack, however, often induces hyperæmia in the site of the pain, as shown by temporary redness of the conjunctiva in cases of neuralgia affecting the ophthalmic division of the fifth pair. Involuntary contractions of muscles and cramps sometimes occur. An increased secretion of tears, saliva, and mucus may accompany paroxysms of neuralgia affecting the several divisions of the fifth pair; and glandular organs within the body are probably in like manner excited in the visceral neuralgias.

Neuralgic affections have no fixed duration. They may end, either spontaneously or under the influence of treatment, in a few hours or days, and they may continue for years or during life, in spite of all methods of treatment. The prognosis is always favorable as regards a fatal termination. Persons who suffer attacks at variable intervals for a lifetime are at length carried off by other maladies not developed in consequence of the continuance of the neuralgia.

Neuralgic pain may proceed from a palpable lesion, such as a tumor either involving or pressing on the affected nerve, the presence of a foreign body, the traction of a cicatrix from neuritis, etc. In such cases the neuralgia is not, strictly speaking, functional. As a functional malady its causation differs in different cases. It proceeds in certain cases from malaria. This is always to be suspected in cases occurring in so-called malarious districts. When due to this cause the paroxysms sometimes recur with the same regularity as the paroxysms of intermittent fever, but it may proceed from malaria when the paroxysms do not exemplify a law of periodicity. It is one of the effects of lead-poisoning. Next to lead colic (and this is, in fact, a neuralgic affection), neuralgia is the most frequent form in which the poisonous effects of lead are manifested. Anæmia, by whatever cause induced, favors the occurrence of neuralgia. A large proportion of those who suffer from neuralgic affections are anæmic.[1] Persons are most likely to suffer from neuralgia between the age of twenty and of fifty. Prior to ten years of age it is extremely rare. Women are more subject to it than men, and of the different species of neuralgic affections, some occur more frequently in the male and some in the female sex. Females, for example, are more subject to intercostal neuralgia, and males to sciatic neuralgia. A neuralgic attack is sometimes referable to exposure to cold, particularly to a current of cold moist air continued upon the part which becomes affected. Cases occur more frequently in the cold than in the warm seasons of the year.

The situation of the pain is not evidence of the seat of the morbid condition giving rise to neuralgia. The mind may refer the pain to the terminal branches or the parts to which they are distributed when the morbid condition is seated at any point in the course of a nervous trunk or at the point of connection of a nerve with the spinal cord. This is illustrated by the fact that after an amputation the condition of the extremities of the divided nerves gives rise to pain which the patient refers to the amputated limb. Anstie held that, as a rule, in the neuralgic affections denominated functional there is some morbid change at the central connection of the nerve or nerves to which the pain is referred. He was also led to think that in a large proportion of cases an hereditary tendency is involved in the causation, and, further, that the development of these affections is favored by exhaustion and debility, aside from anæmia.[2] There is ground for the belief that local diseases may cause neuralgia of nerves at a distance by a reflected morbid influence (reflex or irradiated neuralgias). Weir Mitchell has observed cases in which injury of a nerve was followed by neuralgia in other and remotely-situated nerves.

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[1] With regard to the dependence of neuralgia on anæmia, Romberg says: "It seems as if pain were the prayer of the nerve for healthy blood."

[2] *Neuralgia, and the Diseases that Resemble it*, 1872.

The DIAGNOSIS of neuralgia is to be based on the character of the pain, its situation in the course of a nervous trunk or its branches, and tenderness in certain circumscribed spaces on the surface. Other diagnostic points are the recurrence of pain in paroxysms or marked exacerbations, shifting of the situation of the pain and tenderness, and the difference, as regards pain, between pressure with the ends of the fingers and with the open palm. In the shifting of pain from one nerve to another, which is a highly diagnostic feature, the nervous trunks successively affected do not always have a common origin; and thus the neuralgia may be transferred from a cranial to a spinal nerve. These are the positive diagnostic points. Negative points are those by which inflammation and structural lesions are excluded, such as the absence of fever, etc. In determining the affection to be functional, neuritis is to be excluded, and it is to be ascertained, if practicable, that the pain is not dependent on a local mechanical cause, such as a tumor pressing on the nerve at some point in its course. Syphilitic periostitis is also to be excluded.

The TREATMENT of neuralgia is rational and empirical. The rational treatment consists, in the first place, in fulfilling indications which relate to morbid conditions suspected to stand in a causative relation to the neuralgia. The physician in individual cases is to seek for evidence of malaria or lead-poisoning, conditions having diagnostic criteria irrespective of the neuralgic affection. Appropriate measures of treatment are to be addressed to these or any other associated morbid conditions under the supposition that they may have more or less to do with the existence of the neuralgia. Diathetic conditions, especially the rheumatic and gouty, are to be considered as probably contributing to the neuralgic affection, and with reference to the cure of the latter these conditions claim appropriate measures of treatment. Attention is always to be directed to the coexistence of anæmia. Symptomatic phenomena, together with the venous hum in the neck, may denote the anæmic condition when it is not manifested by pallor of the face and the prolabia. If anæmia coexist, preparations of iron should be continued for a long period. An eligible plan is to give in succession different ferruginous preparations for weeks or months, enjoining upon the patient not to expect restoration to health until after a long continuance of the remedies. This treatment addressed to the anæmia is indicated, provided the anæmic condition continue, notwithstanding the neuralgia may cease. The disappearance of the venous hum is evidence that the anæmic condition is removed.

The relief of pain enters into the rational treatment. In severe paroxysms or exacerbations opiates are called for. Large doses are sometimes indicated by the intensity of pain. The most prompt and effective method of affording relief is the hypodermic injection of a solution of morphia. In the use of opiates for the relief of neuralgic pain the risk of the formation of the habit of taking opium is always to be borne in mind, yet in severe cases which resist therapeutical measures this risk must sometimes be incurred. If the pain be not intense, relief may be obtained by belladonna, hyoscyamus, stramonium, conium, the preparation known as chlorodyne, and the tincture of aconite. Local applications will often do much toward the relief of pain. Of these the most efficient are liniments of the tincture of aconite, chloroform, and opium. Belladonna, stramonium, veratria, and aconitia, applied in the form of ointment, are sometimes efficacious.

An arrest of the paroxysms of pain by opiates is sometimes curative; that is, the paroxysms or exacerbations do not recur. If the development of the affection have been recent, it is not easy to decide whether a cure be effected by the remedy or whether the affection have no intrinsic tendency to continue;

but the paroxysms sometimes cease to recur, after having been once or repeatedly arrested, when the affection has existed for a greater or less period, and in these cases it is fair to attribute a curative efficacy to the arrest. In order to arrest a paroxysm a full opiate should be given as soon as the paroxysm begins, or, if practicable, the occurrence of the paroxysm may be anticipated by a full opiate. Before abandoning this plan of treatment it should be employed in several recurring paroxysms. The continued employment of some form of opiate or of some one of the narcotic extracts, especially belladonna, during the intermissions or remissions, in some cases effects a cure. Gelsemium and the hydrate of chloral or the croton-chloral are to be included among the palliative remedies.

The empirical treatment of neuralgia consists in the use of remedies which experience has shown to be sometimes curative, which are to be tried without following any particular rational indications. These remedies are in some cases efficacious and in other cases devoid of any curative effect; and often there are no means of determining beforehand which one of the remedies will be most likely to prove successful. In rebellious cases the different remedies are to be tried in succession. Methodic counter-irritation is undoubtedly efficacious in a large proportion of cases. Of the different modes of counter-irritation, Valleix preferred the actual cautery, the hot iron being passed along the course of the affected nerve, taking care that cauterization shall be superficial, so as not to lead to deep eschars and suppuration. To avoid the pain occasioned by the cautery he advised etherization. He stated that he had resorted to the actual cautery in more than 150 cases, and always with success if the neuralgia were functional. This method may be resorted to in cases which resist milder means of counter-irritation, together with other empirical remedies. Milder means should first be tried. Of the latter, a succession of small blisters applied over the painful and tender points will frequently prove successful. Prof. Alfred Stillé advises blisters of only half an inch in diameter, the period of application to be from one to two hours only, the vesicated surfaces to be healed as rapidly as possible, and the blisters to be repeated in a few days if the neuralgia continue. He states, as a result of the employment of this method of treatment in almost every case which has come under his observation in public and private practice for many years, that "in no single instance has it failed to mitigate the symptoms, and in very many it alone has achieved a cure."[1] During the process of vesication the neuralgic pain is frequently increased. When quickness of vesication is desirable, it may be effected in a few moments by the strong aqua ammoniæ, and still more quickly by means of a hammer heated by hot water. By the latter means, or by an instrument devised for the operation known as *firing*, a rubefacient effect may be quickly and conveniently produced, and in mild cases this may suffice without vesication. Sinapisms are proverbially useful, and in certain cases will suffice, in conjunction with internal remedies.

Electricity is a therapeutical agent of great power in many cases of neuralgia. Both the faradic and the galvanic forms are useful, but the latter is in most cases to be preferred. Applied in conjunction with acupuncture, it has been successfully resorted to in some rebellious cases. Duchenne recommended the method called cutaneous faradization. The palliative effect of electricity is probably due to a temporary blunting of the excitability of the nerve or nerves to which the pain is referred; their capacity either to originate or to conduct painful sensations being diminished or annulled by passing through them the electrical current. This measure, however, in many cases has not only a palliative but a curative efficacy. The testimony of those who have had great experience in its employment is stronger than that which can be cited in behalf of any other remedy. Strong pressure upon an affected nervous trunk sometimes has a palliative effect. The pressure at first may occasion great pain, but continued or repeated at short intervals it is borne without suffering and diminishes for a time the excitability of the nerve.

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[1] *Therapeutics and Materia Medica.*

Of the internal remedies which experience has shown to be efficacious, the more important are quinia, arsenic, the precipitated carbonate of iron, the preparations of zinc, strychnia, the essential oil of turpentine, the iodide of potassium, and phosphorus.

Quinia, as already stated, is especially indicated when the neuralgia is attributable to malaria, and it will be most likely to prove successful when paroxysms occur periodically; but it is efficacious also in cases in which there is no ground for suspecting a malarious causation and in which there is no observance of the law of periodicity. To be fairly tried, this remedy should be given in full doses—from fifteen to thirty grains daily to an adult—and continued for at least ten days. Of the foregoing list of remedies, this is entitled to the first rank. Arsenic may be tried in cases in which there is an intolerance of quinia or when the latter fails to effect a cure. It is a remedy of great value in neuralgic affections. To be fairly tried, it should be carried to the extent of producing some of its characteristic toxical effects.

The precipitated carbonate of iron, advocated some years ago by Hutchinson, is sometimes efficacious. It is to be given in large doses—namely, from one to two drachms twice or thrice daily. Ferruginous remedies are rationally indicated by coexisting anæmia, and this condition is present in a very large proportion of the cases of neuralgia. The efficacy of the preparation named is doubtless, in a measure at least, attributable to the coexistence of anæmia. The hydrochlorate, nitrate, and valerianate of ammonium have been found in some cases notably efficacious. Bartholow extols as the most successful of the different methods of treatment the injection of from five to ten minims of pure chloroform in the neighborhood of the affected nerve. This treatment is especially useful in neuralgia affecting nerves superficially situated.[1]

Of the preparations of zinc, the valerianate is best suited to cases of neuralgia. Strychnia may be given internally or applied locally upon a blistered surface. In the latter mode of administration the benefit may in some cases be measurably, and perhaps chiefly, due to the counter-irritation. The oil of turpentine is but little used in neuralgic affections, but I have known it to prove promptly successful in doses of a drachm, combined with castor oil given on alternate days, after a variety of remedies had been employed without success. The iodide of potassium has been advocated by Graves, Watson, and especially by Neucort.[2] The writer last named advised its use in large doses. The coexistence of the rheumatic diathesis is regarded as specially indicating this remedy. The bromide of potassium has been found efficacious in certain cases. According to Anstie, this remedy is especially useful in young persons who may be supposed to suffer from the nervous consequences of unsatisfied sexual desire. He thinks its efficacy is rarely secured under a less quantity per diem than ninety grains. In some cases of neuralgia large doses of phosphorus have been found to be of great benefit.

Division of a nerve (neurotomy) and the removal of a section (neurectomy) are surgical procedures resorted to chiefly in cases of neuralgia affecting the fifth or trifacial nerve. They will be noticed in connection with facial neuralgia. Stretching of an affected nerve is another method of treatment, the consideration of which belongs to surgery.

In conclusion, a permanent cure in cases of neuralgia will often depend on the correct appreciation and judicious management of associated morbid conditions, together with hygienic measures to invigorate the general health. Good alimentation is always indicated, and it is desirable that fatty articles of food should enter largely into the diet. Cod-liver oil, if well tolerated, is useful.

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[1] Vide *Practice of Medecine*, p. 630.

[2] "De la Névralgie lombaire, etc.," *Archives générales de Médecine*, 1858.

Trifacial Neuralgia—Prosopalgia—Tic Douloureux.

In this neuralgic affection the pain is seated in the sensory fibres of the fifth or trifacial nerve. Pain and tendernesss on pressure are either limited to, or most marked in, circumscribed spaces situated as follows: The point of exit of the frontal branch above the orbit, the upper eyelid, the upper and lateral portion of the nose, the point of exit of the infraorbitar branch, the lower margin of the malar bone, over the temporal bone, the point of exit of the inferior maxillary branch, and over the parietal bone. It is rare that pain and tenderness are situated in all these spaces, but generally in at least one of the spaces within the range of the three branches of this nerve, whence it receives its name—trifacial. In different cases the pain is either limited to, or especially marked in, different branches of the nerve—namely, the supraorbital, infraorbital, and inframaxillary. It is rare for this affection to be limited to a single branch, with the exception of that portion of the inferior maxillary which is contained within the inferior maxillary bone.

When the first of the three branches of the nerve is the seat of the neuralgia, during the paroxysms of pain there is intolerance of light, with redness of the eye on the affected side, and more or less abundant secretion of tears. In this situation the neuralgic affection has sometimes been called migraine, hemicrania, and browache, but true migraine differs from neuralgia. Increased heat of the nostril and an abundant secretion of mucus are effects of the neuralgic affection when seated in the second of the three divisions, and salivation sometimes attends an affection of the third division. The branches which accompany the facial nerve (portio dura) may be the seat of pain; a fact which formerly led to the supposition that the latter nerve was affected. Spasmodic contraction of the muscles of the face on the affected side is an occasional effect; hence the affection was formerly called *tic douloureux*, or painful spasm. The affection in the vast majority of cases is limited to the nerve on one side; that is, it is unilateral. The right and the left side are affected in about an equal proportion of cases.

Trifacial neuralgia in point of frequency ranks with intercostal and sciatic neuralgia. An occasional cause is caries of the teeth. It is by no means frequently referable to this cause, but that it is so occasionally cannot be doubted. Teeth, however, are often needlessly sacrificed from a supposed causative connection. It is not sufficient to show such a connection that there is tenderness on striking one or more of the teeth. If the neuralgia proceed from this cause, touching the offending tooth or teeth will excite a paroxysm of pain extending into more or less of the branches of the nerve.

Trifacial neuralgia is to be discriminated from inflammation of the membrane lining the supraorbital and the maxillary antrum and from syphilitic periostitis. The two former of these affections are distinguished by the different character of the pain, its fixed situation, and the occurrence of a discharge into the anterior nares. The latter affection is recognized by the fixed tenderness on pressure, the existence of swellings or nodes, the aggravation of pain in the night-time, and other evidence of syphilitic disease. Neuralgia in this situation is often dependent on malaria, and the paroxysms of pain sometimes exemplify the same laws of periodicity which govern the recurrence of the paroxysms of intermittent fever. Trifacial neuralgia rarely occurs in infancy. In the majority of cases the age is between thirty and fifty years, and it occurs oftener in women than in men.

This neuralgic affection is curable in the majority of cases. Exceptionally, it resists all the therapeutical measures which have been considered as applicable to neuralgic affections in general. The branches which are generally affected in these rebellious cases are the frontal and the inferior maxillary. As a last alternative, division or excision of a portion of the affected branch is sometimes resorted to. The long-continued and extreme suffering from frequently recurring paroxysms in some cases warrants this procedure after other measures have been faithfully tried. Mere division of a nervous branch usually affords only temporary relief. The affection returns after the divided extremities have united. A portion of the nervous branch must be removed to prevent or delay reunion. The removal of a portion of the inferior maxillary nerve within the inferior maxillary bone has sometimes proved successful after the failure of all other measures; but in a considerable proportion of cases the affection returns sooner or later. In a case under my observation, in which the patient has suffered from paroxysms of intense pain recurring at intervals of a few moments for the greater part of ten years, the removal of a portion of the nerve by trephining the inferior maxilla, the operation being performed by Prof. Hamilton, procured relief for several months, but the pain subsequently returned.

Facial neuralgia, persisting indefinitely, resisting medication, and characterized by spasmodic movements of the muscles of the face during the paroxysms of pain, is distinguished by Trousseau as *epileptiform neuralgia.* There are few forms of disease more distressing than this or more hopeless as regards the prospect of effecting a cure. A case which came under my observation exemplified the persistency of this form of neuralgia, and also exemption from suffering for an unusually long period procured by simple division of the nerve. The patient, a man aged about sixty, had suffered intensely from neuralgia affecting the supraorbital branch of the fifth pair on the left side for more than two years. The paroxysms of pain recurred at intervals of a few minutes, and were often excruciating beyond description. After trying a great variety of remedies and different systems of practice, the nerve was divided by Valentine Mott, and the patient was comparatively free from suffering for three years. At the end of this period, while under treatment for enlargement of the heart and general dropsy, the neuralgia suddenly returned with the same violence as before the operation. Paroxysms were produced by mastication, by movements of the lips in pronouncing words containing labial letters, by touching the face, and they occurred without any exciting cause. The nerve was divided a second time by Prof. J. R. Wood, with immediate and almost complete relief, which continued until the patient's death, several weeks after the operation.

Carnochan was the first to perform neurectomy beyond the spheno-palatine ganglion for the relief of persistent neuralgia of the superior maxillary branch. He reported three cases of successful performance of this operation in 1857.[1] This operation, since that date, has been performed by James R. Wood and others with success.[2]

Facial neuralgia, after having persisted for a long period, sometimes occasions disturbance of nutrition on the affected side of the face, denoted by tumefaction, an increase of adipose tissue, thickness and firmness of the beard, and it sometimes appears to give rise to pustular or erythematous inflammation. An analysis of 128 cases by Notta showed, as occasional complications, in addition to those already mentioned, paralysis, either complete or incomplete, of the retina, iritis, glaucoma, inflammation and ulceration of the cornea, tumefaction of the tongue on one side, paralysis of the motor oculi, deafness, and anæsthesia of the face on the affected side.[1] Certain of these complications are attributable to paralysis of the vaso-motor nerves. Anstie, who verified the correctness of Notta's observations, observed, in addition, grayness of the hair on the affected side and discoloration of the skin from the presence of dark pigment.[2]

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[1] *Amer. Journ. of Med. Sciences,* vol. xxxv.

[2] For an analysis of 21 cases, and the practical conclusions deduced therefrom, vide article on "Neurotomy of the Superior Maxillary Branch of the Trigeminus," by Frederic S. Dennis, M.D., in the *New York Med. Journal,* June, 1879.

Cervico-occipital Neuralgia.

The pain in this neuralgic affection is seated in the posterior branches of the first four of the cervical nerves, especially in that branch of the second cervical nerve called, from its size and distribution, the occipitalis major. The nerves on both sides may be affected.

Painful and tender points are situated as follows: Between the mastoid process and the cervical vertebræ, over the posterior cervical plexus, on the parietal portion of the head, on the mastoid process, and in the concha auris. Shooting pains start from the cervical vertebræ just below the occiput, and extend over the posterior and superior portion of the cranium. Darting pains are sometimes referred to the external auditory meatus. In cases of neuralgia affecting primarily the cervico-occipital nerves, the pain is likely to extend to the branches of the trifacial distributed to the frontal portion of the head, and in like manner in cases of neuralgia affecting primarily the superior division of the trifacial the pain often extends to the branches of the cervico-occipital nerves.

Cervico-occipital neuralgia is to be discriminated from myalgia affecting the muscles of the neck, or *torticollis*. In the latter affection the pain is limited to the affected muscles, the tenderness on pressure is more diffuse, and it is not confined to circumscribed points. The pain is excited exclusively by movements of the affected muscles, and so long as the head remains motionless there are no painful paroxysms.

Cervico-brachial Neuralgia.

In cervico-brachial neuralgia sensory fibres of the four lower cervical nerves and of the first dorsal nerve are affected. The pain is frequently referred to the shoulder, and it may extend to the neck. It shoots downward along the arm to the forearm and hand, following the course of one or more of the nervous trunks of the upper extremity. The pain may be limited to the ulnar, radial, median, or musculo-cutaneous nerve, giving rise to different varieties of the affection. Of these varieties the most frequent is neuralgia affecting the ulnar nerve. Painful and tender points are situated as follows: Near the last cervical vertebra, near the acromial end of the clavicle, over the upper part of the deltoid muscle, in the axilla, at the inner condyle of the humerus, and near the lower end of the ulna and radius.

Pain referable to the cervico-brachial nerves is sometimes an effect of injury of the upper extremity by a wound, a burn, or a contusion. Functional neuralgia is to be discriminated from the pain incident to neuroma or other morbid growths affecting the periphery of these nerves by the failure to discover a tumor in the course of the nerves. Pain referred to these nerves may be symptomatic of vertebral lesions, and also of cervical arthritis. In cases of the latter, movements of the head, especially those of rotation, even in the intervals between the paroxysms, are painful. Another feature is, that the patient when lying down is obliged to raise the head with a hand on the occiput to avoid pain. Myalgia affecting the muscles of the upper extremity is to be discriminated by the larger extent of surface which is tender on pressure, by the absence of paroxysms of pain except when provoked by movements of the affected muscles, and by the limitation of the pain to the affected muscles.

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[1] *Archives générales de Médecine*, 1854. Vide, also, treatise by Anstie.

[2] For a report of cases observed and collected by Prof. Henry D. Noyes, with reference particularly to the effect upon the eye of paralysis of this nerve, vide *New York Journal of Medicine*, vol. xiv. p. 163.

Herpetic vesicles in patches, constituting herpes zoster, sometimes occur in the course of the nerves involved in this affection.

Dorso-intercostal Neuralgia.

This affection has been considered in the section devoted to diseases affecting the Respiratory System, to which the reader is referred (p. 183).

Lumbo-abdominal Neuralgia.

Neuralgia having its seat in the branches of the five lumbar nerves is of not infrequent occurrence. It occurs much oftener in females than in males. Pain is often referred to the hypogastrium, and it may resemble that of colic. Uterine pain and ovarian pain are incident to this affection. In males the affection is sometimes characterized by pain extending along the spermatic cord into the scrotum, accompanied by notable tenderness of the testicle.

Painful and tender points are situated near the upper lumbar vertebræ, a little above the middle of the crest of the ilium, in the neighborhood of the inguinal ring, in the scrotum, and in the female on the labia majora. Pains are excited by muscular efforts, the acts of coughing or sneezing, straining at stool, and sometimes by micturition.

Myalgia affecting the lumbar muscles, or lumbago, is to be discriminated by the existence of pain and tenderness on both sides, whereas in neuralgia they are generally confined to one side; by the tenderness being diffused over the affected muscles, and not limited to certain points; and by the absence of paroxysmal pain except when produced by movements of the affected muscles.

In the case in which pain extends to the uterus, inflammation of the organ, or metritis, and carcinoma are to be excluded. In uterine neuralgia the os uteri is tender on pressure, and the tenderness is either limited to or most marked on one side. The pains in these varieties of neuralgia may simulate those caused by the passage of a calculus along the ureter, or renal colic. The pains in the latter are more intense and persisting; and they are developed suddenly, whereas the development of a neuralgic affection is generally gradual. The absence of symptoms which distinguish renal colic warrants its exclusion.

Crural Neuralgia.

This neuralgic affection is rare. It is characterized by shooting pains in the direction of the trunk of the anterior crural nerve and its branches, and by painful and tender points in the groin, the inner aspect of the thigh, knee, and ankle.

Sciatic Neuralgia.

Sciatic neuralgia, or sciatica, in frequency ranks with intercostal and facial neuralgia. It occurs in men oftener than in women, the relative proportion of cases being as three to two. It very rarely occurs before the age of twenty and rarely after sixty. It is sometimes symptomatic of pregnancy, of enlargement of the retro-peritoneal lymphatic glands, of accumulation of feces or foreign substances in the sigmoid flexure of the colon, and of intrapelvic tumors; but in the great majority of cases it is a functional affection.

As in the foregoing neuralgic affections, shooting pains, in paroxysms or exacerbations, extend along the trunk and branches of the affected nerve. In indicating the course of the pain the patient often delineates with accuracy the anatomical distribution of the sciatic nerve. The character of the pain is sometimes described as burning and sometimes as a sensation of coldness. The patient feels as if a current of hot or cold liquid were flowing in the course of the affected nerve. A continued contusive pain, more or less intense, exists in the larger proportion of cases. This pain, as in the other varieties of neuralgia, is referred to the circumscribed spaces which are tender on pressure.

Painful and tender points in this neuralgic affection are found in certain of the following situations: On the sacrum, over the sacro-iliac junction, about the middle of the crest of the ilium, at the sciatic notch, between the trochanter major and the tuber ischii, on the thigh along the track of the nerve, in the popliteal space, on the external border of the patella, over the articulation of the fibula with the tibia where the nerve passes around the fibula, over the lower and posterior part of the external malleolus, and on the dorsum and the outer portion of the soles of the feet. The points situated at the sacro-iliac junction, behind the trochanter major, at the head of the fibula, and the external malleolus, are those most frequently painful and tender. Pressure with the finger upon some of the foregoing points may not only be more or less painful, but excite a paroxysm or exacerbation of pain extending to other parts in the course of the affected nerve.

The intensity of the pain, as in the other kinds of neuralgia, varies much in different cases. The movements of the affected limb in some cases occasion such an amount of suffering that the patient remains in bed, keeping the limb as nearly motionless as possible. In other cases the patient is able to walk, but with more or less difficulty. Pain is felt especially when the weight of the body falls upon the affected limb, and relief is found in the use of a cane. Sometimes difficulty in walking is felt only in beginning to walk, and after a short time the patient walks with ease. The suffering is occasionally relieved by exercise. In severe cases muscular twitchings and cramps accompany the paroxysms or exacerbations of pain.

Sciatic neuralgia rarely, as compared especially with facial neuralgia, exemplifies in the recurrence of paroxysms the law of periodicity. Like other neuralgic affections, its duration is very variable. As a rule, in favorable cases it persists for several weeks, disappearing, as it is usually developed, gradually. In the vast majority of cases sooner or later it ends in recovery, but in some cases it continues indefinitely in spite of all remedial measures. If it continue for a considerable period so severely as to lead the patient to avoid as much as possible movements of the affected limb, the repose of the muscles may render them for a time incompletely paralyzed. The muscles under these circumstances become flaccid and diminished in volume.

The DIAGNOSIS of sciatic neuralgia involves its discrimination from muscular rheumatism and from the affection of the hip-joint commonly known as morbus coxarius. Muscular rheumatism here, as in other situations, differs from neuralgia in the limitation of pain to the affected muscles, in the diffusion of tenderness over a considerable space—namely, a space corresponding to the number of muscles affected—and in the fact that paroxysms of pain are excited exclusively by movements of the affected muscles.

Morbus coxarius is distinguished by the pain in the hip-joint produced by pressure upon the great trochanter and by flexion and extension of the thigh. Moreover, febrile movements, general debility, emaciation, etc., in conjunction with the local symptoms, point to the existence of chronic inflammation within the joint.

The term sciatica, as commonly used, is applied to cases in which pain is referred to sacral nerves, although the sciatic nerves may be unaffected. Pain of a neuralgic character in the nerves composing the sacral plexus is often symptomatic of lesions within the pelvis and abdomen, such as aneurismal, carcinomatous, or other tumors, enlargement of glands, etc. and of lesions of the sacrum and vertebral column and those occurring within the spinal canal. Here, too, as in other situations, it may be dependent on neuritis. This is to be inferred from the speedy occurrence of paralysis and atrophy of the muscles to which the nerves are distributed.

The coexistence of two or more of the foregoing different neuralgic affections or their successive occurrence is frequently observed. Proximate or inosculating nerves may be simultaneously or successively affected. Thus, trifacial and cervico-occipital neuralgia are not infrequently associated, and this is true of sciatic and crural neuralgia. A cure or spontaneous cessation of neuralgia in one situation is likely to be followed by the development of the affection in another situation. These facts go to show the dependence of neuralgia on constitutional or general morbid conditions.

Dermalgia.

Neuralgia is sometimes limited to the skin. Cases are occasionally observed in which pain is felt on the surface of the head, trunk, or extremities, together with notable tenderness on pressure, the nervous trunks being free from pain or tenderness. This form of neuralgia has been called *dermalgia*. Under this heading may be placed the form of neuralgia described by Weir Mitchell as incident to injury of nerves, and called by him causalgia. This is characterized by a burning pain in the soles of the feet and palms of the hands. I have reported an instance of causalgia affecting the soles of the feet, occurring without any injury of a nervous trunk or any other symptom; also, a case in which a paroxysmal burning pain was referred to the abdomen and lower limbs.[1]

Myalgia—Muscular Rheumatism.

Neuralgia affecting sensory nerves in muscles may be distinguished as myalgia. Under this head I shall enumerate certain local affections which it has been customary to embrace under the name *muscular rheumatism*. The term rheumatism, as applied to these affections, is manifestly inappropriate, and it is desirable that the name in this application should become obsolete. The myalgic affections were designated by Valleix by the suffix *dynia*, added to the name of the muscle or group of muscles affected. This designation, having the same derivative meaning as words ending in *algia*, is appropriate.

Myalgia is of frequent occurrence as an acute and a chronic affection. The symptomatic features of the acute affection are as follows: The development is usually gradual. A dull pain is at first felt in certain muscles, which increases, and at length becomes more or less severe. The pain is comparatively slight when the affected muscles are at rest. In certain positions the patient may be nearly or quite free from suffering. A constant pain of a contusive character is often felt when the muscles are at rest, and this may be aggravated by spasmodic pains occurring from time to time. In movements which involve contraction of the affected muscles, the pain is severe, cramp-like, and sometimes so excruciating that the patient can hardly repress loud groans or cries. Voluntary movements which occasion the severe pain are avoided as much as possible, but as it is impossible to maintain rigidly-fixed positions of the body as a whole or of its different parts, the affected muscles are at times thrown into painful contractions, however much the patient may desire to avoid them. Movements occurring during sleep occasion the attacks of pain, and hence the patient is awakened at brief intervals. In some cases the affection changes its seat from certain muscles to others, but oftener it remains fixed in the muscles first affected.

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[1] Vide *Clinical Medicine*.

The affected muscles are more or less tender on pressure, the tenderness being diffused over the whole of the space which the muscles occupy, and not limited to certain points as in ordinary neuralgia. The tenderness is usually not great. Aside from the tenderness, there are no local characters, such as swelling, heat, or redness. The local phenomena are not those of inflammation, as in articular rheumatism. If the local symptoms denote inflammation of the muscles, the affection is not myalgia. Uniform firm pressure over the affected muscles is not only well borne, but it affords relief in cases of muscular rheumatism.

There is little or no constitutional disturbance beyond that occasioned by the suffering and loss of sleep. The appetite and digestion may not be impaired, and there is no fever.

In the subacute or chronic form of the affection pain similar to that which belongs to the acute form may be excited only when the affected muscles are contracted with unusual force. The patient experiences suffering when he makes certain violent .movements. In other cases pain is excited by particular movements when they are first made after a period of rest, and the pain shortly disappears if the movements be continued. The chronic oftener than the acute affection is likely to shift its situation, affecting now certain muscles and now those in another part. If the chronic affection remain fixed in particular muscles, they may become somewhat atrophied in consequence of their being kept as much as possible in a state of inaction.

The DURATION of acute myalgia varies between a few hours and a week. Exceptionally the affection continues for several weeks. The duration of the chronic form is indefinite. It is often a very obstinate affection, persisting for many months. The course of the latter is usually marked by remissions or intermissions. In certain situations—namely, the loins, the walls of the chest, and muscles of the scapulæ—the chronic affection is likely to be long persisting.

The following are the principal situations of myalgia in the acute and the chronic form:

1. *The Muscles of the Head.*—Situated in the occipito-frontal muscle, the affection is distinguished from neuralgia affecting the trifacial and the occipital nerve by the pain being referred to both sides of the head, by its being excited by movements of the muscle and comparatively slight so long as the muscle is at rest, and by the absence of tenderness in disseminated points. The muscles of the eye are sometimes affected. Movements of the eye then occasion more or less severe pain. The temporal and the masseter muscle may be affected, causing pain in mastication.

2. *The Muscles of the Neck.*—In this situation the affection, as it commonly occurs, has been known by the name *rheumatic torticollis.* This name is applied to the affection when situated in the muscles of the antero-lateral aspect of the neck. The muscles on the posterior part of the neck are sometimes the seat of the affection. Valleix applied to the affection in the latter

situation the name *cervicodynia.* In this situation the affection is to be distinguished from ordinary occipital neuralgia.

3. *The Muscles of the Back.*—Valleix called the affection in this situation *dorsodynia.* Seated in these muscles, the pain is caused especially by movements which approximate the scapulæ.

4. *The Muscles of the Loins.*—In this situation the affection is commonly known as *lumbago.* Valleix designated it *lumbodynia.* This is a frequent seat of the affection in a chronic form. It is to be discriminated from lumbar abscess, lumbo-abdominal neuralgia, and the pains incident to aneurism of the descending aorta.

5. *The Thoracic Muscles.*—Here the affection goes by the name *pleurodynia.* In making the diagnosis pleuritis and intercostal neuralgia are to be excluded. This may easily be done by ascertaining the absence of the diagnostic characters of the latter two affections. The pain excited by forced breathing, coughing, and sneezing is not unlike that in pleuritis and intercostal neuralgia. In this situation the pectoral and the intercostal muscles are affected.

6. *Muscles of the Shoulder.*—Valleix entitled the affection here situated *scapulodynia.* It is seated especially in the deltoid muscles, and it is liable to be confounded with arthritis situated in the shoulder-joint.

7. *The Muscles of the Extremities.*—Seated in the extremities, the affection is to be discriminated from neuralgia affecting the nervous trunks, and from the affection commonly known as syphilitic rheumatism.

8. *The Abdominal Muscles.*—The pain and soreness caused by the affection in this situation may suggest as probable or possible the existence of peritonitis. This disease is to be excluded by the absence of the general symptoms and by firm pressure with the palm of the hand being well borne, as in cases of cutaneous hyperæsthesia.

9. *The Visceral Muscular Structures.*—The tongue and the muscles of the pharynx and œsophagus may be the seat of myalgia, giving rise to pain in speaking and in deglutition. Examples, however, are extremely rare. Seated in the stomach, intestines, and uterus, as it probably may be, to distinguish the affection from visceral neuralgias is impossible; and in fact to consider it as distinct from the latter is to make a distinction without any essential difference. There is reason to believe that the diaphragm may be the seat of myalgia. It is perhaps more reasonable to consider this as the affection, rather than diaphragmatic pleuritis, in certain cases characterized by the instinctive arrest of diaphragmatic movements in consequence of pain, and by the intense suffering caused by coughing and sneezing, febrile movement and pleuritic effusion being wanting. It may be doubted if the affection be ever seated in the muscular walls of the heart.

In all these situations the most marked diagnostic feature is the connection of cramp-like pain, more or less severe, with the movements which bring into play certain muscles in the parts to which the pain is referred, pain, irrespective of contractions of the affected muscles, being comparatively slight and sometimes wanting. This diagnostic feature distinguishes myalgia from neuralgic affections seated in the different nervous trunks. Another diagnostic feature is the diffusion of soreness over the affected muscles, whereas neuralgia seated in nervous trunks is characterized by tenderness in circumscribed points. Inflammation affecting muscles is distinguished from myalgia by the constancy of pain or the absence of the remissions or intermissions which belong to the latter affection, by the existence of swelling and sometimes redness, by the greater degree of tenderness on pressure, and by the presence of more or less symptomatic fever.

The **TREATMENT** of the acute form of the affection consists of sinapisms, followed by soothing, emollient applications over the affected muscles, rest in the position most favorable to avoid contractions of the affected muscles, and the employment of anodynes either internally or by hypodermic injection. In the chronic form small blisters in succession are useful, but not in the same degree as in cases of ordinary neuralgia. The employment of the induced electrical current I have known to be beneficial in obstinate cases. The constant current has also been found highly efficacious. Friction or shampooing, either dry or with stimulating or anodyne liniments, affords relief and promotes a cure. "Firing" is recommended. The bromide of potassium and of ammonium I have found useful. The hydropathic treatment is said to prove successful in some obstinate cases, water being employed locally and generally. The salts of quinia in full doses are sometimes efficacious, especially when the affection is intermittent. Measures to invigorate the general health are important in chronic cases. If anæmia exist, preparations of iron should be continued for a long time in small doses or until the venous hum disappears from the neck. Anstie recommends as especially useful the muriate of ammonia in doses of from twenty to thirty grains.

Persons liable to this affection are likely to have an attack after exposure of a portion of the body to a current of air, especially during sleep or when the body is perspiring. The susceptibility to this cause of disease is to be overcome by the daily use of the sponge-bath, by avoiding an over-abundance of clothing, and by becoming habituated to out-of-door exercise in all kinds of weather.

Cephalalgia—Headache.

Pain in the head is a symptom occurring in many affections, such as active cerebral congestion, cerebral meningitis, cerebritis, the essential fevers, acute dyspepsia, syphilitic periostitis of the head, intracranial tumors, etc. etc. It is frequently due to neuralgia affecting either the cervico-occipital nerves or the supraorbital division of the fifth pair. Pain limited to one side of the head, or hemicrania, may be due to neuralgia affecting the nerves just named. According to Valleix, idiopathic cephalalgia or headache is in all cases resolvable into hemicrania (migraine). This opinion does not seem to me to be correct. In certain cases the pain does not follow the direction of these nerves, it is not limited to one side of the head, disseminated tender points may not be found, and in its behavior the affection differs from neuralgia affecting these or other of the nervous trunks and branches. Hence, cephalalgia or headache may denote a functional neuropathic affection distinct from hemicrania. The seat of the affection when confined to one side in certain cases has been supposed to be the branches of the trifacial nerve which are distributed to the dura mater. It has also been supposed that the sensory filaments entering into the composition of the cerebral membranes and of the brain may be the seat of neuralgia.

Cephalalgia is extremely common. It occurs in paroxysms, lasting usually from twelve to twenty-four hours, in some cases being of shorter duration, and occasionally continuing for two or three days. There are few persons who do not occasionally experience an attack after childhood or adult age, and there are very many who are subject to attacks recurring sometimes at regular, but usually at irregular, intervals for many years or during the lifetime. A host of persons suffer from it who either do not consider it of sufficient importance to resort to medical treatment, or, having found medical treatment ineffectual, resign themselves to it as an irremediable malady.

The pain in different cases or in different attacks in the same case differs much in degree. An attack is often not severe enough to prevent habitual occupation, but in some instances the intensity of the pain is such that the patient is unable to be about, and perhaps is obliged to take to the bed. The pain is sometimes referred to the forehead, sometimes to the occiput, and sometimes to the whole head. The character of the pain as described by patients differs. It is sometimes sharp or lancinating, like the pain in neuralgic paroxysms; and in other cases it is a dull, heavy pain. Patients sometimes say that they feel as if the head would burst. Firm pressure upon the head, especially over the temples, often affords momentary relief. Frequently rubbing or shampooing the scalp with considerable force diminishes the pain. There is frequently an abnormal sensitiveness to light and sounds, and in severe attacks the patient seeks perfect quietude in a dark room away from all noise. The appetite during the attack is either impaired or lost. The odor of food is often disagreeable. Nausea and vomiting occur in a certain proportion of cases, and then it is common to call the affection megrim or *sick headache.* Without nausea or vomiting the affection is commonly known as *nervous headache.* The face is in some cases flushed, the eyes are suffused, and the temperature of the head is raised; and in other cases, especially if nausea and vomiting occur, the face is pallid and the head may be cool. Free vomiting, although the stomach may not contain any aliment, is sometimes followed by relief.

Accompanying the foregoing local symptoms is more or less general debility or prostration. There is rarely fever, the pulse is generally small and feeble, and the surface of the body cool. Occasional shiverings are not uncommon. Disturbed action of the heart, or palpitation, is not uncommon during the attack.

The attack gradually passes off, sometimes with the occurrence of free evacuations from the bowels or an abundant secretion of urine or a slight perspiration. The attack is often developed in the morning, pain being experienced on awakening from sleep, and it may continue until after sleep is obtained on the following night. Persons subject to the affection are able sometimes to predict its advent by a feeling of lassitude, depression of spirits, or a sense of indefinite discomfort. After the attack has passed off, there is frequently, for some time, a sense of soreness in the head, with more or less general debility. Often, however, the mental faculties are unusually clear and the spirits are buoyant after recovery from an attack.

The affection involves a constitutional predisposition. Persons are subject to periodical attacks of headache, as of asthma or epilepsy. The predisposition existing, various exciting causes may determine an attack, such as mental excitement, deprivation of sleep, bodily fatigue, exposure to the sun's rays, and dietetic imprudences. Not infrequently attacks occur without any obvious exciting causes. Women are more subject to the affection than men, and it often occurs at the menstrual period. The predisposition varies in degree in different cases, as shown by the frequency or rareness of the attacks. The predisposition frequently ceases after the age of forty or fifty; and persons who have experienced attacks more or less frequently from adult age up to the periods of life just named, thereafter are exempt from their recurrence. This may be stated as a rule, but, unhappily, there are not a few exceptions.

The affection is neuralgic in its character, but it is difficult to determine its precise seat. It is a reasonable supposition that it involves a toxical agent in the blood. Cephalalgia, it is well known, is produced by different toxical agents—namely, opium, alcohol, strychnia, quinia, and the accumulation of urea. Assuming the correctness of this supposition, the nature and source of the toxical agent or agencies are unknown. It has been conjectured that the immediate causative condition may be either spasm or paralysis of the muscular fibres of the arteries within the skull from an influence exerted through the vaso-motor nerves. The pallor and coolness of the face and head in some cases, and the redness and heat in other cases, sometimes bilateral and sometimes unilateral, afford support to this conjecture; and it is not an incongruity that anæmia and congestion alike may give rise to the cephalalgia. In accordance with these pathological views, inhalation of the nitrite of amyl has been suggested as an appropriate remedy when the appearances denote anæmia, and ergot when there is congestion. Assuming the existence of spasm or paralysis of the arterial muscles, it is reasonable to suppose that the vaso-motor influence depends on a toxical agent acting on the sympathetic nerves. It has been shown that a certain number of cases of migraine are of reflex origin. In certain cases, migraine is the result of some affection of the nasal mucous membrane, and in obscure cases it is well to make an examination of this region. Other cases of migraine appear to be of a myalgic nature, the occipito-frontalis muscle being chiefly involved.

Periodical headache, as regards successful treatment, belongs among the opprobria of medical art, yet in not a few cases much benefit may be derived from treatment. The treatment embraces measures to be employed at the time of an attack and during the intervals.

If patients be not affected unpleasantly by opiates, an attack may be sometimes warded off or abridged or its severity lessened by a full dose of opium in some of its forms, or of its alkaloids, morphia or codeia. The carbonate of ammonia is sometimes successful in affording relief. A saline purgative, taken at the beginning of the attack, is in some cases an effectual remedy. The bromides given in full doses are sometimes efficacious. This statement will also apply to the hydrate of chloral in doses of fifteen grains, repeated once or twice after an hour's interval if relief be not obtained.

Various palliative measures may be resorted to with advantage. The inhalation of a little of the vapor of chloroform is sometimes an efficient palliative. Evaporating lotions to the head—alcohol, spirits, vinegar, or ether—in some cases afford marked relief. In some cases a towel or napkin wrung out in water as hot as can be borne, and wound around the head, is more efficient than cold applications. Warm stimulating pediluvia are useful. Strong coffee or tea affords marked relief in some cases. Electricity has been found in some cases effective in arresting and preventing the recurrence of the paroxysms. The electrical treatment should not be abandoned until after a fair trial of the faradic and the galvanic current. Guarana, given in the form of powder, infusion, or extract, sometimes is an efficient palliative remedy. This is an uncertain remedy, and generally after a few paroxysms it fails to afford relief. In cases of migraine dependent upon some disease of the nasal mucous membrane local treatment applied to this part has proved effective.

During the intervals remedies which are sometimes useful by way of prophylaxis are—nux vomica or strychnia in small doses, arsenic, small doses of quinia, belladonna, and the preparations of zinc, especially the valerianate. These remedies may be tried in succession. Hygienic measures are important. In general terms, the liability to attacks is less the more closely the laws of health are observed. Avoidance, as far as possible, of everything which experience shows in individual cases to act as exciting causes is an important part of the management.

CHAPTER VII.

THE NEUROSES.

Chorea.—Anomalous Muscular Movements.—Local Spasms.—Tremor.—Epilepsy.

CERTAIN of the neuroses or functional affections of the nervous system are characterized by disordered muscular movements. Chorea is one of these affections. Anomalous movements proceed from disordered volition. Disorders manifested by involuntary movements are grouped under the names tremor and local spasms. In these groups belong senile trembling or paralysis agitans, writers' cramp, and athetosis. Epilepsy is an important functional affection. The foregoing affections will be considered in this chapter.

Chorea.

The affection called *chorea*, *chorea sancti Viti*, or *St. Vitus's dance* is characterized by irregular clonic contractions of more or less of the voluntary muscles, giving rise to movements which are either involuntary or not under the guidance of the will. The manifestations of the affection are usually at first limited to a portion of the body, to one of the upper extremities oftener than elsewhere, and sometimes they are confined for a time to the fingers of one hand. The movements increase and progressively extend to both upper extremities, the muscles of the face, and frequently to the lower extremities and to the trunk. The appearances consist of frequently-recurring or unceasing movements of the parts affected, producing in the face varied and ludicrous grimaces, and in the extremities and trunk grotesque contortions and fantastic gesticulations.

The spectacle afforded by the manifestations of this affection is comical, and it is difficult to realize that it is not a performance for the amusement of observers. The condition has been well designated an "insanity of movement." The condition, however, if the affection be severe, is truly distressing. The incessant activity of the muscles induces fatigue. The patient may be unable to perform the voluntary acts which the wants of the body require, and is completely dependent upon others. Speech may be impaired or lost. Locomotion may be impossible. The isolation is sometimes more complete than in cases in which a considerable portion of the body is paralyzed or several senses are abolished. Want of ability to direct and harmonize voluntary movements is a feature of this affection. Acts of volition give rise to the irregular movements, but movements to a greater or less extent take place without the stimulus of volition; that is, they are involuntary. In the latter respect chorea differs from locomotor ataxia, the movements in ataxia being never wholly involuntary. The movements in chorea are usually suspended during sleep, but they sometimes continue, although always more or less diminished. They are increased generally when the patient is conscious of being observed and under any emotional excitement. The movements are frequently more marked on one side of the body, and this is generally the left side. They are sometimes confined to this side, and the affection is then called *hemichorea.* The choreic manifestations are confined to the voluntary muscles. The sphincters are rarely affected; and this is true of the muscles concerned in respiration, inclusive of the laryngeal muscles, as also those of the pharynx. In extreme cases the disordered movements are so violent as to give rise to contusions and excoriations unless the patient be properly protected. In consequence of incessant movements it may be difficult or impossible to feed the patient. This severe form of the disease is called *chorea major*, and it is happily very rare.

The affection is not accompanied by fever. If this exist it is due to an intercurrent or coexisting affection. The appetite is usually more or less impaired and the bowels are constipated. Anæmia often coexists. Irritability of temper is a notable feature. The mental faculties are weakened. The grimaces give to the physiognomy an appearance of idiocy, and if the affection becomes chronic patients fall into a condition of apparent imbecility. The mind fails in these cases from the deficiency of exercise of the mental faculties. If the speech be lost, it is difficult to estimate the amount of intelligence which is preserved. The power of the affected muscles is more or less diminished, and this impairment of power or incomplete paralysis is more marked on the side more affected with the chorea. Anæsthesia of the surface on one or both sides of the body is sometimes observed; oftener, however, there is hyperæsthesia, which is especially marked in the tract of nerves. Tenderness along the spinal column exists in a large proportion of cases. There is an exaggerated susceptibility to reflex excitations, and the electrical excitability of both the muscles and skin is abnormally increased. Pressure upon tender points over nervous trunks excites the choreic movements.

Endocardial murmurs are not infrequently found in cases of chorea. If these be aortic or pulmonic, accompanying the first sound of the heart, they are to be regarded as inorganic or anæmic murmurs. They do not constitute evidence of endocarditis. Nor is a mitral murmur in this affection always evidence of endocarditis. I have observed a mitral systolic murmur during the continuance of chorea, and the disappearance of the murmur after recovery from the chorea.

As one of the neuroses, this affection of course has no ascertained anatomical characters. In cases which have proved fatal morbid changes, not uniform in character or seat, have been found in the nervous centres. Minute embolic obstructions in the corpus striatum have been observed by J. Hughlings Jackson and Bastian in hemichorea. The opinion is held by many that there is a pathological connection between it and rheumatism. The statistical researches of Sée and Henri Roger in France appear to show that in a considerable proportion of cases chorea either follows or is coincident with rheumatism. The same results have not been obtained by the analysis of cases in Germany. Endocarditis appears to coexist in a certain proportion of cases without other evidence of rheumatism; but it is probable that in some of these cases the existence of endocardial inflammation has been incorrectly inferred from physical signs. It is certain that endocardial inflammation does not exist in a sufficient number of cases to afford any ground for the theory of Kirkes that chorea is caused by the passage into the blood of the inflammatory products of endocarditis. It is perhaps correct to say that there is no essential pathological connection between rheumatism and chorea, but that in children the former predisposes to the latter disease.

The CAUSATION is obscure. It is common to suspect the presence of worms in the alimentary canal, but there is little evidence that the affection is caused by worms. It is sometimes traced distinctly to fright or to violent mental excitement, as a fit of anger. Its development is favored by anæmia, and in most cases patients are anæmic. There is reason for suspecting sexual excitation as a cause in some instances. It may be produced by unconscious imitation. This has been shown repeatedly by its prevalence in schools. It occurs occasionally in pregnancy (*chorea gravidarum*).

Chorea occurs especially between ten and fifteen years of age. It rarely occurs at less than six years of age. It may occur at any period of life. Henri Roger has reported a case occurring in a female aged eighty-four years. Graves observed a well-marked case in a male aged seventy-two years. On the other hand, cases have been observed in infants shortly after birth. Girls are more liable to it than boys, the ratio being as three to one.

The DURATION varies from a few weeks to several months. The average duration is between two and three months. In the vast majority of cases the termination is in recovery. After a variable duration the affection tends intrinsically to this termination. In a very small proportion of cases it becomes chronic and is incurable. Life may be destroyed by intercurrent affections which, in general, do not suspend the choreic phenomena. A fatal result may take place without any superadded disease, the patient being worn out by prolonged irritation, loss of sleep, and inanition. The incessant movements sometimes give rise to contusions and excoriations which may lead to erysipelas and abscesses, and these may contribute to a fatal result. Occurring in pregnancy, it often leads to abortion or premature delivery, and hence is not without danger.

Relapses are likely to occur after intervals varying between a few months and two or three years. As a rule, the relapses are shorter than the primary attacks.

A number of remedies has been advocated as effecting a cure of this disease. In general, when different methods of TREATMENT of any disease are found to be successful, it is a fair inference that the disease ends favorably from an intrinsic tendency. This is undoubtedly true of chorea. Of the diverse remedies indicated, none can be relied upon for promptly arresting the course of the disease, and it is doubtful if any exert a special curative agency. It by no means follows, however, that much benefit may not be derived from judicious treatment. The greater part of the remedies are doubtless more or less useful, and the physician is to exercise judgment in adapting them severally to individual cases, and in employing them successively in the same case.

Rational indications, in the first place, are to be sought for in coexisting disorders or morbid conditions. If there be symptoms, aside from the chorea, pointing to rheumatism, remedies addressed to this condition are called for. Anæmia in a large proportion of cases calls for appropriate treatment. Constipation, if present, claims attention. Purgative remedies are among those advocated as efficacious in curing the disease. Causes of peripheral irritation, especially those of sexual origin, should be removed.

Of remedies, other than purgatives, which experience has shown to be useful, the more important are—arsenic, the carbonate and prussiate of iron, zinc, strychnia, opium, cannabis indica, and various antispasmodics. Of the efficacy of arsenic there is abundant and strong testimony. Fowler's solution is the most eligible preparation. Beginning with three or four drops three times daily, the doses should be gradually increased until the characteristic effects are observed. Of the preparations of iron, the carbonate and prussiate have been supposed to exert a curative effect, aside from their usefulness with reference to coexisting anæmia. Of the preparations of zinc, the oxide has been especially advocated as curative in this affection. Strychnia was advocated by Trousseau, who preferred the sulphate dissolved in syrup. He began the treatment with a very small dose—namely, one-twenty-fifth of a grain once daily—and increased the dose very gradually. Opium was also advocated by Trousseau as a curative remedy. He gave it in a full dose, producing slight narcotism, which is to be maintained for several days. He affirmed that this method of treatment is often extraordinarily successful.

To the efficacy of cannabis indica testimony is borne by many. Cimicifuga in some parts of this country has been a popular remedy, and its value is attested by many eminent physicians. Conium pushed to its physiological action has been employed with benefit. Of antispasmodic remedies, those which have been found especially useful are hyoscyamin, valerian, asafœtida, and camphor. Keeping the patient daily under the influence of chloroform for half an hour or more has been found serviceable. This measure is hardly to be advised save in cases in which the movements are unusually violent and constant. In chorea major it may be necessary to anæsthetize the patient in order to introduce the stomach-pump for purposes of alimentation. The application of ether spray to the spine, for four or five minutes at a time daily or on every alternate day, has been found efficacious. The testimony respecting the usefulness of electricity is conflicting. Benedikt and Jaccoud claim signal success from galvanization of the spine. Beard and Rockwell have found general faradization uniformly successful. The result of galvanization of the spine in a few cases was unsatisfactory in their hands, and also in the experience of Meyer. This discrepancy may be perhaps in a measure explained by differences as regards the mode of employing both the direct and induced current, and also by differences as regards the period of the disease when the treatment was entered on, bearing in mind that this disease may be said to have a self-limited duration. The bromides, in my experience, have failed to meet the expectations which I had been led to form of their usefulness in this affection.

Hygienic measures are certainly not less important than medicinal remedies. Alimentation as nutritious as possible is desirable. Daily exposure and, if practicable, exercise in the open air are highly important. The cold sponge-bath is useful, provided it be not dreaded by the patient and it be followed by brisk reaction ; and the shower-bath, with the same provisions, is admissible. On the other hand, the tepid bath as a palliative measure is highly useful. In the Children's Hospital in Paris for many years much reliance has been placed on gymnastic exercises for the cure of chorea. Of course these are practicable only in the cases in which the ability to co-ordinate movements by the will, although more or less impaired, is not lost. The exercises consist of systematic movements performed under the direction of assistants and enlivened by music.[1] After the choreic movements have ceased, more or less inability to co-ordinate movements, together with muscular weakness, remains, and these effects of the disease are to be removed by exercise. The power of again directing the muscles is to be acquired as in infancy, but it is acquired rapidly.

Anomalous Muscular Movements—Local Spasms—Tremor.

Certain abnormal muscular movements are considered, incorrectly, as denoting varieties of chorea. Persons sometimes without motive or purpose, impelled apparently by an irresistible impulse, perform extraordinary acts. One of these is leaping suddenly and violently, and sometimes jumping upon chairs or tables. Such movements have been considered as denoting a variety called *chorea saltatoria.* A patient subject to epilepsy was brought to me for examination, a young man aged about twenty, who at variable intervals had paroxysms of jumping vigorously for a few seconds. He declared he was unable to avoid these movements. In other cases there is a propensity to run, and the patient unexpectedly, when walking, rushes forward at a rapid pace for several minutes. These movements have been considered as denoting another variety, called *chorea festinans vel procursiva.* Rotating and vibrating movements are performed in other cases (*chorea oscillatoria*). Moving the head and body alternately backward and forward, constituting what has been called *salaam convulsions*, belongs in the same category. In a case which came under my observation the patient, a young married woman, had for several months during her waking hours constantly pounded some object before her with both hands. In another case a young unmarried woman had frequently-recurring paroxysms in which for several hours she rotated the head rapidly, and also at times she performed rapid projectile movements of the tongue. This patient for three years maintained the position of emprosthotonos in sitting, walking, or standing, always ceasing to maintain it when recumbent.

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[1] For details concerning gymnastic exercises, vide papers by M. Sée and by M. Blache in *Mémoires de l'Acad. de Médecine,* Paris, 1855. For a summary of these details, vide Meigs on *Diseases of Children.*

These movements, although abnormal, proceed from the action of the will. They denote a species of mental aberration. They are manifestations of a delirious volition, resembling in this respect certain of the convulsive movements in hysteria. They are sometimes connected with cerebral disease, but in most cases they are purely functional. In some cases it is undoubtedly true that patients are unable to resist the impulse which impels to the abnormal acts, but a morbid propensity to excite wonder and interest doubtless enters into the rationale in some cases.

The treatment embraces remedies and hygienic measures to invigorate the nervous system, together with efforts to overcome the propensity which gives rise to the abnormal movements.

Allied to the cases just referred to are those in which twitching of certain parts becomes habitual. Thus some persons acquire the habit of jerking the head, raising the shoulders, or making other motions, especially under mental excitement. In many, if not most, of these cases the habit may be overcome by persevering efforts before it becomes confirmed. Convulsive movements of certain of the muscles of the face, producing sometimes notable grimaces, are involuntary and closely allied to chorea. These often become confirmed and irremediable. Spasm of the eyelids is called blepharospasmus. When the angles of the mouth are raised the peculiar expression is significantly styled risus caninus, or the sardonic grin. The convulsive movements or spasms of the muscles of the face, called by Romberg mimic or histrionic, may be either clonic or tonic. They are oftener the former. The involuntary grimaces which are sometimes produced occasion much mortification. The clonic spasms come in paroxysms which generally last but an instant. They are especially liable to occur under mental excitement. Sometimes they interfere with mastication and articulation. They are almost always unilateral. Involuntary or spasmodic movements of the head, with or without grimaces, characterize some cases. In a case at Bellevue Hospital there was violent jerking of the head from side to side at intervals of a few seconds, and with these there were rapid lateral oscillations of the eyeballs. Aside from these symptoms there was no evidence of cerebral disease. They followed a debauch. The patient soon left the hospital, the movements continuing. In another case the head was moved spasmodically to the left side at short intervals, with some contraction of the facial muscles on that side. Notable improvement in this case took place under the use of the bromide of potassium. Pressure over the branches of the trifacial nerve and at certain points on the cervical portion of the spinal column sometimes has an inhibitory effect upon facial spasms. These are known as "pressure-points." They may be found in local spasms situated elsewhere, and the galvanic current applied at these points not only has the same inhibitory effect as pressure, but it may prove curative. These pressure-points should be sought after in all cases of local spasm. Pressure at certain points is found in some cases to increase the spasms instead of having an inhibitory effect. Hypodermic injections of morphia are sometimes an effectual method of treatment if the spasmodic affection have not been of long duration. The injections should be made at the "pressure-points." Facial spasms often continue, and become permanent in spite of treatment. Electricity and counter-irritation are chiefly to be relied upon, and should be faithfully tried.

An affection characterized by inability to co-ordinate the muscles engaged in the act of writing has been called *chorea scriptorum*, or *writers' cramp* (graphospasmus). Whenever the act of writing is attempted, the flexor muscles especially, but sometimes the extensors of the thumb and fingers, either become rigidly contracted or contract irregularly, and guidance of the pen is impossible. Persons who employ certain muscles constantly in other acts are sometimes similarly affected. Thus, pianists and violinists are no longer able to play, printers cannot compose, tailors and seamstresses lose the power of guiding the needle, and turners are unable to move the lathe with the foot. In all these instances other movements, calling into exercise the same muscles, are performed without difficulty. In the treatment of writers' cramp and allied affections discontinuance of acts which occasion the spasm is essential. The period of rest should extend over several months. If this be resorted to early, recovery may be hoped for. Galvanic electricity is useful. If discontinuance of writing cannot be enforced, as in cases in which the livelihood of the patient depends on writing, the habit should be acquired of using the pen with the left hand. This is sometimes only a temporary resource, as the affection is liable to occur in the left hand after some use. Changing the size of the pen and adopting contrivances to change the action of muscles which are co-ordinated in writing will sometimes enable writers to continue their occupation. The affection consists in impairment of the power of co-ordination for the performance of certain acts, and it is a consequence of the long-continued performance of these acts. It is a fair inference from these facts that it is a central, not a peripheral, affection. If timely and complete rest be not resorted to, recovery is not to be expected.

Painful contraction of muscles, irrespective of affections of which this is a symptom, such as tetanus, epidemic cholera, etc., and not produced by any obvious cause, is known as *cramp*. Some persons are liable to suffer much from cramp of the muscles of the leg or feet, occurring especially at night. A method of treatment proposed by Bardsley of Manchester, England, is said to be generally successful. This consists of sleeping on an inclined plane, the bed being twelve inches higher at the head than at the foot.

Under the name *essential or idiopathic spasm of the muscles of the extremities* (*contracture essentielle ou idiopathique des extrémités*) French writers describe an affection characterized by tonic contraction occurring with either intermissions or marked exacerbations. This affection has also received the name of intermittent tetany or tetanilla. It occurs especially in young subjects between the age of seventeen and twenty-five and in children between one year and three years of age. Females more than males are subject to it. The puerperal state and lactation predispose to it. It is observed as a sequel of certain diseases, such as the continued or eruptive fevers, articular rheumatism, and chorea. In children it appears to be connected with dentition, intestinal worms, and gastric disorders. The spasm or cramp affects first the muscles of the upper extremities, especially the flexors of the forearm. It extends generally to the lower extremities, and here the extensors are the muscles most liable to be affected. The hand is often firmly closed, the fingers concealing the thumb, which is in close apposition to the palm, and the flexion may embrace both the phalanges and the metacarpal bones; or, more rarely, the fingers are separated and partially flexed, so as to give rise to the claw-like appearance. Sometimes the position of the fingers is like that in the act of writing. Exceptionally, the extensors of the wrist are affected. The contractions are powerful. Generally they are bilateral, but they are sometimes confined to one side. In some cases the spasm extends to the muscles of the arm, shoulder, thigh, trunk, and neck, together with those of the jaws. In these cases the affection simulates tetanus. It has been called intermittent tetanus. In tetanus the trismus is primary, whereas in this affection it is secondary. Moreover, in this affection the associated symptoms denoting gravity of disease are wanting. The intermissions sometimes observe the laws of periodicity governing the different types of intermittent fever. The affection belongs among the neuroses; that is, it is a functional affection, the duration varying generally between one week and three weeks. Relapses often occur. The spasm sometimes leads to paralysis of the muscles which are affected. The affection tends to a favorable termination. Cases, however, have been observed in which a fatal result has been due to paralysis following spasm of the muscles concerned in respiration and to spasm of the glottis. The indications for treatment relate to the pathological conditions with which it is associated. Quinia in full doses is indicated if the affection be intermittent. Belladonna, opium, or the hypodermic injection of morphia may be resorted to for palliation of the pain and spasm.[1]

A peculiar affection of the voluntary muscles was first described by Thomsen in 1876 as occurring in a number of members of his own family. The affection is sometimes called *Thomsen's disease.* It has received from Strümpell the name *myotonia congenita.* It is a rare affection, but a number of cases have been reported. The affection appears to be congenital, existing throughout life. It affects frequently several members of a family, and apparently with preference the male members. The characteristic of Thomsen's disease is a peculiar stiffness and tension of the voluntary muscles, amounting to a continued tetanic spasm. This tension is most evident when the patient begins to use the muscles after a period of repose. It lessens and may disappear after the muscles have been for some time in use. The muscular stiffness is increased by psychical excitement. Rapid muscular movements involving precision may be impossible. In most of the cases hitherto observed the muscular development has been excessive. Upon electric excitation the spasm of the muscles is longer than in health, continuing after the circuit is opened. This indicates some congenital anomaly in the muscular structure. Sensibility is intact, and there are no noteworthy symptoms other than those mentioned. No benefit has hitherto been derived from any therapeutic measures.

Tremor—that is, alternate contraction and relaxation of muscles in rapid succession—is a symptom of certain lesions of the nervous centres, especially of multiple or disseminated sclerosis of the brain and spinal cord. It is not infrequently incident to advanced age, and it is then observed especially in the upper extremities, but in some cases constant lateral motions of the head take place during waking hours, and they do not always cease during sleep. The *senile trembling*, as it is called, although not dependent on any known lesions, is irremediable. Tremulousness, especially of the hands, occurs in early and middle life as a result of the abuse of alcohol, the habitual use of opium, nicotism, sexual excesses, and other causes which tend to depress or disorder the nervous system. Under these circumstances it may be purely functional. The trembling sometimes extends to the lower extremities and to the trunk.

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[1] Vide *Traité de Pathologie interne*, par Jaccoud.

The treatment in cases of tremor from these several causes of course involves, as the first and most important measure, the withdrawal of the causes. Other measures indicated are those which have reference to associated disorders and the general condition of the system.

The affection known as *paralysis agitans*, sometimes called Parkinson's disease from an English author who described the affection in 1817 under the name shaking palsy, belongs in the group of affections characterized by tremor rather than among the paralytic affections. It is to be included with the neuroses, inasmuch as it has no ascertained anatomical characters. Diverse morbid changes have been found after death in different cases, but in some cases no changes have been discovered.

It is an affection of middle or advanced life, the age being very rarely less than forty years. Men much oftener than women are affected. These are all the facts at present known respecting the etiology.

The tremor consists of feeble, rapidly-recurring, rhythmical movements, at times, under mental excitement or physical exertion, much increased in range and force. The movements generally begin in the fingers and hands, they extend gradually over the upper extremities, and after variable periods the affection invades the lower limbs, the facial muscles, the muscles of articulation, those of the head, and sometimes the trunk. They are in some instances for a long time or permanently unilateral. The muscles which are involved retain their normal electrical excitability. Paresis follows the tremor, the latter preceding the former for a considerable or long period. Stiffness of certain muscles, especially of the flexors, is gradually induced. The head and the trunk incline forward; the elbows stand out from the chest; the forearm and hands are moderately flexed; the thumb and forefinger are sometimes in the position of holding a pen; and the fingers may be distorted as in cases of chronic rheumatism. The lower limbs are often correspondingly affected. They are knock-kneed, the feet have the appearance of talipes equinus, and there is virtually incomplete paraplegia. Festination, or a tendency to run forward, is sometimes observed. The tremor, as a rule, either ceases or is much diminished during sleep.

This affection is to be distinguished from tremor incident to cerebro-spinal sclerosis. The differential points are as follows: In sclerosis, paralysis is either coincident with or antecedent to the tremor; the tremor is excited by volition; it begins in the lower limbs; and is not very infrequent at less than forty years of age. *Per contra*, in paralysis agitans, paralysis does not precede the tremor or accompany it at an early period; the tremor is not dependent on volition; it begins in the upper limbs; and persons less than forty years of age are exempt from this affection.

The independence of volition distinguishes the tremor from chorea. The muscular movements are shorter in paralysis agitans than in chorea. The greater degree of tremor, and often the age, serve to distinguish this affection from senile trembling. The tremulousness from alcoholism and other causes which are readily ascertained in individual cases is excluded without difficulty.

As regards the pathology, it is to be said, as of other functional affections, that the existence of morbid changes is logically certain, but their nature and their seat are yet to be ascertained. The affection is probably not myopathic or peripheral, but whether the morbid condition be seated in the brain or spinal cord, or in both, is an unsettled question.

It is also an unsettled question whether recovery ever takes place. If the few reported instances be not open to distrust, it is certain that the probability of recovery is so small as to be almost nil in any individual case. The affection, however, is not incompatible with the duration of life and fair general health for an indefinite period. Patients generally die from intercurrent affections. The affection proves fatal, *per se*, by inducing general paralysis after many years' duration, death taking place by exhaustion.

The objects of TREATMENT are the prevention of the continued progress of the affection and more or less amelioration as regards the tremor. Various remedies in the hands of different observers have appeared to be not without considerable effect in certain cases. The objects of the treatment are of sufficient importance to render the trial of these various remedies a duty, without abandoning a case as offering no encouragement in the way of improvement. Elliotson claimed to have effected a cure by the carbonate of iron; Brown-Séquard, by the chloride of barium; and Reynolds and Remak, by galvanization of the spine. Arsenic, administered hypodermically, has been found highly useful by Eulenburg. Charcot and others have given hyoscyamus with benefit. Strychnia was advocated by Trousseau. The iodide of potassium in large doses proved beneficial in a case observed by Villemin. Other remedies are—opium, belladonna, ergot, the Calabar bean, curare, chloral hydrate, and the bromides. None of these can be expected to have more than a palliative effect. Measures to improve and invigorate the general health are perhaps more likely to be of real benefit. Among these, the sponge-bath and the cold pack are to be included. Bandaging the limbs and applying to them the tincture of aconite, small doses of strychnia being also given, diminished the tremor considerably in a case under my observation.

Mercurial tremor denotes trembling movements of the limbs, the muscles of the face, the tongue, and sometimes the trunk, due to poisoning by mercury. The tremor occurs especially when the patient makes voluntary movements, in this respect being analogous to chorea. This affection, formerly not infrequent, of late years is rarely seen, in consequence of improvements in mechanical processes involving the use of mercury and the better observance of precautionary measures. The inhalation of vaporized mercury is most likely to give rise to it, but it has been produced by the use of mercury as a medicine and by mercurial inunctions. In the only case which has fallen under my observation the gums were swollen, reddened, and spongy, and the teeth had been loosened. These appearances, together with other symptoms of the mercurial cachexia, and generally knowledge of the fact of exposure to mercurial poisoning as incident to the occupation of the patient, will lead to the diagnosis.

Recovery may be expected under the use of tonic remedies with an invigorating regimen, provided the continued introduction of mercury into the system be prevented. The iodide of potassium is supposed to effect the elimination of mercury from the body.

A variety of spasmodic movements was described by Hammond in 1871, and designated *athetosis* (ἀθετος, "without fixed position"). It is characterized by persistent irregular movements of the fingers and toes and inability to retain them in any fixed position. The movements are wholly independent of volition. The patient can rarely arrest them by an act of the will, even for an instant. In most instances the affection is confined to one side (hemiathetosis), but in some instances both sides are affected (bilateral athetosis).

It may affect either the fingers or the toes separately, or both conjointly. In rare instances spasmodic movements of either the face, neck, or forearm are associated. The movements of the fingers and toes are constant during waking hours, and sometimes continue, although diminished, during sleep. If they cease during sleep, the fingers rest in abnormal relative positions. The abnormal movements resemble those of chorea, but differ from them by their slower rhythm and greater regularity. They are also distinguished from choreic movements by affecting chiefly or exclusively the fingers and toes. In many cases more or less anæsthesia was present on the affected sides. The muscles of the forearm involved in the movements may become hypertrophied.

In a large proportion of cases hemiathetosis is a sequel of hemiplegia. The movements of the fingers and toes are on the paralyzed side. Some writers consider that lesions involving the posterior third of the posterior division of the internal capsule are especially liable to be followed by athetosis. Others think that the lesion is to be sought for in the motor cortical tract of the brain. Further observations are needed to determine the pathology of this affection. Athetosis may occur as a primary affection without paralysis. Although bilateral athetosis is not associated with well-marked paralysis on both sides, there is often general muscular weakness. The affection in this form occurs in idiots or those who are congenitally feeble-minded. It doubtless proceeds from a cerebral affection, although it has been observed in connection with locomotor ataxia.

Experience has furnished few data for a favorable prognosis. Gnauck has reported a cure effected by the constant electrical current and the use of bromide of potassium. Gowers and Oultmont have found this agent essentially beneficial.[1]

The anomalous movements described by S. Weir Mitchell under the name "post-paralytic chorea,"[2] and by Charcot under the name "post-hemiplegic hemichorea,"[3] are analogous to those of athetosis. The terms athetosis and post-hemiplegic chorea have been used to describe the same affection. As already remarked, athetosis may occur independently of hemiplegia. Charcot places the lesion of post-hemiplegic chorea in the posterior part of the internal capsule and the surrounding brain-substance. The choreic movements may, however, follow lesions of other parts of the brain.

Epilepsy.

Epilepsy is a chronic paroxysmal affection. The paroxysms are characterized by loss of consciousness, and in the most characteristic cases by convulsive movements extending over more or less of the voluntary muscular system. Convulsions analogous to those of epilepsy are symptomatic of different cerebral affections, of uræmia, of injuries of the head, and they may be caused by certain toxical agents; for example, strychnia. These are to be distinguished as epileptiform convulsions. Epilepsy, as one of the neuroses, is a functional affection; that is, it is not necessarily connected with any appreciable lesions. Related to the affection, sometimes preceding its development, and sometimes occurring in those who are epileptics, are attacks in which convulsive movements are slight and sometimes wanting. Epileptic attacks in which convulsions are slight or wanting constitute that form of the disease which is called mild epilepsy, or *epilepsia mitior*, also by French writers *le petit mal* and *epileptic vertigo.* They may be distinguished as *epileptoid attacks.* In distinction from epilepsia mitior, the common form with general convulsions is called *epilepsia gravior*, or *le grand mal.* This form is to be first considered.

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[1] Vide *Real-Encyclopädie*, Wien und Leipzig, 1880, art. "Athetose."

[2] *Am. Journ. of Med. Sciences*, Oct., 1874.

[3] *Leçons sur les Maladies du Système nerveux*, Paris, 1877.

In the majority of cases a paroxysm occurs without premonition. The loss of consciousness is as sudden as if produced by a stunning blow upon the head. The person falls, and hence the affection has been popularly known as the "falling sickness." In a minority of cases there is a brief warning of the approaching fit. Generally the premonition consists in a sensation which the patient is unable to describe. The sense of a "cold vapor" emanating from some part of the body and mounting to the head has been called the *aura epileptica.* This sensation was first described by Galen, and, as remarked by Herpin, "the commentators of Galen and those who copied from them handed down the phrase, until the existence of an aura seemed to be accepted as an ascertained fact." It is a traditional error to consider such a sensation as a frequent warning of an epileptic paroxysm, and it is certainly extremely rare for the patient to experience any sensation emanating from a particular part of the body. The onset of the paroxysm is often marked by a loud, short, and piercing shriek or cry, which from its intensity and unnatural character is sometimes truly terrific. The face at the time of the seizure becomes notably pale. The direction in which the fall takes place is almost always forward on the face. The face is not infrequently wounded or bruised by the force of the fall, and severe burns are sometimes caused by falling upon heated stoves or into the fire. The convulsive movements at once begin. These are at first tonic; that is, the muscles are forcibly contracted, and the contraction persists for several seconds. The muscles of the face, neck, arms, forearms, the lower extremities, the abdomen, and chest are involved, and have a tetanic rigidity. In most cases the muscles of one side of the body are more affected than those of the other side, and sometimes, although very rarely, the convulsions are limited to one side. The tonic convulsions last for a period varying between a few seconds and half a minute or somewhat longer. The convulsions then become clonic; that is, the muscles present forcible contractions and relaxations in rapid succession. The face is hideously distorted, the head is moved upon the trunk, the upper and lower limbs and the body jerk with violence. The tongue is sometimes caught between the teeth and is wounded by the tonic contraction of the muscles of the lower jaw. This more frequently happens during the clonic convulsions. Respiration is nearly suspended by the tonic convulsions and is irregular and difficult during the clonic. A quackling noise accompanies the respiratory acts. The face during the convulsions becomes deeply congested and livid. Convulsive masticatory movements are accompanied by the ejection of foamy saliva, frequently commingled with blood from the wounded tongue or from a portion of the cheek caught between the teeth. The violent clonic convulsions continue for a period varying between half a minute and three minutes. The period to the observer seems to be much longer, but if noted by the watch it is rarely found to exceed three minutes, and generally it does not last more than a minute. The convulsive movements now become less rapid and violent, the embarrassment of respiration diminishes, the patient fetches a deep sigh, and the paroxysm, so far as the convulsions are concerned, is ended. The paroxysms in different cases, and different paroxysms in the same case, differ much as regards the violence of the convulsive movements and the extent of the muscular system involved. The convulsions are sometimes specially marked in the face and upper extremities, and they may be limited to these parts. Paroxysms differ also as regards duration. The violence of the convulsive movements of the muscles of the shoulder is sometimes sufficient to produce luxation of the humerus. A case in which this repeatedly occurred has fallen under my observation. During the convulsions the urine and feces are sometimes expelled, and an emission of semen may take place. The pupils during the paroxysm are dilated, and they do not contract on exposure to a bright light. The eyelids, however, close when the conjunctiva is touched, showing the preservation of the reflex function.

After the convulsions have ceased consciousness may be speedily regained, and the patient may only experience a sense of fatigue, with usually a disposition to sleep; but in most cases the state of coma continues for a variable period. Frequently the respiration for some time is stertorous and the lips are puffed out with expiration. The patient remains in this state for a period varying between a few minutes and half an hour; the pupils are still dilated and do not contract on exposure to light, and he cannot be roused from the comatose state. Gradually he emerges from this state, opens his eyes, appears confused, and some time elapses before he is able to appreciate his situation and reply to questions. If he attempt at once to walk he reels like a person intoxicated. Occasionally the patient passes from the apoplectiform state into one of delirium. He talks incoherently, manifests hallucinations, and sometimes the delirium assumes the form of mania, during which he may be dangerous to himself or others. After recovery of the mental faculties there is complete inability to recall anything which has occurred during the paroxysm.

The paroxysms recur after intervals which are extremely variable in different cases. The intervals may extend over many months or years. On the other hand, they recur in some cases daily and even many times daily. When a paroxysm has once occurred, other paroxysms are almost sure to follow sooner or later. In most cases the paroxysms at first are more widely separated than after they have repeatedly recurred; and they become, as a rule, more and more frequent, until the affection is confirmed. In some cases the recurrence is periodical. In women sometimes paroxysms occur regularly at the menstrual period. Periodicity, however, is not the rule, the intervals generally varying considerably in the same case; but in certain cases of confirmed epilepsy about the same number of paroxysms take place in successive periods of a quarter, a half, or the whole of a year. Exceptionally, the recurrence of the paroxysms becomes less frequent after a time, and a patient who for several years has been subject to paroxysms after short intervals may afterward have long intermissions. Occasionally, having recurred more or less frequently for months or years, they cease altogether for a considerable period, and then return with not less, and perhaps greater, frequency than before.

In some cases paroxysms occur in rapid succession during a day or during several successive days. This condition constitutes the so-called *status epilepticus*. As many as one hundred and even a larger number of paroxysms have been known to occur during twenty-four hours. In a case under my observation paroxysms took place at intervals of a few moments for fourteen consecutive days. The patient recovered, and the paroxysms did not again recur for several months. When a series of paroxysms thus occurs, the mind is notably impaired for a time. The patient is childish and perhaps idiotic for several days, but gradually recovers the habitual state of the mental faculties. Hemiplegia occasionally follows a series of paroxysms. This occurred in the case just referred to. The paralysis in such cases is usually of brief duration.

Epileptic paroxysms occur at all hours of the day or night. They are likely to occur during the night. In some cases for a greater or less period they occur only at night. In these cases the affection may have existed long before the fact is ascertained. I have known a case in which, the paroxysms never occurring in the daytime, the existence of the affection was for a long period concealed from the patient. It is a curious fact that the paroxysms are usually suspended during the course of an acute disease. A patient, for example, subject to daily paroxysms became affected with typhoid fever. During the career of the fever no paroxysms occurred, and among the events denoting convalescence was the return of the epilepsy.

It was for a long time believed that the morbid condition giving rise to epileptic paroxysms was seated in the medulla oblongata and pons Varolii. This view still has its advocates, but there is an increasing tendency to refer the origin of epileptic paroxysms to the motor area of the cerebral cortex, and it must be admitted that there are weighty arguments in favor of the latter hypothesis. The fact that loss of consciousness is a constant factor in the epileptic paroxysm, and that it may occur without convulsions, favors the cortical theory of epilepsy. It has been shown experimentally that irritation of the motor cortical area causes paroxysms in no way distinguishable from epilepsy in human beings. In experimental epilepsy, as well as in many cases of human epilepsy, it has been noted that the convulsions proceed from one group of muscles to another in an order corresponding to the relative situation of the motor centres in the cortex. Thus, if the spasm begin in the face, the arm is next affected, and then the lower extremity. Extirpation of a motor centre in the cortex prevents the development of epileptic convulsions in the muscles whose cortical centre is destroyed. Injury or disease of the motor cortex is very frequently followed by typical epileptic paroxysms. In incomplete epilepsy or petit mal the manifestations are for the most part psychical. The facts mentioned give great plausibility to the cortical theory of epilepsy.

As regards the immediate cause of the epileptic paroxysm we possess no positive knowledge. Epileptiform and true epileptic paroxysms may be caused by various intracranial lesions, such as tumors, syphilitic affections, meningitis, and fracture of the skull. With the exception of these cases, epilepsy is to be regarded as a neurosis without any known anatomical basis. Except in symptomatic epilepsy, post-mortem examination has failed to reveal any lesion to which the epilepsy could reasonably be attributed.

It is very generally believed that the immediately exciting cause of an epileptic paroxysm is cerebral anæmia resulting from vaso-motor spasm. This view is rendered probable by the known effects of sudden cerebral anæmia from hemorrhage, from ligation or compression of the arteries carrying blood to the brain, by the notable pallor of the face at the beginning of an attack of epilepsy, together with the appearance of the fundus of the eye as observed by means of the ophthalmoscope. While the efficacy of cerebral anæmia as an exciting cause of the epileptic paroxysm cannot be doubted, it is not certain that it is operative in all cases of epilepsy. The experiments of Brown-Séquard and others on animals show a pathological connection between peripheral irritations and epileptiform convulsions. Such a connection exists in some, but not by any means in all, clinical cases.

In a large proportion of the cases of epilepsy no sources of centric or of excentric irritation are apparent. That under these circumstances the epileptic paroxysms are due to the action of an internal and at present unknown toxical agent seems to me the most rational hypothesis. Epilepsy, according to this hypothesis, is a toxæmia analogous to uræmia, the toxical agent being produced at variable intervals, the quantity and the continuance of its production not being sufficient to endanger life; and in this respect the contrast with uræmia being striking. If this pathological view be correct, knowledge

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of the nature and source of the toxical agent, which may perhaps be acquired, will, as we may hope, render this disease controllable.

Epilepsy is classed among the hereditary diseases. It has, however, been customary to consider the existence of other nervous affections in parents or progenitors as evidence of an inherited predisposition. Heredity, limited strictly to this disease, does not often enter into the causation. While the possibility of the transmission of the disease to offspring should not be ignored, it is to be borne in mind, as a fact which may be of inexpressible comfort to many persons, that an epileptic parentage by no means entails this or any other disease on descendants either proximate or remote. It would be easy to collect instances of families in which the husband or wife were subject to epilepsy, whose children and grandchildren were free from the disease and in all respects healthy.

Age is of considerable weight in the causation. In a small proportion of cases the disease either exists from birth or is developed in infancy. In nearly one-third of the cases it dates from two to ten years of age, and in a somewhat larger proportion from ten to twenty years. The number of cases in which the development is between twenty and thirty years is much less, and at a later period of life the number is comparatively small. In the great majority of cases at the beginning of the disease the age is between two and twenty years.

Of particular causes little is positively known. It is certain that intemperance as regards the use of alcohol may give rise to it. Epileptic paroxysms occur not infrequently in drunkards, and cease to recur in cases in which reformation of intemperate habits is effected. It is one of the occasional effects of lead-poisoning. Venereal excesses, and especially habits of masturbation, have been supposed to be not infrequent causes. The supposition is highly probable, but an accumulation of facts establishing this causative connection is desirable. A female patient, twenty-three years of age, unmarried, who had been subject to paroxysms occurring almost nightly for two years, stated to me, voluntarily, that she was taught to masturbate at six years of age, and continued the practice almost daily until she was fourteen. At this age she became convinced of the sinfulness of the habit, and discontinued it. She was afterward tormented with sexual desires, and experienced the venereal orgasm almost nightly during sleep. She was led to disclose this private history by a conviction, at which she had arrived of her own accord, that it was important with reference to the source of the epilepsy. To what extent the antecedent history in cases of epilepsy would furnish corresponding facts can only be conjectured. The number of instances in which congenital phimosis exists in epileptics, with cessation of the paroxysms after removal of the prepuce, show a causative connection. Epilepsy has been known to occur in persons affected with tænia, and to cease after the expulsion of the worm.

In persons subject to epilepsy the paroxysms in the great majority of cases occur without obvious exciting causes. The causes determining the attack, as a rule, are intrinsic. Persons not infrequently are able to judge, by experience, from certain indefinite sensations that they will ere long have a paroxysm. The sensations disappear after the paroxysm has occurred, and it is not uncommon for persons to feel better after recovery from the immediate effects of a paroxysm than for some time prior to its occurrence. It has been supposed that the aura which sometimes precedes the paroxysm denotes the existence of an exciting cause emanating from the part to which the aura is referred. It is more correct to regard the aura as a peripheral sensation dependent upon some central irritation. A case has fallen under my observation in which the patient, a female, experienced the first paroxysm during the first act of sexual congress after marriage. Subsequently, a paroxysm occurred at each marital connection, but after a time paroxysms took place without this exciting cause, and the patient became a confirmed epileptic. It is a remarkable fact that an attack may occur from imitation. An instance of this has fallen under my observation. A woman subject to epilepsy had a series of paroxysms in quick succession. Her female attendant while these paroxysms were in progress first manifested hysterical phenomena, and finally she experienced a distinct epileptic paroxysm. She had never before had an attack of epilepsy, nor did another paroxysm take place, the person remaining within my observation for several years afterward. It is well known that dogs, cats, and other domestic animals are liable to epilepsy. Prof. Dalton has related to me the following interesting illustration of the effect of imitation in the canine race: A dog not previously affected with epilepsy was in company with another dog which was subject to this affection. The latter being seized with an epileptic paroxysm, the former immediately afterward had a similar attack. This occurred under Prof. Dalton's observation.

The DIAGNOSIS of epilepsy in its ordinary form rarely offers much difficulty. The disease is, in general, readily ascertained when it has become confirmed. In cases in which the practitioner has not an opportunity of witnessing any of the paroxysms, he may generally obtain sufficient information respecting the distinctive features to feel sure of the diagnosis. The prominent points of inquiry are—the sudden seizure, the cry at the beginning of the paroxysm, the loss of consciousness, the tonic and clonic convulsions, embarrassed respiration, ejection from the mouth of frothy saliva often mixed with blood, and the short duration of the fits. An examination of the tongue, if repeated paroxysms have occurred, in some cases shows cicatrices, resulting from the wounds of this organ, which are quite diagnostic.

The affection is to be discriminated from hysterical convulsions, but these present points of difference which are usually sufficiently marked. The convulsive movements are not purely automatic, as in epilepsy, but proceed from a delirious volition. They are of longer duration than the convulsive movements in a paroxysm of epilepsy, and they are developed gradually. Foaming at the mouth does not accompany hysterical convulsions, and the pupils are not dilated. Epilepsy, however, may be developed in persons subject to hysteria, and the two affections may be associated.

If called to a patient not known to be an epileptic, in the comatose state which succeeds the convulsions, apoplexy might at first be suspected; but if the convulsions which precede this state have been observed by any one, information respecting their occurrence suffices for the diagnosis. If the convulsions have not been observed, the appearance of foam and blood upon the lips is diagnostic. The age of the patient is to be considered, and the absence of hemiplegia, which often occurs in cases of apoplexy and rarely in cases of epilepsy.

Epileptiform convulsions, or eclampsia, occurring in children, in pregnant women, or in connection with Bright's disease, are to be discriminated by means of the antecedent and coexisting symptoms. Judging from the convulsions and associated nervous phenomena, uræmia and epilepsy might easily be confounded. Uræmic convulsions, however, are rarely of as brief duration as an attack of epilepsy. Examinations of the urine will suffice for this differential diagnosis; it is, however, to be borne in mind that directly after a paroxysm of epilepsy the urine contains a trace of albumen. A paroxysm of convulsions in an infant or child often with good reason occasions anxiety lest it may prove the beginning of epilepsy. It is not always practicable to determine this point at once. There is more reason to apprehend epilepsy in proportion as the convulsions are not otherwise to be accounted for. Their non-recurrence affords the only positive proof that they are not epileptic.

It is desirable to determine whether epileptic paroxysms be connected with lesions affecting the cerebro-spinal system, or whether they be idiopathic. With reference to this point, symptoms denoting lesions are to be sought for in the intervals between the paroxysms. Persisting pain in the head, paralysis, and disturbance of the intellect greater than the epileptic paroxysms will account for are symptoms pointing to cerebral lesions. Lesions are to be inferred if the epileptic paroxysm succeed an injury of the head.

Epilepsy is one of the affections sometimes simulated by malingerers. More or less of the appearances which are presented in epileptic paroxysms may be voluntarily produced, but a malingerer must be very well acquainted with the disease to represent accurately the phenomena in the order in which they succeed each other, and certain of the phenomena are beyond the power of the will. To determine a case of deception, let it be observed if the face become at first pale and afterward deeply congested and livid, if a cry be uttered, if the fall be forward on the face, if the convulsions be at first tonic and afterward clonic, if foamy saliva be ejected from the mouth, and if the tongue or cheek be wounded by the teeth. Let the pupils be examined, for these cannot be voluntarily dilated and their mobility on the approach of light cannot be prevented. Let the duration of the fit be noted, and the convulsions will be likely to be unduly prolonged. Let it be ascertained if, accompanying and succeeding the convulsions, there be insensibility to pricking, burning, or pinching the skin. Let the attention be directed to the time and place in which the paroxysms take place. Malingerers will not select the night or a situation in which they will not be observed. They are not likely to fall in a way or in a position to receive injury. The aspect of confusion and shame which is manifested on recovering consciousness after an attack of epilepsy is not easily feigned.

Epilepsy may continue for a long time before its existence is ascertained if the paroxysms occur only at night. The disease is to be suspected when patients complain of awakening from time to time with headache, lassitude, and a wounded tongue. Under these circumstances the diagnosis is to be settled by causing the patient to be watched during the night.

Owing to the terrible nature of the malady, and the apprehension felt by those in company with an epileptic lest a paroxysm may occur, persons are naturally sensitive with respect to themselves or their relatives being said to have epilepsy. For this reason there is sometimes a disposition to conceal the nature of the paroxysms even from the physician. To avoid the depressing effect upon the mind of the patient which a knowledge of the disease is likely to produce, it is sometimes judicious to refrain from calling it epilepsy. In the case of a lady who was under my observation for many years the nature of the disease was never communicated to her. The paroxysms, which were often frequent and severe, were always called nervous attacks, and she finally died of an intercurrent affection without knowing that she had been an epileptic. In this case ignorance of her disease enabled her to participate in many of the enjoyments of life from which, owing to her sensitive nature, she would have shrunk had she been aware that she was affected with epilepsy.

The PROGNOSIS in cases of epilepsy, as regards immediate danger from the paroxysms, is always favorable, and epilepsy does not give rise to any particular disease or diseases of a dangerous character. Epileptics, as a rule, are not short-lived, and in the majority of cases death arises from other diseases, to the development of which the epileptic paroxysms have not contributed. In another point of view, however, the prognosis is most unfavorable—namely, as regards the persistence of the malady. In the vast majority of cases the disease becomes confirmed, and as a rule the intrinsic tendency is to increasing frequency of the paroxysms.

In another point of view the prognosis is unfavorable—namely, as regards the mental condition. To a certain extent the disease tends intrinsically to impairment or disorder of the faculties of the mind. Many epileptics, especially if the paroxysms recur at short intervals, sooner or later fall into imbecility. To a greater or less extent this is due to causes other than the intrinsic tendency of the disease. If epilepsy occur in early life, the mental faculties are not developed by education and those occupations which involve their exercise, and in the cases in which the disease occurs at a later period the mental faculties progressively fail from disuse. The unhappy sufferer from this terrible disease is likely to lose interest in those objects or pursuits in life which incite to the exercise of the mental faculties. This explanation in part of the apparent effect of epilepsy on the mind has an important practical bearing.

Reference has been already made to mild attacks having an evident relationship to ordinary epilepsy, which may be called *epileptoid attacks.* It is important for the practitioner to recognize and appreciate the significance of these attacks. They are considered by French writers as belonging to a variety of epilepsy which they call *petit mal,* in contradistinction from the ordinary form, which they call the *grand* or *haut mal.* These attacks sometimes precede the development of ordinary epilepsy, and they sometimes occur intercurrently in persons who are confirmed epileptics.

The epileptoid attacks are multiform. A sudden loss of consciousness for a few seconds occurs in some cases, during which the body may remain fixed, the person apparently being lost in deep abstraction, the mental faculties resuming their operation as if nothing had taken place as soon as the attack passes off. In a case of confirmed epilepsy under my observation the patient at one time in the intervals between the paroxysms was subject to frequent attacks of the kind just described. During the attacks she remained fixed in the position in which she happened to be when they occurred. For example, if standing and arranging her hair, the hands were fixed precisely as they were at the instant of the seizure. On some days these attacks took place a great number of times, and the patient did not appear to be conscious of their occurrence. In the fixation of the body and limbs these attacks resemble catalepsy. In the case just referred to the only convulsive movements were of the muscles of mastication. Similar attacks are sometimes accompanied by convulsive movements of other of the facial muscles, of the muscles of the neck, and by slight laryngeal spasm. In some cases the patient falls, but recovers and rises in a few seconds. Loss of consciousness and falling in some cases are unattended by any convulsive movements. Trousseau relates examples of this kind. Of course in such cases epileptoid attacks are to be discriminated from syncope.

In other epileptoid attacks sudden delirium occurs, continues for a few seconds, and passes off, leaving the mind in the condition in which it was at the instant of the seizure. The delirium is manifested in different ways. Trousseau cites a case in which the patient uttered a burst of laughter, and when asked why he laughed he looked surprised, having no knowledge of what he had done. The same author cites examples of persons engaged in conversation, debate, or other occupations abruptly, as if struck by a sudden thought, going into the street bareheaded, and walking until they recovered themselves, when they were able to return and proceed with the matter in which they were engaged as if nothing had happened.[1] The following example came

[1] Vide *Clinique médicale,* tome ii.

which is essentially that of epilepsy. Hence the obvious importance of not overlooking their significance, in order to prevent, if possible, the fully-developed malady which is foreshadowed. Facts showing how frequently epileptoid attacks occur without being followed by the graver form of the disease are wanting, but it is certain that the latter by no means invariably follows. The physician, therefore, should not excite unduly the fears of the patient or friends by predicting that these minor forms are necessarily the precursors of the graver form.

The TREATMENT of epilepsy relates to the management of the paroxysms and to measures for effecting a cure. The latter are to be employed in the intervals. Curative treatment, as in other of the neuroses, may be conveniently considered as rational and empirical.

The rational treatment consists in deriving, as far as possible, indications from an investigation of each case with regard to the state of the system, coexisting disorders, and circumstances which may be suspected of standing in a causative relation to the malady. If the patient be of a full habit or plethoric, dietetic and other measures addressed to this state are rationally indicated. If, on the other hand, the patient be anæmic, measures of an opposite character are indicated. Disorders of the digestive system, uterine affections, and, in short, morbid disturbances anywhere in the economy, should receive appropriate treatment, as they may possibly be in some way concerned in the development or perpetuation of the malady. The presence or otherwise of tæniæ is to be ascertained. Inquiries with respect to syphilis are not to be forgotten. Phimosis calls for appropriate surgical treatment. Habits of life with respect to the probable or possible causation are to be scrutinized. Overtasking of mind or body, undue exposure on the one hand, and sedentary habits on the other hand, the facts with regard to mental and moral influences, the abuse of alcoholic stimulants, the immoderate use of tobacco, and especially over-indulgence in venery or the practice of masturbation, are to be inquired into. In brief, an important part of the rational treatment consists in enforcing observance in all respects of the laws of health. It is unnecessary to consider the many and varied details which this part of the treatment involves.

The empirical treatment consists in the employment of remedies or therapeutical measures which experience has shown to be sometimes curative. The number of these is large. It would require many pages simply to enumerate the drugs the curative efficacy of which, in certain cases, has been attested by honest and competent observers. The list is so extensive, the testimony with respect to particular remedies is so conflicting, and the instances of incurable epilepsy are so many, that practitioners are likely to enter upon the treatment of a case without much expectation of success. For the reasons just stated the disease is not infrequently allowed to continue without persevering efforts to effect a cure; and this fact may in a measure account for the number of cases in which patients remain hopelessly epileptics. Of the host of remedies which have been employed, many have doubtless proved successful in a certain proportion of cases; but without any disposition to deceive either themselves or others practitioners have often been led to over-estimate the success of different remedies by not making allowance for the following facts: An active remedy will frequently prevent recurrence of the paroxysms for a considerable period. Under the use of a certain remedy a patient passes weeks or months without a paroxysm; he is ready to believe himself cured, and the physician is happy to participate in the belief. Now, sooner or later, the paroxysms in most cases return, and the patient despairs of benefit from treatment or consults another physician.

The return of the malady may not be known to the physician, who had supposed that a cure was effected, and in the mean time he may have reported the case as one cured by the remedy employed. Again, the paroxysms cease in some cases to recur for weeks or months or years without any treatment. I have notes of a case in which paroxysms more or less in number every month, and sometimes fifteen or twenty in twenty-four hours, had occurred for sixteen years, a great variety of remedies having been tried without avail; and at length, after all treatment had been discontinued, the paroxysms ceased to recur for ten or twelve years, when they again returned, but at long intervals. Making, however, due allowance for the temporary interruption of the malady either by the treatment or spontaneously, there remain a certain number of undoubted cures effected by different remedies. In view of this fact, cases should not be abandoned without the faithful trial of many remedies. Of the various remedies which have been considered as curative, those within late years and at the present time in most repute are the following: The nitrate and other preparations of silver, preparations of zinc, digitalis, opium, the narcotic extracts, especially belladonna, and, above all, the bromide of potassium.

The nitrate of silver has long been a remedy for epilepsy, and its occasional efficacy rests on abundant testimony. That it will not effect a cure in the majority of cases is not a sufficient reason for failing to make trial of it in cases which resist other remedies. Beginning with a fraction of a grain three times daily, the dose may be gradually increased to three or four grains. An effect of the prolonged use of this remedy in some cases is to be borne in mind—namely, permanent blueness of the skin. To avoid this effect, the remedy should be suspended for a time after having been continued for two or three months. The oxide of silver is less likely to produce discoloration of the skin, but is less powerful as a remedy.

Of the preparations of zinc, the oxide has been found curative by different observers, and was advocated especially by Herpin, who claimed that in his hands a cure was effected by this remedy in 26 of 42 cases. The mode of administration advised by Herpin is to give at first from six to eight grains daily in divided doses an hour after each meal. The quantity given daily is to be increased by two grains each week until it reaches forty-five grains, this quantity to be continued for three months. The remedy is not to be discontinued after the cessation of the fits.

The prospect of effecting a cure according to Herpin, by this or any remedy, other things being equal, is proportionate to the small number of fits which have occurred. If there have been more than five hundred fits, the probability of a cure is extremely small. In a few cases under my observation in which the treatment as laid down by Herpin was faithfully carried out the remedy proved unsuccessful. The cases reported by this author, however, afford indubitable evidence of a certain amount of curative power; and with reference to any remedy for this disease there is no known means of determining beforehand whether it will be likely to prove successful in any particular case. In the cases in which I have tried this remedy it has been difficult to continue it long after the maximum of quantity is reached, on account of the strong antipathy to it which the patient acquires. In a paper published after his prize essay Herpin advised the lactate in preference to the oxide of zinc. Babington advocated the sulphate of zinc, and carried the quantity given *per diem*, for several weeks in succession, to thirty-six grains without producing nausea. The phosphate and the valerianate of zinc have been recommended.

The ammoniated copper has been found curative. Of 12 cases treated exclusively with this remedy by Herpin, 4 were cured. The dose is half a grain, increased gradually to four or five grains. The sulphate of copper has also been employed successfully.

Digitalis is said to have been long a remedy of much repute in Ireland for epilepsy, and its efficacy was attested by Sharkey, Crampton, McCormack, and Corrigan. The infusion is the preparation to be preferred. The quantity given daily is to be increased to the amount which is tolerated, and continued steadily for several months.

Opium is among the remedies recommended as sometimes curative, and perhaps it deserves a trial more extended than appears as yet to have been given to it. The evils of the habitual use of opium are to be considered, but with a fair probability of effecting a cure of the epilepsy the physician would be warranted in exposing his patient to this danger.

Of the different narcotic extracts, belladonna has the strongest claims, based on the testimony of different observers. Trousseau was an ardent advocate of this remedy as capable of effecting a cure in a certain proportion of cases, and of frequently ameliorating the condition of epileptics when it fails to prove curative. Trousseau insists that a fair test of this remedy requires its continuance for a period not limited to months, but embracing several years. His mode of administration was to begin with a small dose of the extract (gr. $\frac{1}{8}$) once daily, to be continued for a month without increase. At the end of each month the dose is to be doubled, until as large a dose as can be conveniently borne is reached. The tolerance of the remedy differs in different cases. After the disease is perceptibly modified the doses are diminished in the same manner as they were increased. Atropine may be employed in lieu of belladonna, the mode of administration being similar. With the use of belladonna or atropine Trousseau frequently combined the employment of the nitrate of silver, the sulphate of copper, and the lactate of zinc.[1]

Of all the remedies at the present time known to have any controlling influence over this disease, the bromide of potassium justly takes the lead. This remedy has not proved as successful in effecting permanent cures as was anticipated twenty years ago, when it was first employed in this disease; but in a considerable proportion of cases paroxysms do not recur while the remedy is in use, and when this result is not obtained the recurrence of the paroxysms is often rendered less frequent and their severity is diminished. It has effected permanent cures, making due allowance for occasional spontaneous recoveries, but probably the number of instances is not large. The remedy may be carried to the extent of producing distinct bromism. The doses required differ considerably in different cases owing to difference of tolerance. For an adult a scruple of the salt may be given, at first twice or thrice daily. These doses are to be increased until the desired effect upon the disease is produced, unless bromism be induced, even if forty, fifty, or sixty grains in each dose be reached. Whenever bromism is produced, the remedy should be discontinued for a few days and then resumed. If no effect upon the paroxysms be produced within two or three months, it is useless to continue the remedy longer. Having ascertained the effect produced upon the disease and the tolerance of the remedy, it should not be discontinued, except at intervals, for one, two, or more years; that is, provided the paroxysms be prevented or rendered infrequent. It does not seem to be as yet ascertained how far the other bromide salts share in the efficacy of the bromide of potassium. They should be substituted for the latter if it be not well tolerated. The hydrobromic acid appears not to have the same effect as the bromide of potassium, and it has even been thought to increase the frequency of the paroxysms. It is of course important not to carry the use of the bromide of potassium to the extent of causing an alarming degree of bromism; and the fact that, given too largely or continued in large doses too long, it is a dangerous toxical agent, is not to be lost sight of. The following case is of interest as showing the value of the remedy and the tolerance of large doses for a long time: An epileptic patient came to me about six years ago, and was placed upon the bromide of potassium, which was given pretty steadily for two years, the quantity *per diem* being about a drachm. During this period the paroxysms were infrequent and light. The quantity *per diem* was increased to two drachms, and continued steadily for about two years. During this period he had but a few paroxysms, and they were not severe. Of his own accord, from some misapprehension, he increased the quantity *per diem* to three drachms, which he took for about two years. During this period he was exempt from paroxysms. At the end of the two years he came to see me, and I was struck at once by his appearance. His face had a vacant, idiotic expression, and he walked with a staggering gait like that of a drunken man. Recognizing bromism, I then ascertained the quantity which he had been taking for the past two years. I directed him at once to suspend the remedy. He did so for a week or two, but on account of inability to sleep he resumed the dose of a drachm at night. Meanwhile, he had a severe paroxysm. After a few weeks the bromism had entirely disappeared. He has continued the remedy in the dose of a drachm at night, and for two months there has been no recurrence of the paroxysms.

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[1] *Clinique médicale.*

The fact of syphilis having existed at a period even remote should lead to the employment of the antisyphilitic medication—namely, the iodide of potassium and the bichloride of mercury.

It will suffice to mention some other remedies which are worthy of trial in the cases in which the bromides fail, such as strychnia, or nux vomica, recommended in small doses, especially by Marshall Hall; cotyledon umbilicus, an ounce of the fresh juice or half a drachm of the inspissated juice to be given twice daily; indigo, beginning with moderate doses and increasing to the amount which the stomach will bear; valerian in large doses, continued for a long period; musk, camphor, cimicifuga, and the oil of turpentine.

Van der Kolk asserted that counter-irritation over the nucha by means of wet cupping, setons, and issues proved successful in his hands. Others also have claimed success for these measures.

Under the supposition that the paroxysm depends upon a morbid agency springing from some part more or less remote from the nervous centres, and that the aura indicates the point of departure of this agency, it has been proposed to interrupt the nervous communications with the part by means of ligatures, section of nerves, the actual cautery, and even amputation of an extremity; but in the great majority of cases paroxysms are not preceded by an aura, and when this exists there is no ground for the conjecture that it denotes a causative agency. It is claimed that in some instances in which an aura starts from an extremity, a counter-irritant applied between the starting-point and trunk has succeeded in arresting the paroxysm.

Surgical measures for the cure of epilepsy, in addition to those just alluded to, are—ligation of the carotids, tracheotomy, and, after injuries of the head, trephining. Ligation of one or both of the carotids has been repeatedly tried, but the results do not warrant its being recommended. The same is true of tracheotomy, which was warmly advocated by Marshall Hall. Trephining, when there is reason to believe that the epilepsy may depend on depressed fracture of the internal table of the skull, spicula of bone, or a collection of blood or pus, is a warrantable operation. In 35 cases collected and analyzed by Prof. Stephen Smith, the results of this operation were as follows: "Relieved, but not cured, 3. Immediate relief after operation and no further note of result, 2. Relieved for one month or under when last seen, 3;

between one and six months, 3; between six months and one year, 6; between one and five years, 3; set down *cured*, but lapse of time from date of operation to time last seen not given, 7."[1]

It remains now to consider the treatment called for during the paroxysms. These are rarely witnessed by the physician save when a series of paroxysms occur in rapid succession. The duration of a single paroxysm is so short that if the physician do not happen to be present it ends before he reaches the patient. The instructions to friends or attendants should be to place the patient on a bed if practicable, and not to attempt to restrain by force the convulsive movements. All articles of dress which constrict the neck or chest should be removed or loosened. To prevent wounding of the tongue a roll of linen or a piece of soft wood may be introduced between the teeth. A congregation of spectators around the patient should be prevented, and free ventilation should be provided for. The patient should be kept quiet after the paroxysm has ceased, and be soothed by cooling lotions to the head.

Epileptics should sleep in beds with raised sides to prevent injury from being thrown upon the floor by the convulsive movements if a paroxysm occur during the night. They should be under observation, as far as practicable, during the night, for death from suffocation has resulted from the face being pressed into the pillow during a paroxysm.

When a series of paroxysms occur measures may be employed to prevent their recurrence. Ether, valerian, belladonna, and opium may be given for this object. The most effective measure is the inhalation of four or five drops of the nitrite of amyl at the instant of a premonition or of the occurrence of a paroxysm. This drug arrests the paroxysm by relieving the spasm of cerebral arteries. Patients may be provided with this remedy for the purpose of warding off a paroxysm. A paroxysm may perhaps be postponed, but it is doubtful if it be preventable, by any measures resorted to when premonitions are experienced. In a case under my observation a friend sat by the side of a patient the whole of an afternoon, and when the premonition was felt dashed a little water into the face. The paroxysm was apparently prevented for the time, but it occurred as soon as the effort to prevent it was discontinued.

A highly important point connected with the management of epileptics relates to measures to prevent deterioration of the mental faculties. In so far as impairment of mind proceeds from disuse of the mental faculties, it may be obviated or retarded by taking pains to secure as much exercise of these faculties as practicable. It conduces greatly to an alleviation of the unhappy condition of epileptics for them to participate, as far as practicable, in the interests and social enjoyments of life; and if practicable it is sometimes desirable to conceal from them the nature or the extent of the malady with which they are afflicted. I have known marriage to be advised by the physician in the case of a young woman. Such advice cannot be too strongly censured. There is no ground to expect that the malady will be favorably affected by marriage. With reference to this step, it is the duty of the physician to explain fully to the family of the patient, if not to the patient, the nature of the malady.

The existence of epilepsy is an important fact in certain medico-legal cases. In the transient delirium which sometimes succeeds epileptic paroxysms, and which may be incident to epileptoid attacks, violence to others, and even

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[1] Vide article on the "Surgical Treatment of Epilepsy," etc., *New York Journal of Medicine*, March, 1852. Prof. Smith in this article gives a summary of facts with respect to setons, issues, cauterizations and moxas, operations on the scalp, tracheotomy, amputation, operations on the nerves, operations on the arteries, and trephining.

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homicide, may be committed. The occasional occurrence of delirium, lasting for hours or days, in connection with the disease, is to be borne in mind in giving medical testimony. An epileptic under my observation, a quiet, docile man, has repeatedly said to me, with an appearance of much concern, that he had fears that he might kill some one. He stated that he was conscious at the moment of an intense desire to kill any one who irritated him.

The treatment of cases in which epileptoid attacks occur, either with or without the occurrence of epilepsia gravior, is essentially the same as in cases of the latter form of the disease.

CHAPTER VIII.

THE NEUROSES (CONTINUED).

Hysteria.—Hystero-epilepsy.—Catalepsy.—Ecstasy.—Somnambulism.—Tetanus.

Hysteria.

THE name hysteria, as commonly used, embraces a multiplicity of morbid phenomena. It is used to denote an abnormal condition of the nervous system and of the mind, entering largely as a morbid element into a variety of affections. The hysterical condition, as regards the mind, involves a morbid susceptibility to emotions and a defective power of the will to restrain their manifestations. A person, whether male or female, is said to be hysterical who weeps or laughs irrepressibly on inadequate occasions and when these emotional acts are incongruous. Frequently these manifestations of opposite emotions occur in alternation and with an abrupt transition. Weeping and laughter are physiological acts when they represent emotions which are natural; that is, excited by adequate causes and not exceeding the bounds which observation shows to be consistent with health. These acts are pathological when they do not represent corresponding emotions or when they proceed from emotions which are unnatural in view of the circumstances under which they occur. The emotions may be real, but morbidly excited or in morbid excess. On the other hand, the hysterical manifestations in some cases simulate emotions which do not exist. Patients sometimes weep violently without experiencing mental anguish, and laugh immoderately without enjoyment. An intelligent and estimable lady in advanced life, who was under my observation for many years, was subject to spells of uttering loud explosive sounds resembling those of laughter, from which she declared she was unable to refrain. They were called, in the family, her laughing or screaming fits. These were the only hysterical phenomena in the case, and it was frequently necessary to administer a full opiate to arrest them.

A tendency to exaggerate subjective symptoms of disease often enters into the hysterical condition, proceeding sometimes from an exaggerated sense of existing symptoms and sometimes from a morbid desire to excite interest or sympathy. It is an important part of the knowledge and tact of the practitioner to make due allowance for this tendency in his investigation of symptoms. In some cases a morbid perversion of the mind leads patients to undertake to practise gross frauds as regards their ailments. They pretend to have extraordinary disorders, and resort to ingenious and persevering efforts of deception. Here, too, the knowledge and tact of the physician are called into requisition. He meets not infrequently with hysterical malingering as well as hysterical self-deception. Owing to the facts just stated, hysteria is associated in the minds of many with either imaginary complaints or imposition; and hence many are loath to consider themselves as affected with hysteria, and the physician learns reserve in the use of this term in his intercourse with his patients.

Other symptoms of the hysterical condition are a painful sense of constriction in the epigastrium and the sensation of a foreign body in the throat, known as the *globus hystericus*. To these a great number and variety of morbid sensations might be added, such as cephalalgia, *tinnitus aurium*, eructations, abdominal tympanites, irregular chills with yawning and stretching, diuresis, twitching of muscles, cramps, etc. etc. The nervous and mental disorder which constitutes what is commonly called the hysterical condition may be associated also with a host of affections. This condition may enter more or less as an element into almost any disease. There are certain affections often found associated with symptoms denoting this condition. It will suffice to enumerate the more important of these, as follows: 1. Neuralgia, especially supraorbital, cervico-occipital, and dorso-intercostal. A form of neuralgia almost pathognomonic is that in which the patient describes the pain as if produced by driving a nail into the head (clavus hystericus). 2. Hyperæsthesia of the surface, either limited to certain situations or diffused over the body. Affecting the abdominal walls, this simulates in some cases peritonitis. 3. Anæsthesia, local or general. This is sometimes unilateral (hemianæsthesia), and it is generally of the left side. Not infrequently the sensibility to tact remains while the sensibility to pain is lost (analgesia). 4. Different varieties of functional paralysis, especially paraplegia and nervous aphonia. 5. The so-called hysterical cough. (Vide p. 298.) 6. Hyperæsthesia of the joints simulating grave articular disease. 7. Pain and tenderness over one or both of the ovaries, oftener on the left side. 8. Retention of urine. 9. Notable diminution or suppression of urine (ischuria).

What I have called the hysterical condition embraces the milder manifestations of hysteria. Severer forms of the affection are hysterical paroxysms characterized either by convulsive movements or coma more or less complete or delirium. In some cases convulsions constitute the most prominent feature of the paroxysms, coma in these cases usually coexisting. In other cases coma occurs without convulsions, but the coma may precede or follow convulsions, and in some cases delirium is the prominent feature. The severer forms of hysteria, therefore, may be considered as threefold—namely, hysterical convulsions, hysterical coma, and hysterical delirium.

Hysterical convulsions occur in persons subject to the milder manifestations of hysteria, and the latter are frequently premonitions of the former. The development of the paroxysm is generally gradual; the convulsions are preceded by notable distress, referred to the epigastrium, by a sense of choking or suffocation, and sometimes by a feeling as if a solid body or ball ascended from the abdomen to the throat (*globus hystericus*). The convulsive movements are often first manifested in the muscles of the eyelids and eyeballs. Other of the muscles of the face are rarely affected, and hence movements more or less rapid of the eyes and constant nictation are highly distinctive of an hysterical paroxysm if the movements be limited to these facial muscles. These muscles may be alone affected, but frequently the convulsive movements extend to the extremities and trunk.

A distinctive feature of the convulsive movements of the body and limbs is they are not purely automatic. Patients throw their limbs in various directions; they tumble about the bed with violence, rolling from the bed to the floor, and striking with force against solid substances if not prevented; they struggle with those who attempt to restrain them, and sometimes show a prodigious degree of strength. After making active movements for several minutes they remain comparatively quiet for a time, and the convulsions are then renewed. At times they grasp the throat as if to remove an obstruction, strike the breast, or tear the hair. These movements are in great measure voluntary. They are directed by a delirious volition, in this respect differing essentially from the convulsions of epilepsy or eclampsia, which are purely automatic or involuntary. Automatic movements, however, in certain cases enter more or less into hysterical convulsions. Tonic spasm of the posterior muscles of the trunk, bending the body backward so that the trunk, head, and lower limbs form the arc of a circle, constituting what is known as *opisthotonos*, occurs in some cases of hysteria. The jaws are sometimes firmly closed, as in trismus. In the intervals between the active movements the muscles are often rigid.

Consciousness during the convulsive movements and in the intervals appears to be lost, but generally the patient has more or less cognizance of what is said and done; a fact which the practitioner may often turn to good account in the management. The face is sometimes flushed and sometimes pale. Respiration is often notably disturbed, in some cases being extremely rapid and irregular, in other cases slow and suspirious. It is extremely rare for foamy saliva to be ejected from the mouth or for the tongue to be bitten. The spasm of the larynx and muscles of respiration which characterizes epilepsy is wanting; the nostrils are usually dilated; and niçtation and movements of the eyes generally continue. The pulse is frequently but little or not at all affected. The heart, however, sometimes acts with violence and irregularity. The tranquillity of the circulation in some cases is a striking feature. The duration of the paroxysm varies much in different cases. It very rarely, if ever, is limited to a few moments; and it may continue for a period varying from half an hour to several hours. The convulsive movements gradually go off, and the disappearance of the paroxysm is frequently accompanied by cries, groans, sighs, loud weeping, or spasmodic laughter. Copious diuresis often occurs at the termination of the paroxysm.

It is important to discriminate hysterical convulsions from epileptic paroxysms and epileptiform convulsions or eclampsia. The foregoing sketch presents certain points which in general suffice for the differential diagnosis—namely, the evidence of volition in the character of the movements; the gradual development of the attack; absence of convulsive movements of the muscles of the face except those of the eyes; absence of foamy saliva and blood upon the lips; the respiration disordered, but not suspended, by spasm of the larynx and respiratory muscles; the long duration of the paroxysms; the incomplete loss of consciousness; and the occurrence of symptoms denoting the hysterical condition prior to and following the paroxysm. It is a fact, however, not to be lost sight of, that epilepsy or eclampsia may be conjoined with hysterical convulsions.

Hysterical coma enters more or less into the paroxysms just described. It may precede and follow them. It is sometimes accompanied by convulsive movements limited to the eyelids or eyes, but it occurs without any convulsions. In a case of simple, ordinary hysterical coma the patient lies apparently in a state of profound, tranquil sleep. The countenance presents a natural appearance, and the respiration is quiet, with perhaps occasional sighs. The pulse is regular, and is neither accelerated nor retarded, and there is frequently more or less rigidity of the muscles. Attempts to rouse the patient from this state are ineffectual or answers to questions are obtained with much difficulty and in feeble whispers. The muscles offer resistance to efforts to separate the jaws. The quasi-comatose state continues for a variable period, often for hours and sometimes for days, if not successfully treated. Patients in this state are not completely unconscious. They are cognizant of what is said and done. Sensibility to pain is blunted, but not abolished; pinching or pricking the skin may be borne without flinching, but stronger impressions, such as are produced by the application of a heated hammer or the prolonged cold douche, occasion suffering.

Attacks of this kind occasion great alarm, and the physician will naturally participate in the apprehensions of friends if he fail to determine the nature of the affection. It is to be discriminated from apoplexy, and the diagnostic points are as follows: The attacks do not present the stertor of apoplexy, and hemiplegia is wanting; the pupils respond readily to light; the patient is perhaps known to be subject to hysteria. The symptoms of the hysterical condition precede the coma. The age of the patient will be likely to be under the period when apoplexy is most liable to occur; and in ordinary hysterical coma the nature of the affection is generally speedily declared by the efficacy of certain measures of treatment to be presently mentioned.

Uræmic coma is to be excluded. Antecedent and coexisting symptoms relating to the urine, general dropsy in some cases, and the absence of hysterical premonitions, will suffice for this differential diagnosis. Abercrombie related cases of fatal coma occurring in women which are liable to be mistaken for hysteria, nothing being found after death to account for the coma except cerebral congestion. Williams described these cases in the following terms: "A young female becomes anæmic, and after exhibiting various symptoms of feeble general circulation, with headache, drowsiness, and impaired sensorial functions, suddenly becomes worse, passes into a state of stupor, with dilated pupils, sometimes varied by slight manifestations of delirium, throbbing of the carotids and partial heat of the head, and dies comatose. On opening the head a small quantity of serum is found under the arachnoid and in the ventricles, sometimes with a little lymph (in one case there was none). The vascularity of the membranes is remarkable, but the vessels most distended are the veins, and in the larger of these and in the longitudinal sinus there is a firm coagulum."[1] I had met with cases answering to this description many years ago, before uræmia was as well understood as now. The existence of uræmia in such cases seems now to afford a rational interpretation of the phenomena; yet I have met with a case in which repeated careful examinations of the urine gave no evidence of renal disease.

There is a possibility of confounding the coma dependent on meningitis with hysterical coma. Meningitis is to be excluded by the fact that the coma has not been preceded by the diagnostic symptoms of that disease—namely, pain in the head, intolerance of light, suffusion of the eyes, throbbing of the carotids, etc. The thermometer may aid in the exclusion of meningeal or any other acute inflammation. If an acute inflammation exist, the heat of the body is more or less raised; whereas hysteria, existing alone, does not usually give rise to any increase of heat. By this also meningitis may be excluded in cases of hysterical delirium. Hysteria may, however, be attended by rise of temperature, so that the existence of pyrexia in itself does not positively exclude the existence of hysteria.

Under the head of hysterical coma may be embraced certain extraordinary cases in which the mental faculties, the senses, and sensibility are suspended more completely and for a longer period than in ordinary cases. These cases are likely to occasion much embarrassment in the mind of the young practitioner. As an illustration, in the early editions of this work I have given

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[1] *Principles of Medicine.*

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with considerable detail an account of a hospital case of which a synopsis is as follows: The patient, a man twenty-five years of age, was admitted in a state of unconsciousness, the previous history not having been ascertained. For seven days he remained most of the time in an unconscious state. The eyes were sometimes open and sometimes closed. He appeared to take no notice. The pupils were mobile, and neither contracted nor dilated. Flies creeping over the face, and even over the conjunctiva, did not disturb him. Drinks introduced into the mouth were retained there for some time, and the greater part escaped. The respirations were perfectly normal; the pulse 76 and regular; the skin was natural, and there was neither distension nor tenderness of the abdomen. He lay motionless, not changing his position, and giving no manifestation of suffering.

The treatment consisted of the cold douche (which had no effect), enemata of turpentine and asafœtida, and fluid nourishment by the mouth. The urine was withdrawn with the catheter. On the fourth day there was marked improvement. The eyes were open and denoted intelligence, but he did not speak or give heed to questions. He took nutriment freely and with apparent relish. On this day he had a paroxysm of violent weeping.

He relapsed into the former condition on the fifth day. Castor oil was prescribed on this day, no dejection having taken place since his admission. He urinated voluntarily while preparations were making to introduce the catheter. The bowels were moved freely after four minims of croton oil had been given. He resisted the administration of the oil as well as of drink and nourishment. The dejections were not passed in bed, but he disposed his body so that they were passed upon the floor. On the seventh day, there being no material change, "firing" was resorted to by means of a hammer heated by boiling water. He bore the heated iron for some time without manifesting pain, but on continuing the application he made vigorous resistance, and at length protruded the tongue when requested, and took brandy and water freely.

On the eighth day the improvement was marked. He took, however, but little notice, and replied to questions slowly in a feeble whisper. On the eleventh day he was dressed, without any effort on his part, and made to sit up. He remained sitting in one position, the eyes fixed in one direction. He protruded the tongue slowly and partially when requested, but did not reply to questions. On the twelfth day there was a marked change. He greeted me with a smile and offered his hand. He walked of his own accord.

He relapsed on the fourteenth day into the former state of unconsciousness, and "firing" was again resorted to. By this measure he was roused sufficiently to take nutriment. He continued in this state until the seventeenth day. On that day he was made to walk about the ward with assistance, and the experiment was tried of leaving him unsupported. After remaining motionless for several minutes he dropped down, but in a way not to receive any injury.

On the eighteenth day he talked and walked about of his own accord. On the nineteenth day he again relapsed into silence and unwillingness to take food. "Firing" was resumed, and by means of this measure he was made to walk into the open air. On the twentieth day he walked about and talked freely. After this date he progressively improved, and he was discharged well at the end of three weeks.

Nothing could be ascertained respecting the cause of this attack. The patient stated after his recovery that he recollected what had occurred during his illness. I did not discover that the mind was exclusively occupied by any dominant idea, as in ecstasy. Consciousness was not lost, but the exercise of the faculties dependent on the cerebro-spinal system was in a great measure suspended. A morbid moral perverseness appeared to be an element in the case, as in most cases of hysteria, but it is certain that the patient was not a malingerer.

The diagnosis of hysterical coma in this case was based on the symptoms which have been stated, together with the absence of fever, as well as of the evidence of disease of the kidneys or of any other organ. I have met with a few other cases presenting the same characteristic features, and in two of these instances the patients were of the male sex. In one of the cases catalepsy existed. In each case the termination was in recovery.

Hysterical delirium occurs generally subsequent to, or in alternation with, the paroxysms of convulsions or of coma which have been described, but it also occurs alone. It is preceded and followed by symptoms characteristic of the hysterical condition. The delirium is active, and is manifested in some cases by wild, excited talking, the mind passing rapidly from one topic to another. The mind may run on either gay or grave topics, or there may be an incongruous union of both. Patients are sometimes violent. Occasionally they use vulgar and obscene language. In some cases the mind acts under the influence of insane delusions, and these sometimes involve spectral illusions.

Of the pathology of hysteria in the severer forms just described, all that can be said, with our present knowledge, is, that it involves a functional morbid condition affecting the nervous system, especially in its relations with the mental faculties. An important element of the affection is disorder and weakness of the will; the affection, however, is by no means purely mental, but proceeds from a pathological state of the cerebral organs with which the perceptive and emotional faculties, together with the will, are connected. The three forms of hysteria which have been described differ considerably as regards the morbid manifestations, but that they are different forms of one affection is shown by their coexistence or occurrence in alternation, by certain symptoms characteristic of the hysterical condition which occur alike in connection with each form, and by the fact that what we know of the causation applies equally to the three forms.

Hysteria may occur at any period of life, but in the majority of cases patients are between fifteen and twenty years of age. I have known it to be well marked in a girl ten years old. It is of rare occurrence either before the evolution of the sexual instinct or after the period when it may be supposed that venereal desires have ceased; but it sometimes continues into advanced age. The symptoms which characterize the hysterical condition are not very uncommon in males. The name hysteria, therefore, which in its etymology refers to the uterus, is a misnomer.

The causation appears to require a peculiarity of constitution. There are some persons who are constitutionally prone to hysteria, and there are many who are incapable of having it. The hysterical diathesis often exists in members of the same family. A great variety of causes may contribute to the development of the affection, among the more frequent being anæmia, overtaxing of mind and body, mental anxiety or grief, and the prostration incident to various diseases. The influence of causes pertaining to the sexual system has doubtless been much exaggerated; but the agency of excessive venereal indulgence or of masturbation on the one hand, and of continence on the other hand, in certain cases is not to be doubted. Sudden disappointment, affliction from loss of friends, violent anger, jealousy, and other kinds of strong mental excitement, often act as exciting causes of an hysterical paroxysm. A desire to excite in the minds of friends or others anxiety or alarm, and to furnish occasion for interest or sympathy, contributes to that abandonment or perversion of the power of the will which enters into the paroxysms. The hysterical constitution may be developed, and is often fostered, by the absence of wholesome restraint and discipline in the management of youth, and by an unfortunate habit to which women, more than men, often become addicted—namely, of concentrating unduly the attention on personal sensations and sentiments.

As regards PROGNOSIS, hysterical attacks are proverbially devoid of danger. The practitioner, however, is not to lose sight of the fact that hysteria may be associated with affections which are serious. He should not be too ready to set down morbid phenomena to the account of hysteria; and it behooves him to be careful not to confound hysteria with other affections.

The TREATMENT of hysteria is to be considered with reference—*first*, to the hysterical condition; and *second*, to the paroxysms which have been described.

The hysterical condition calls for remedies to soothe the nervous system, but opiates are to be avoided. Valerian, asafœtida, lactucarium, ether, belladonna, hyoscyamus, and the valerianate of ammonia are appropriate remedies. Of these, the first two are the most efficient. The bromide of potassium has in some cases a remarkably soothing effect. Alcoholic stimulants should be recommended, if at all, with great reserve, lest the patient take advantage of the sanction of the physician and resort to them more frequently and freely than prudence warrants. The most important part of the treatment is hygienic. The object is to invigorate both body and mind. With regard to bodily vigor, good diet, abundant exercise in the open air, and regular habits as regards sleep are important. With regard to mental vigor, healthful occupation of mind, and avoidance as far as possible of everything calculated to produce undue development of the sentiments and passions or to excite the imagination, are to be enjoined. The causes which may be suspected in individual cases are to be removed as far as practicable. The patient should be encouraged to endeavor to increase the power of the will to resist a tendency to give way to emotional disturbance. For this purpose the discreditable character of the affection may sometimes be referred to with advantage. The moral management will involve details suited to individual cases, which must be left to the discretion, delicacy, and tact of the practitioner. Cases in which ailments are exaggerated or simulated with a view to excite attention, curiosity, or commiseration are unhappily not infrequent. As the determination of the patients in these cases is not to get well, the varied measures of treatment which are adopted prove of course ineffectual. These cases sometimes tax severely the patience and temper of the physician.

In treating cases of hysterical convulsions it is to be borne in mind that if left to pursue their course, simply preventing the patient from the self-infliction of wounds or bruises, they would end spontaneously; and it would perhaps be as well for the patient if the paroxysm were allowed to exhaust itself; but the anxiety and alarm of friends generally require a resort to measures to arrest the convulsive movements. The prolonged application of the cold douche to the head is generally successful. The head of a patient is to be held over a tub and cold water poured upon it continuously until the convulsions cease and the patient admits being relieved and begs that the measure may be discontinued. Remembering that consciousness is not usually abolished, and that the convulsions depend upon a delirious volition, the physician should take care to state repeatedly that the douche is to be persisted in until the patient is able to express relief, and is to be repeated if the relief be not permanent. It is unquestionable that the efficiency of this measure depends considerably, if not chiefly, on its moral effect. The physician should also give to the friends, within the hearing of the patient, positive assurances that there is no danger and that the paroxysm will certainly be arrested. After the convulsions cease, if the patient be much distressed, an opiate may be given, either by the mouth or by the hypodermic method, but if valerian, asafœtida, or ether will suffice, it is to be preferred.

In an ordinary paroxysm of hysterical coma the douche, as just described, will almost invariably succeed in restoring consciousness. The same course is to be pursued, with respect to moral management, as in paroxysms of hysterical convulsions. The treatment after consciousness is restored is the same as in cases of convulsions after these have ceased. In cases of prolonged hysterical coma "firing," repeated from time to time, is the most effective measure. Nourishment should be given, forcibly if necessary, and as sensibility and consciousness return the exercise of the will in taking food, sitting up, and performing other voluntary acts should be enforced.

In hysterical delirium, if mild, the remedies which have been mentioned, valerian, asafœtida, etc., with soothing management, may suffice. If the delirium be violent, the tartrate of antimony and potassium, carried to the point of nausea, will be likely to tranquillize the patient.

After the paroxysms have ceased in each of the three forms the treatment resolves itself into that called for by the hysterical condition.

Among the most remarkable phenomena of hysteria is the influence exerted upon anæsthetic parts by the application of various metals. The application of metals for this purpose is called *metallo-therapy.* The main facts were discovered long ago by Burge; but they have been more fully studied in late years by Charcot and his pupils in the Salpêtrière in Paris, where most extraordinary and unusual forms of hysteria are observed. It is found that the application of certain metals to an anæsthetic part restores sensation in this part and often over a wider district, and strangely causes a transfer of the anæsthesia to an exactly corresponding part of the opposite side. The most efficacious metals are iron, gold, silver, zinc, and copper, but others are also employed. Not only metals, but magnets, blocks of wood, mustard plasters, and other agents may produce similar effects. In a similar way have been produced restoration and transference of unilateral loss of vision, hearing, taste, and smell. An explanation of these strange phenomena has not been given, nor is the practical application of metallo-therapy of much importance.

Hystero-epilepsy.

Hysteria and epilepsy are distinct affections, but they are not antagonistic to or incompatible with each other. A person may be subject to both, and it may happen that hysterical phenomena may precede or follow an epileptic paroxysm. The term hystero-epilepsy, as used by Charcot and others, however, is applied to cases in which hysteria simulates more or less of the phenomena of epilepsy. In an hystero-epileptic paroxysm hysteria is the primary and essential malady, but in addition there are tonic or clonic convulsions which are automatic. The tongue or cheek may be wounded and foamy saliva escape from the mouth. There may be an aura which, according to Charcot, often has its source in one of the ovaries. Pressure over the region of the ovary sometimes excites, and methodic compression sometimes averts, a paroxysm. An important point of distinction between a combination of the two affections and the simulation of epilepsy is that the latter does not involve a tendency to the recurrence of epileptic distinct from hysterical paroxysms.

The prognosis is more favorable than in cases of epilepsy existing independently of this etiological connection, and the treatment has relation chiefly to the hysteria.

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Catalepsy.

The affection called catalepsy, as this term is now commonly used, is evidently allied to one of the forms of hysteria—namely, hysterical coma. In the cataleptic state the sensory functions, volition, and consciousness are for the greatest part or entirely suspended. So far, the state is essentially the same as in some cases of hysterical coma; but superadded is a peculiar wax-like rigidity of the voluntary muscles, retaining the limbs and trunk in a fixed position, the different parts of the body preserving the positions in which they may be placed by the hands of another. This superadded feature is characteristic of catalepsy.

The cataleptic state is generally preceded by symptoms of the hysterical condition, but it is sometimes developed suddenly; that is, without premonitions. The patient while in this state remains immovable, preserving the position in which the body happened to be at the time of the attack. In some cases the rigidity of the muscles is such that they offer considerable resistance when efforts are made to alter the position of any part, but in other cases the different parts are easily moved. The trunk or limbs are retained in positions which in health would require a strong exertion of the will, and for a longer period than would be possible in health. Thus, the limbs are sometimes kept for a long time extended, or, the patient lying on the back, the lower limbs may be raised and the trunk elevated, so that the body rests only on the sacrum; and this position, which, voluntarily assumed, would soon become insupportable, is preserved for a considerable period. This remarkable feature is more or less marked in different cases; in other words, the catalepsy may be complete or incomplete.

As in cases of hysterical coma, the vital functions may be but little or not at all disturbed. The circulation may be regular, respiration natural, the temperature normal, and, if food be ingested, the processes involved in nutrition may be well performed.

The affection is paroxysmal, but different cases differ widely as regards the frequency and the duration of the paroxysms. They are sometimes short, lasting but a few moments; not infrequently they continue for several days; and they may extend to weeks and even months. In the majority of cases the duration is three or four hours. A single paroxysm only may occur, and, on the other hand, paroxysms have been known to be repeated daily for a long period. A case is cited by Puel in which twelve hundred paroxysms occurred within twenty-eight months.

Catalepsy in this country, exclusive of cases of so-called mesmerism or animal magnetism, must be extremely rare. With pretty large opportunities for clinical observation for more than forty years, I have met with but 2 well-marked cases. The statistical researches of Puel, embracing results of an analysis of 148 cases, showed that the disease is not peculiar to females. Of these 148 cases, 80 were in females and 68 in males.[1] In one of the cases which I have seen the patient was of the male sex. A large majority of cases are between ten and thirty years of age. The affection, however, may occur at less than ten years and at an advanced period of life. It appears to be induced especially by mental causes. Melancholy, overtaxing the intellect, and violent excitement of the passions favor its development, and causes frequently determining an attack are hatred, jealousy, fright, domestic affliction, reverses of fortune, etc.

The PROGNOSIS is always favorable as regards danger to life. It has, however, been observed to precede the occurrence of insanity, paralysis, and epilepsy.

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[1] *Mém. de l'Acad. de Méd.*, Paris, 1856. Vide Valleix, *op. cit.*

The principles of TREATMENT are essentially the same as in cases of hysterical coma. The condition of the muscles calls for the use of friction with stimulating liniments. Forcible alimentation may be required if the cataleptic paroxysm be prolonged.

An abnormal state, analogous to that of catalepsy, is among the curious phenomena of hypnotism, formerly known as mesmerism or animal magnetism. The consideration of these phenomena—which of late years have deservedly received scientific attention—does not come within the scope of this work.

I shall content myself with a bare allusion to certain abnormal conditions which properly belong to the domain of psychology. One of these is the condition known as *ecstasy*. In this condition the mind, absorbed in a dominant idea, becomes insensible to surrounding objects. In some cases during the ecstatic state the body remains immovably fixed, as in catalepsy. Extraordinary visual hallucinations occur in some cases. The mental condition differs from that of catalepsy in this: the mind is active, and thoughts or visions which occur are recollected afterward, whereas in catalepsy the action of the mind is suspended and the period passed in the paroxysm is a blank in the patient's memory. Another name for the ecstatic state is *trance*. As in some cases of catalepsy, the respiration and circulation may become so feeble that without close examination life may be supposed to be extinct. If the breathing be not readily ascertained and the pulse be inappreciable, auscultation with the binaural stethoscope can hardly fail to reveal the heart-sounds in such cases.

Another condition is that called *somnambulism*. This condition is incident to sleep. It embraces the mental and physical performances, sometimes very extraordinary, which are observed in sleep-walkers. Similar phenomena are observed in the condition known as the *mesmeric or magnetic sleep*.

Tetanus.

The term tetanus is applied to an affection characterized by persisting rigidity of the muscles of the jaw, and frequently of the greater part of the voluntary muscular system, with paroxysms of tonic spasms, the intelligence being preserved. In the great majority of cases this affection follows a wound or local injury of some kind. It is then distinguished as *traumatic tetanus*, and when not traumatic it is distinguished as *idiopathic tetanus*. Its symptomatic characters are essentially the same whether the disease be traumatic or idiopathic. On account of the large preponderance of traumatic cases it belongs among the surgical affections, and is treated of at length in comprehensive works on surgery. I shall consider it as an idiopathic affection.

Idiopathic tetanus is everywhere rare, and in cold or temperate climates it is one of the rarest of affections. It is less rare in tropical climates. It is one of the affections to which newly-born children are subject, especially in tropical climates, but occasionally everywhere. In adults it seems sometimes attributable to exposure to cold, and with this apparent causation it is called by some writers *tetanus a frigore*. It has been conjectured that the disease is sometimes a manifestation of rheumatism.

In many cases idiopathic tetanus is developed abruptly, without premonitions; but in a certain proportion of cases it is preceded by indefinite ailments, such as chilly sensations, sense of fatigue, etc. The muscles first affected are those of the neck and lower jaw. The affection is sometimes limited to these muscles, and it is then distinguished as *trismus*. The jaws are firmly shut by the rigid contraction of the muscles, and hence the affection is known as *lockjaw*. The mouth in some cases cannot be opened by any force which it would be prudent to employ, and drink, nourishment, or remedies can be taken only through the spaces between the teeth. The muscles of the face are frequently involved, giving rise to distortions of the features which are sometimes terrific. The angles of the mouth are drawn backward and upward, giving the expression called *risus sardonicus*. The tongue is sometimes severely bitten by being caught between the teeth. The pharyngeal muscles are not infrequently involved, preventing deglutition.

If the affection extend to the muscles of the trunk, the abdomen becomes retracted and rigid and the respiratory movements of the chest are restrained. The limbs become rigidly extended. The entire body is sometimes immovable and stiff, so that it may be raised by the head or feet as if it were a statue.

Rigidity of the muscles is persistent, but at intervals spasm is added to the persisting contraction. In the paroxysms of spasm or convulsions the body is frequently bent backward by the predominant force of the posterior muscles of the trunk; and in extreme cases the body forms the arc of a circle, resting on the head and sacrum, and sometimes on the head and heels. This is called *opisthotonos*. A much less frequent form of curvature is produced by the predominant contraction of the muscles on the anterior portion of the trunk. This is called *emprosthotonos*. A lateral curvature, still more rare, is called *pleurosthotonos*. The paroxysms vary in duration, and they occur at intervals which vary much in different cases. They are attended with severe pain. The paroxysms may be excited by any mental emotion and by impressions transmitted from the surface of the body. In this respect, as also in the character of the convulsions, they resemble those which take place in cases of poisoning by strychnia.

As already stated in the enumeration of the characters distinctive of tetanus, the intelligence is preserved. Delirium and coma are wanting. In general, the paroxysms are more frequent during the day than during the night. If deglutition be prevented by pharyngeal spasm, the patient suffers from thirst and hunger. Foamy saliva under these circumstances collects and escapes from the mouth. The bowels are usually constipated, but in some cases involuntary dejections occur. Retention of urine may occur, or, on the other hand, it may be passed involuntarily. The circulation may be but little or not at all disturbed, save as an effect of disturbance of the respiration. Respiration is embarrassed in proportion as the respiratory muscles are affected. The embarrassment is usually great during the paroxysms, and fatal apnœa may be induced. The temperature of the body varies much in different cases. In a certain proportion of cases there is moderate or slight fever. In other cases there is notable pyrexia, the thermometer in the axilla sometimes showing a rise to 110° F. The temperature is measurably an effect of the muscular spasms. The temperature is always raised during exacerbations in which the muscular contractions are increased. Profuse sweating occurs in the exacerbations, and more or less perspiration exists in the intervals. Contraction of the pupil to the size of a pin's point during the exacerbations is often observed, the contraction going off in the intervals. A frequent symptom is pain at the pit of the stomach, piercing through to the back.

The disease sometimes destroys life within twenty-four hours. On the other hand, in rare cases it continues for several weeks, when it is called chronic tetanus.

In a large proportion of cases the affection ends fatally after a duration varying between twenty-four hours and twenty days, the average duration being not far from ten days. The fatality is somewhat less from idiopathic than from traumatic tetanus. The mode of dying may be by apnœa or by asthenia. The danger of apnœa is in proportion to the amount of embarrassment of respiration. Death by asthenia is caused by the exhaustion incident to long continuance of the rigidity and spasms, together with the difficulty in some cases of alimentation. It has been noticed that in some cases rigor mortis succeeds the spasms during life without any appreciable interval of muscular relaxation.

The distinctive characters of the affection render the DIAGNOSIS sufficiently easy. It is to be distinguished from spinal meningitis and from cerebro-spinal meningitis by the absence of local symptoms denoting inflammation, and from the latter of these affections by the absence of cephalalgia, delirium, and coma. Absence of coma distinguishes it from epilepsy. Moreover, the latter is purely paroxysmal, the muscles not being contracted in the intervals. Absence of characters distinctive of hysteria sufficiently distinguish it from that affection. It is, however, to be recollected that trismus, with or without convulsions, is an occasional manifestation of hysteria, and under these circumstances is unattended with danger. The phenomena produced by strychnia in poisonous doses bear the closest resemblance to those of tetanus, and to discriminate between them is of great importance in a medico-legal point of view. Within late years strychnia has been repeatedly employed for homicidal and suicidal purposes. The following differential symptoms are quoted from Todd:[1]

"*As regards the Tetanoid State from Strychnine.*—*a.* The *rapid* supervention of tetanoid convulsions affecting chiefly, and with most intensity, the muscles of the trunk and spine, causing an active and violent opisthotonos rarely met with in tetanus. *b.* A rigid and tetanoid state of the muscles of the lower extremities, with somewhat less intensity than those of the trunk; the limbs extended and the feet drawn powerfully inward by the action of the tibialis postici muscles. The upper extremities affected also, but in a less degree, and the hands generally semiflexed. *c.* The trismus existing only imperfectly, and the *facies tetanica* very slightly or not at all; swallowing perfect, but the mode of deglutition peculiar, the patient snapping at the liquid offered and gulping it down with an effort, in a manner very similar to that in which hydrophobic patients swallow. *d.* The attacks of opisthotonos very frequent, seemingly exquisitely painful, and ushered in by a cry more or less loud.

"*As regards Tetanus.*—*a.* The symptoms coming on *gradually*, and the trismus the earliest, the most prominent, and most important one. *b.* The *facies tetanica* a very characteristic symptom. *c.* The attacks of opisthotonos less frequent, less extensive, and less severe than in poisoning by strychnine. *d.* The extremities the last parts affected, and suffering much less from the tonic spasms than other parts. *e.* Deglutition slow and difficult, and sometimes impossible, owing to the spasmodic closure of the mouth."

To these differential points it is to be added that a fatal dose of strychnia kills quickly, usually within an hour, whereas in fatal cases of tetanus life is prolonged generally for two or three days.

Punctate hemorrhages are very frequently found in various parts of the central nervous system, particularly in the medulla oblongata and spinal cord, after death from tetanus; but these changes are probably secondary, as they are found after death from other convulsive diseases. No morbid changes have been discovered to which tetanus can be attributed. In the present state of our knowledge, therefore, the disease is to be considered as functional, the changes which may be found after death being either secondary or associated by coincidence. With respect to the pathology, the close analogy of the symptomatic phenomena to those produced by strychnia is a strong point in favor of the supposition that the disease involves the presence in the blood of a toxical agent acting upon the spinal cord and the medulla oblongata.

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[1] *Clinical Lectures on the Nervous System.*

The symptoms show an exaggerated reflex excitability of the medulla and the cord as a marked pathological feature of the disease.

The TREATMENT of tetanus by different methods has proved unsuccessful in a large proportion of cases. Among the measures which have seemed to be sometimes successful are—opium given in large doses, aconite, alcoholic stimulants carried to the extent of producing intoxication, and the inhalation of chloroform. Other remedies advocated as sometimes successful are belladonna, cannabis indica, and quinia in large doses. Bloodletting and counter-irritation over the spine have been largely tried, but are not to be recommended. Ice applied to the spine has been found useful. Cold affusion has been employed with success. The high temperature of the body in some cases indicates antipyretic measures of treatment.

In addition to the remedies already named, others more recently employed are the hydrate of chloral, the Calabar bean, curare, and the nitrite of amyl. Cases which have ended favorably under treatment by these remedies have been reported; but clinical facts do not, as yet, warrant positive conclusions respecting the remedial power which they respectively exert over the disease. The great number of remedies advocated as possessing curative efficacy in tetanus suggests a reflection which has been mentioned in other connections: When cases of a disease have ended favorably under a host of remedies it may be reasonably conjectured that the ending in these cases was due more to an intrinsic tendency of the disease than to the remedies used. It remains to be determined in how large a proportion of cases tetanus would end in recovery without any actual medicinal interference.

In the following case improvement and recovery took place after all treatment had been discontinued: A boy seven years of age, the son of a poor washerwoman, came under my observation about a fortnight after the beginning of the disease. He then had every few minutes paroxysms of opisthotonic spasms, with rigidity of the limbs, pronation of the thumbs, erection of the penis, and firm closure of the jaws. The paroxysms lasted two or three minutes; they were accompanied by great embarrassment of breathing; they occurred spontaneously, but were brought about whenever the body was moved. During the paroxysms the body could be lifted like a statue by raising one leg. The angles of the mouth were depressed and the features expressed great distress. A certain amount of trismus was constant; and the mouth could not be opened sufficiently for the patient to take food freely or to protrude the tongue. The pain was severe during the paroxysms. The mind was clear. He obtained but little sleep. The pulse was not accelerated. The disease could not be referred to any cause.

The case had been treated with morphia, quinia, and the valerianate of zinc. The patient was kept under the influence of chloroform for several hours. The effect was to relax the muscles of the jaw and to lessen the frequency of the paroxysms. Owing to a misapprehension this patient had no subsequent treatment. At the end of ten days he was found to have notably improved, and he shortly recovered. After the trial of the chloroform, nothing was done but to give nutritious food. The improvement was very gradual, slight spasms occurring from time to time after he had convalesced sufficiently to be up and about.

A highly important part of the treatment relates to the quietude of the patient. The body should be moved as little as possible, and all excitement of the mind should as far as practicable be avoided. Light, currents of air, and noise should be excluded. Nutritious alimentation is an important part of the treatment. If deglutition be impossible, food should be introduced by means of a stomach-tube, which may be passed through the nostril.

CHAPTER IX.

THE NEUROSES (CONCLUDED).

Rabies.—Delirium Tremens—Alcoholism.—Hypochondriasis.—Pathophobia.

THE affections known as *rabies canina*, or *hydrophobia*, and *delirium tremens*, are to be included among the neuroses. The consideration of these, and of hypochondriasis or pathophobia, will conclude the section of this work devoted to diseases affecting the nervous system.

Rabies.

The affection called *rabies canina*, and more commonly known as *hydrophobia*, is due to the action of a special poison, a virus derived from the mouth of some animal of the canine or feline race—namely, the dog, wolf, fox, or cat. The virus is communicated by inoculation; that is, by the bite of an animal affected with the disease. An animal affected with the disease in this way communicates it to other animals, herbivorous as well as carnivorous. The former rarely communicate it, because they rarely bite other animals or man. That it may be communicated by herbivorous to carnivorous animals appears to be proven. The virus is contained in the fluids of the mouth, in the central nervous system, perhaps in the blood, and in other organs. It appears that the virus enters the body only by inoculation. It is not absorbed from the healthy cutaneous or mucous surface. It has been communicated from man to inferior animals by inoculation. The disease in man is much oftener derived from the dog than from any other animal; and hence the name *rabies canina*. Owing to the pains taken to confine or to destroy animals suspected of having the disease it is rarely seen in man. A few cases have come under my observation.

The disease does not make its appearance for some time after the bite. The usual period of incubation is stated to be from thirty to forty days. There is considerable variation in this period in different cases. In one of the cases which I have observed the patient was bitten seven weeks, and in another case eight weeks, before the disease appeared. There is ground for distrusting the accuracy of statements as regards cases in which the disease is said to follow very quickly after the bite, and also when several years have been supposed to elapse. The error in the first of these cases probably consists in confounding the disease with tetanus. The utmost possible duration of the period of incubation cannot be stated; and this is a point in the natural history of the disease of no little consequence with reference to the anxiety of persons after having been bitten by an animal known or suspected to be rabid. It is probable that the period is very rarely if ever less than eight or ten days, and that it very rarely exceeds a year.

The development of the disease is gradual. It is stated that certain morbid sensations emanating from the cicatrized wound precede other symptoms; but if this be true of some it is certainly not so of all cases. There were no such premonitions in the cases which I have observed. The premonitions are—notable depression of spirits, so that this premonitory period has been called the *stadium melancholicum;* change of disposition, restlessness, mental agitation, vigilance or disturbed sleep, cephalalgia, chilly sensations, loss of appetite, a sense of distress referred to the epigastrium, and sometimes nausea and vomiting. Two or three days elapse before the distinctive characters of the disease are fully declared.

When the disease is developed the most prominent feature is laryngeal spasm excited by the effort to swallow water. The patient has a dread of water because efforts to swallow it occasion violent paroxysms of suffocation, and are generally unsuccessful. The association with these paroxysms gives rise to the fear of water, or hydrophobia. After they have repeatedly been thus produced the mere sight of water may be sufficient to provoke them; but this is not always the case. In the cases which I observed the patients did not suffer any inconvenience from seeing water or hearing the sound of water. Patients are sometimes able to swallow other liquids. In one of the cases which I have noted spirit was taken without much inconvenience, and in another case at times water was taken from a teaspoon without exciting spasm. Pieces of ice were taken without any inconvenience. The paroxysms excited by attempting to swallow water are extremely violent. The patient with a nervous determination drinks precipitantly, and instantly respiration is arrested, the whole frame is agitated, terror and distress are depicted on the countenance, and the water is forcibly ejected from the mouth and nostrils. In some cases paroxysms of laryngismus occur spontaneously. In one of the cases which I have noted they were repeated forty or fifty times in an hour, the patient suddenly and quickly raising himself in bed, the spasm lasting a few seconds, and in the intervals the respiration was hurried and panting. In other cases the patient is free from paroxysms if he do not attempt to swallow liquids. A current of air may excite laryngeal spasm, and the patient complains that it takes away his breath. A bright light, the reflection from a mirror or any polished surface, and any mental excitement have the same effect. The countenance expresses excitement, anxiety, and terror.

An abundant secretion of tenacious mucus from the fauces, together with an increased flow of saliva, leading to frequent and sometimes almost constant expuition, is a distinctive feature. An examination of the throat shows the fauces to be more or less reddened. At first, the mind is simply excited, but in the course of the disease delirium becomes developed. The patient talks wildly and incoherently. Sometimes the delirium assumes the form of mania; the mind acts under the influence of hallucinations, and forcible restraint in some cases becomes necessary. There is no foundation for the vulgar notion that patients assume the character of the animal from which the virus was received and attempt to bite persons around them. In some cases delirium is manifested by excessive tenderness and affection. Satyriasis has been observed in males, and nymphomania in females.

Convulsions occur in a large proportion of cases. These are sometimes clonic, extending more or less over the voluntary muscular system, and they are sometimes tonic, as in tetanus. The pulse becomes frequent and small. Febrile movement does not belong to the history of the disease; the temperature of the body being increased but little or not at all. The surface presents capillary congestion, and is sometimes covered with clammy perspiration. The vital forces progressively fail: the patient is worn out with continued vigilance, the paroxysms of laryngismus, the convulsions, and innutrition; and the mode of dying is by asthenia.

Of all the diseases in the nosology, there is no one in which the intrinsic tendency to death is greater than in this. The disease runs a brief career. death usually taking place on the second or third day, and always within five days, after it has become fully developed.

Small hemorrhages are often found in the central nervous system after death from hydrophobia, but these are probably secondary. Of more importance, however, is the accumulation of small round cells, especially about the blood-vessels. These accumulations have been found with considerable constancy in the posterior half of the medulla oblongata, particularly at or near the deep origin of the nerves in this situation. Similar but less marked changes have been found in the cerebral cortex and elsewhere in the central nervous system. Hyaline metamorphosis of the vascular walls in the nervous centres has also been observed.

The diagnostic features of rabies are highly distinctive. It is distinguished from tetanus by the absence of trismus and the occurrence of delirium. In some cases of tetanus the deglutition of liquids is impossible; but the difficulty arises from spasm of the pharyngeal, not involving the laryngeal, muscles, the latter being affected in rabies. Moreover, convulsions occur at a late period in rabies, and the convulsions are tetanoid only in a certain proportion of cases. The only difficulty of diagnosis relates to the discrimination of cases of true rabies from those in which, to a greater or less extent, hydrophobic phenomena are simulated. Notable repugnance to liquids, susceptibility to currents of air and to light reflected from a mirror or some polished surface, frequent sputation, together with delirium and convulsions, are occasionally incident to various acute diseases; but the hydrophobic phenomena are especially simulated under intense apprehension of rabies after a bite has been inflicted by an animal suspected of being rabid. Excessive fear leads to the belief in the mind of the patient that the disease exists, and this belief leads to the production of certain of the phenomena, especially horror of liquids, delirium, and sometimes a fancied disposition to bite others. Among the points to be considered in this discrimination is the time which has elapsed after the bite. If the morbid phenomena occur immediately or quickly afterward, or, on the other hand, after a very long period, the reality of the disease is very doubtful. If it be evident that the horror of liquids is purely mental or dependent entirely on spasm of the pharynx, as in some cases of hysteria—in other words, if laryngeal spasm be excluded—the affection is not true rabies. The continuance of the hydrophobic phenomena is another point. If they continue for many days or weeks, the affection is not true rabies, for the latter affection ends fatally within five days. The opinion which some have entertained that rabies may be developed in man spontaneously probably has no foundation other than the cases in which hydrophobic phenomena have been incidental to other diseases.

Of the great number and variety of remedies which have been tried for the cure of rabies, one only at the present time claims consideration. This remedy is curare. A few cases have been reported of the successful employment of this remedy administered by subcutaneous injection. There is room for doubt in these cases concerning the diagnosis, and hence other cases must be observed before it can be asserted positively that the remedy has proved in any instance successful. One of the cases reported as cured by this remedy came under my observation. The patient had been bitten by a dog undoubtedly rabid. A servant in the family was bitten by the same dog, and died with rabies. Many of the symptoms in the case which ended in recovery pointed to rabies, but hydrophobia was wanting.[1] In a case reported by Dr. Offenberg of Wickrath (Rhein-Preussen) all the diagnostic symptoms, inclusive of hydrophobia, were apparently well marked. The patient, a maid-servant, had been bitten by a dog evidently rabid, and a member of the family in which she lived, having been bitten by the same dog, died with rabies. The curare was given in successive doses, as follows: two doses of 0.02

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[1] Vide report of this case by Dr. B. A. Watson in *Am. Journ. of Med. Sciences*, July, 1876.

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gramme (gr. $\frac{1}{3}$) after an interval of fifteen minutes; an hour after the last dose 0.03 gramme (gr. $\frac{6}{18}$), the latter repeated twice after an hour's interval, once after an interval of two hours and thirty minutes, and again after two hours and twenty minutes. Temporary general paresis was produced by the remedy.[1] This drug is of uncertain strength, and it must therefore be used with caution. Curare has repeatedly failed to exert any curative influence in hydrophobia.

There is no prophylactic treatment to be relied upon save excision or effectual cauterization of the wound as quickly as possible after it has been inflicted. Amputation of fingers or toes, and even of larger members, is advisable if the extent or character of the wound be such that the excision and cauterization cannot be effectually employed.

It is desirable, of course, to know whether an animal which has inflicted a wound be really rabid. Dogs and cats are often supposed to be rabid when they are affected with epilepsy or some disease other than rabies. It is a popular error that dogs are especially liable to become rabid during the so-called dog-days; that is, the hot months of summer. Cases are quite as likely to occur at other periods of the year. When rabies is suspected the animal should be confined, and not killed until the character of the disease is fully declared. Rabies in the dog is to be suspected when the animal manifests a notable change of habits, becoming shy and irritable, eating straw, bits of paper, etc., and refusing food. When the disease becomes developed the appearance is much changed. The look is depressed and haggard, the ears and tail droop, the quality of the bark is altered, the eyes are watery, the fauces are reddened, saliva flows from the mouth, and there is febrile movement. Dread of water is no test of the malady in the dog and other animals. They frequently lap water without difficulty. Delirium supervenes, and then the dog snaps at his master as well as others, and at any animal which comes in his way. He seldom becomes ferocious, but bites and runs away. He appears sometimes to have hallucinations and snaps at invisible objects. Convulsions may or may not occur. Death takes place usually within five days.

Persons bitten by an animal known to be rabid are by no means invariably affected with rabies, even if no preventive measures be resorted to. The chances of escape are a little more than three to one. The exemption may proceed from an insusceptibility to the poisonous action of the virus, but probably often, in those who escape, the inoculation does not take place. If the bite be inflicted through clothing, the virus will be likely to be wiped away from the teeth before they penetrate the skin. The inoculation is more likely to take place from the bite of the wolf than from that of the dog. Of 250 cases collected by Renault of persons bitten by the wolf, 164, about two-thirds, became affected by rabies.[2]

At the present time (1886) great interest is aroused in a method of inoculation which is claimed by its discoverer, Pasteur, to be preventive of hydrophobia, and which was reported by him to the French Academy of Sciences in October, 1885. Pasteur was led to the adoption of this method by the following series of experiments on dogs and rabbits: If a portion of the brain-substance of a mad dog be injected beneath the dura mater of a rabbit, the latter animal is affected with a disease supposed to be hydrophobia after a period of incubation of fifteen days. Successive inoculations in a series of rabbits of portions of the spinal cord from rabbits dead of experimental hydrophobia cause a gradual diminution of the period of incubation, which after forty to fifty inoculations becomes only seven days, where it seems to remain without much further diminution. The spinal cord of these rabbits contains the hydrophobic virus. Preservation of these cords in dry, warm air leads to a gradual deterioration of the poison, which is finally extinguished. If dogs be subcutaneously inoculated, first with the weak virus (that is, with cords preserved for twelve or fifteen days), and then daily with progressively stronger virus, it is found that they acquire immunity against the strongest virus (that is, fresh cords from hydrophobic rabbits). Pasteur has had the opportunity of applying this method of preventive inoculation to a large number of human beings who have been bitten by animals supposed to be rabid. Up to the present time (June, 1886) of nearly 1000 persons thus inoculated, 8 have died of hydrophobia. Of the fatal cases, 5 were from hydrophobia following wolf-bites (out of 38 wolf-bitten persons treated by Pasteur).

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[1] For a translation of the history of this case, by Dr. S. W. Williams, with full details, vide *New York Med. Record*, Aug. 9, 1879.

[2] Jaccoud, *op. cit.*

The following considerations make it impossible at the present time to judge of the value of Pasteur's inoculations in human beings: It is not known what proportion of the patients treated by Pasteur were bitten by actually rabid animals, and it is probable that the proportion is not large. Of those bitten by rabid animals, in a considerable number of cases the wound soon after its reception was treated by excision and by cauterization and disinfection, and there is reason to believe that these measures are sometimes protective. Furthermore, it is established that of those bitten by rabid dogs a large number, probably at least two-thirds, never develop hydrophobia, even when no preventive treatment has been adopted. At the present writing the period of time allowed for incubation has not elapsed.[1]

Delirium Tremens—Alcoholism.

This affection proceeds from the abuse of alcohol. The various pathological effects of alcohol are considered as incident to a toxical condition, called *alcoholismus* or *alcoholism.* These effects enter directly into the causation of many affections, such as cirrhosis of the liver, fatty liver, epilepsy, muscular tremor, gastritis, pyrosis, and various dyspeptic disorders. Indirectly, alcoholism favors the production of nearly all diseases by lessening the power of resisting their causes, and it contributes to their fatality by impairing the ability to tolerate and overcome them. Taken in sufficiently large quantity, alcohol produces certain immediate effects which belong to the state commonly known as intoxication or drunkenness. The opportunities of observing these effects are, unhappily, but too common. The coma which exists when a person is profoundly intoxicated—or, in common parlance, dead-drunk—is liable to be mistaken for apoplexy. The differential points have been presented in treating of the latter. Intoxication falling short of coma sometimes comes under the cognizance of the physician under circumstances in which the nature of the difficulty is not suspected by friends, as in women and children. Knowledge of the characteristic phenomena is then brought into exercise. The physician in these cases is sometimes bound by delicacy and prudential considerations to withhold an enunciation of the diagnosis.

The habitual use of alcohol beyond certain limits produces a deleterious influence on the whole economy. In cases of chronic alcoholism the digestive powers are weakened, the appetite is impaired, the muscular system is enfeebled, and the generative function decays. The blood is impoverished and nutrition is imperfect and disordered, as shown by flabbiness of the skin and muscles, emaciation, or an abnormal accumulation of fat. Pain in the limbs and back is a symptom of frequent occurrence. Wilks and other writers have called attention to the occasional occurrence of paralysis, particularly in the form of paraplegia, in drunkards (alcoholic paraplegia). The breath and emanations from the skin have a characteristic odor. The deleterious influence on the mental is not less marked than on the physical powers. The perceptions are blunted, the intellectual and moral faculties progressively deteriorate, until at length the confirmed inebriate, miserably cachectic in body and imbruted in mind, has but one object in life—namely, to gratify the morbid craving for alcohol. He exemplifies a variety of the form of mental derangement called dipsomania, from which recovery is extremely rare.

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[1] There is not a strict analogy between Pasteur's experiments on dogs and his inoculations of human beings, as in the former the inoculations are made before, and in the latter after, the reception of the strong hydrophobic virus. (For a full description of Pasteur's methods, vide article by H. M. Biggs in the *N. Y. Med. Journ.*, March 27, 1886, and for a critical analysis of these methods and results, the remarks of A. Flint, Jr., in the same number of the *Journal.*)

Delirium tremens, called also *mania a potu*, is an affection incident to alcoholism. It has been a mooted question whether the affection ordinarily known by these names be due to the sudden withdrawal of an habitual amount of alcoholic stimulation, or whether it be a direct consequence of the long-continued action of alcohol on the brain. Both explanations are correct. In a large proportion of cases its development is evidently owing to the use of alcohol being suspended or much diminished. Thus, it occurs in persons who voluntarily undertake to abandon intemperate habits, who are unable to obtain liquor, or who are prevented from drinking by the occurrence of some disease or accident. It is notoriously common among inebriates who are thrown into prison and among those admitted into hospitals. It often follows paroxysms of intemperance in periodical drinkers when the stomach refuses further alcoholic libations. On the other hand, it is sometimes developed notwithstanding the continued use of the habitual amount of alcohol.

The SYMPTOMS attending the access are anorexia, insomnia, muscular tremor, especially tremulousness of the tongue, and notable dejection. This state is known among drunkards as "the horrors." The mental depression is so great as not infrequently to lead to suicide. There is no pain in the head or febrile movement. The pulse is generally feeble and the surface is cool. This stage continues for one, two, or three days.

The development of the disease is characterized by mental aberration of a peculiar character. The derangement is characteristic, although presenting considerable diversity in different cases. The expression becomes wild, and the eyes are either vacant or staring. The movements are quick. The patient is constantly in motion. He talks incoherently. His mind wanders from one subject to another. He fancies that he has important business to attend to, wishes to go out, and requires to be constantly watched. To these manifestations of mental disorder are added illusions of the senses, phantasms, and hallucinations of various kinds. He sees imaginary objects, such as mice, dogs, cats, lice, snakes, and ferocious animals. He hears noises of animals or men, answers imaginary questions, and often fancies the proximity of persons who are bent on insulting or ridiculing him or from whom he apprehends personal violence. Uncouth and unnatural representations appear to him to be going on. At first he may be able to appreciate the unreality of what he sees, hears, and imagines; but after a time his morbid perceptions become delusions, and, however extraordinary and absurd, they are real to the sufferer. They are sometimes of a character to inspire the greatest possible terror. Thus, patients not infrequently leap from windows and are dashed in pieces, believing that they are pursued by wild beasts or by men who seek their lives. A patient who escaped in his night-clothes under a sense of personal danger ran barefooted over frozen ground for fifteen miles before he was overtaken by men on horses who followed in pursuit. The degree of terror must be fearful to lead to such an amount of physical effort and endurance. In some cases the delusions are ludicrous. Thus, a patient declared that he should be able to sleep, but whenever he fell into a doze there were persons under his bed who tickled his fundament with straws. Under the apprehensions which the delusions excite patients are sometimes violent and dangerous, but they are usually controlled without much difficulty.

The insomnia continues. The patient gets no sleep for two, three, or four days, and sometimes for a still longer period, after the development of the affection. The delirium is always much worse during the night. Tremulousness of the muscles continues, but this is not always a marked symptom, although it enters into the name. Vigilance being a more constant symptom, it has been proposed to call the affection *delirium vigilans* instead of *delirium tremens.* Anorexia continues, the bowels usually are constipated, the skin is cool, the pulse is feeble, and, except under mental excitement, may be but little or not at all accelerated; the patient makes no complaint of pain in the head or elsewhere, and generally declares that he is well.

If the affection end favorably sleep at length occurs. If the patient once become soundly asleep, he will be likely to remain so for many hours, or if he awake the speedy return of sleep may be counted upon. After a protracted sleep, generally the delirium is found to have disappeared. Occasionally it continues, but is diminished, and disappears after renewal of sleep. Sleep appears to be not merely a criterion, but a means of convalescence. After sleeping much of the time for one or two days, convalescence is established, and the patient recovers more or less slowly or rapidly according to the duration of the affection and his physical condition at the time of the attack. If the affection pursue an unfavorable course, the insomnia persists and notable prostration ensues. The condition is not unlike that in an advanced stage of typhus; the delirium continues, with efforts to get out of bed; subsultus tendinum occurs; the pulse becomes more and more frequent and feeble; the skin is sometimes bathed in perspiration; and death takes place by asthenia, although the fatal termination is usually preceded by coma.

The PROGNOSIS is generally favorable. When not associated with other affections a fatal termination is rare. The affection destroys life *per se,* chiefly in cases in which repeated attacks have occurred and the constitution is broken by a long-existing alcoholic cachexia. Occurring after wounds, surgical operations, or accidents, and associated with other affections—as, for example, with pneumonitis—it adds much to the gravity of the prognosis.

The symptomatic phenomena show the affection to be cerebral, but the nature of the morbid condition, as in the other neuroses, is unknown. It is important to bear in mind that inflammation is not involved, nor is there ground to believe that congestion is an essential element. There is usually some increase of the cerebro-spinal fluid, and the ventricles of the brain may contain an abnormal quantity of serum. The substance of the brain may be œdematous. This condition is sometimes called the wet brain of drunkards. All that can be said of the pathological character with our present knowledge is, that the prolonged toxical action of alcohol on the brain induces a peculiar morbid condition giving rise to those manifestations of disorder which are characteristic of this affection, especially when the use of alcohol is from any cause suspended. There is reason to believe that the morbid phenomena relating to the mind, as well as the progressive impairment of the vital forces, are to a considerable extent due to the insomnia. This is to be inferred from the fact that the delirium generally ceases after sleep has continued for several hours. Inability to sleep is an essential feature of the affection and the chief obstacle in the way of a favorable termination.

In general, the DIAGNOSIS is not attended with difficulty. The characteristics of the delirium render the affection easily recognizable when fully developed; moreover, the known habits of patients generally lead the practitioner to expect it. Tremulousness of the tongue and limbs, with vigilance, if the patient be a drunkard, denotes an impending attack. If now the patient complain of having remarkable visions or manifest illusions of the senses, the development is progressing. The cases in which the practitioner hesitates in coming to a diagnosis are those in which the habits are unknown. The affection may be developed in secret drinkers and in persons who are supposed to be perfectly temperate. I have known a person to be attacked who was supposed by his friends never to taste any alcoholic beverage. As the diagnosis implies intemperance, caution in forming an opinion in some cases is advisable, as well as prudence in expressing the opinion when formed. Of course it is a breach of medical ethics to reveal the character of the affection, save to the patient and, under certain circumstances, to his friends.

Excesses in drinking sometimes occasion a form of delirium differing, pathologically, from delirium tremens, but liable to be confounded with it. The delirium now referred to is an active delirium, characterized sometimes by violence or fury and sometimes by hilarity. It is a form of acute mania due to active cerebral congestion, conjoined with the immediate effect of alcohol on the brain. It is occasioned by a protracted paroxysm of drinking rather than by the habitual use of alcohol. This form of delirium has been distinguished as *delirium ebriosum.* It is accompanied by pain in the head, increased heat, throbbing of the carotids, and febrile movement; in short, by the symptoms of active cerebral congestion. It is of brief duration, ceasing when alcoholic stimulants are discontinued. It thus ceases under the circumstances which frequently occasion delirium tremens. It may be followed by delirium tremens. Aside from the discontinuance of stimulants, it claims the treatment indicated by active congestion of the brain—namely, bloodletting, purgatives, etc., measures not suited to the treatment of delirium tremens.

Finally, with respect to the diagnosis of delirium tremens, meningitis is excluded by the absence of cephalalgia, intolerance of light and sounds, throbbing of the carotids, febrile movement, etc. Acute gastritis, which may present some of the phenomena of impending delirium tremens, is to be excluded by the absence of vomiting, gastric pain, tenderness over the stomach, etc. Gastritis, however, is often present in delirium tremens. Typhoid and typhus fever are excluded by the absence of the diagnostic symptoms other than delirium of these diseases.

The TREATMENT of delirium tremens was formerly by bloodletting and other of the so-called antiphlogistic measures, under the belief that it involved either inflammation or cerebral congestion. These measures are now abandoned as not only uncalled for, but injurious. Within late years various curative methods of treatment have been advocated on the grounds of success; and in endeavoring to judge of their respective merits it is to be borne in mind that under favorable circumstances the intrinsic tendency of this affection is to recovery. It may be said of this affection, as of others tending in like manner to recovery, that a large proportion of recoveries cannot be cited in evidence of the curative efficacy of any particular method of treatment, but that a large proportion of fatal cases favors the presumption that the treatment was injurious. Excluding cases in which the affection occurs in connection with wounds, surgical operations, and accidents, or with other affections, and the cases in which it occurs in persons whose constitutions are shattered by intemperance, judicious management without therapeutical measures would, in the great majority of cases, prove successful. It does not follow from this statement that remedial measures are not often useful.

The great object of treatment is to procure sleep. Opium has been employed largely for this object. The statistical researches of John Ware showed that the fatality was greater under the opiate plan of treatment than when an expectant or eclectic plan was adopted, and that the fatality is proportionate to the excessive use of opium. Opium used indiscriminately and largely is undoubtedly injurious, but that its moderate employment is useful in certain cases is not to be doubted. The use of opium as advised by Stillé may be safely adopted. This author says: "The best mode of exhibiting the medicine (opium) is undoubtedly to begin with a quarter of a grain or its equivalent, and progressively to augment the dose by small and hourly additions until sleep is produced or a sufficient degree of tranquillity obtained." I have not infrequently found a full dose, repeated, if required, after intervals of several hours, to act very favorably. The remedy should never be pushed to the extent of inducing narcotism, and if on a fair trial it be not found to tranquillize, it should be discontinued.

Cases have been treated satisfactorily by simply continuing the use of alcoholic stimulants in moderate quantity. In general it is injudicious to discontinue entirely the use of stimulants so long as the affection continues. The time for breaking off the habitual use is after sleep has taken place and the patient is convalescent. Stimulants are to be given freely in cases in which the symptoms denote failure of the vital powers.

Tartar emetic in nauseating doses is useful in certain cases. This remedy, if not pushed too far, exerts a sedative influence, diminishes delirium, and disposes to sleep. It is suited to cases in which there is considerable constitutional vigor, and it should be very cautiously given to patients much enfeebled or in cases in which there is a tendency to prostration. Emetics have been advocated as curative, but the evidence of this is unsatisfactory, and they are now very rarely employed. They are objectionable on the score of perturbation. Cathartics are objectionable on the same score. These evacuant remedies have been employed under the idea that they eliminate the alcohol from the system; but the affection is due to the toxical effects of alcohol, not to its actual presence in the system; and, indeed, the attack often appears to proceed from a diminution of the amount of alcohol habitually present.

Cold affusions, the cold douche to the head, and the shower-bath are useful in some cases. They are of doubtful expediency if employed against the will of the patient. He should be induced to concur in the propriety of making trial of them. They may do harm to feeble subjects or if the powers of life be much depressed.

Digitalis has been advocated as a curative remedy. It was introduced by Dr. G. M. Jones of Jersey, England, who was led to ascertain its efficacy by a patient affected with delirium tremens having taken an ounce of the tincture by mistake. Subsequently he employed it in 70 cases with the loss of only 1 case. No practitioner would have ventured to give this remedy in the doses which experience has abundantly shown may be given without any unpleasant consequences in this disease. The dose advised by Dr. Jones is half an ounce of the tincture, which may be repeated, if required, after an interval of four hours. Many cases in which this plan of treatment has apparently proved efficacious have been reported. I have tried it in a few cases, in one of which it appeared to act like a charm, but in the others no curative effect was apparent.

The inhalation of chloroform in some cases tranquillizes the patient and induces sleep. This is a remedy to be employed with great circumspection. Its effect has been happy in several instances under my observation. In one case the effort to employ it excited such a degree of mania that I was led to desist. The hydrate of chloral may be employed. The use of this remedy calls for circumspection. The bromide of potassium, given in large doses, is a valuable remedy in this disease. This remedy is chiefly relied upon in a large majority of the great number of cases treated at Bellevue Hospital. Hyoscyamin has lately been employed with much success as an hypnotic in this disease.

As regards the measures which have been referred to, and others, they are to be employed according to circumstances proper to individual cases. Each of the measures is suited to some cases, and not to other cases. They are to be employed tentatively, the practitioner being guided by the effect in deciding whether to continue or discontinue them. Not infrequently they may be tried successively in the same case. In short, the appropriate treatment as regards these measures is expectant and eclectic, and they will be employed judiciously or otherwise according to the judgment of the practitioner.

The management in other respects is important. Quietude is to be enforced. Visitors, who are often actuated by curiosity, are to be excluded. The circumstances surrounding the patient should as far as possible be regulated with a view of promoting sleep. Much may be done by the exercise of tact in soothing the patient, combing the hair, bathing the face, resorting to warm pediluvia, etc. I have known muscular exercise carried to the point of fatigue to be useful. Alimentation is highly important. The patient should be encouraged as much as possible to take nutritious and easily-digested food.

It is the duty of the physician, after recovery from this disease, to inform the patient of its character, and to point out the inevitable consequences of the habits which have induced it; and the patient will be aided in an effort to emancipate himself from the slavery of intemperance by remedies and hygienic measures which tend to invigorate the body, thereby strengthening the mental powers.

A craving for alcoholic excitation is a result of habit. It is sometimes induced by dyspeptic disorders, mental depression, or a sense of prostration incident to various diseases, and it is sometimes a constitutional idiosyncrasy. The power to resist it is impaired in proportion as the faculties of the mind are weakened by indulgence. It can be overcome only by sufficiently long-continued total abstinence, together with measures to remove disorders and to promote mental occupation and invigoration of body and mind. There is no drug which has a special curative efficacy. Persuasion, the fear of consequences, and moral considerations are often unavailing. The unhappy slaves of intemperance should be considered as suffering from disease, and should be treated as patients until the morbid craving is extinguished by prolonged abstinence. In many instances effective treatment is practicable only in institutions established for this purpose.

The "opium habit" is to be regarded in the same light. Generally, instant and complete withdrawal of every form of opiate is perfectly safe and is the preferable course. Under judicious medical treatment and encouragement patients will be more likely to endure the intense suffering from the sudden disuse of opium than to persevere in a gradual diminution of doses during a period of weeks or months.

Hypochondriasis—Pathophobia.

Although hypochondriasis belongs in the list of disorders of the mind, the mental aberration is not regarded as amounting to insanity, and patients with this affection are not proper subjects for treatment at lunatic asylums. Cases are not infrequent, and they claim the services of the general practitioner. In view of these facts, it is appropriate to consider the affection as one of the neuroses. The name hypochondriasis—which from its derivation is supposed to have reference to morbid sensations sometimes felt in the hypochondriac regions—has very little significance as indicating the character and seat of the affection. The names pathophobia and nosophobia are much more expressive of the malady.

The characteristic feature of the affection is a morbid apprehension of either the existence of, or a liability to, some serious disease. In its mildest form it consists of a feeling of extreme delicacy of constitution or of a peculiar susceptibility to the causes of disease. Existing in this form, it leads to an anxiety concerning health and over-precautions for its preservation. In the severest form of the affection patients suffer from the conviction that they have an incurable malady and that life is in imminent danger. Different cases represent gradations between these extremes. The mental depression or despair which may be produced by a knowledge of the fact that a serious or fatal disease actually exists does not constitute hypochondriasis. The hypochondriac is the victim of a delusion with respect to his condition; and the mental suffering arising from this error of belief often exceeds that which would be likely to be experienced if the fancied disease really existed. Generally, the hypochondriacal delusion is accompanied by more or less mental depression, and often by marked melancholia, but in some cases patients are resigned and cheerful. Fancied diseases are sometimes the occasion of profound religious sentiment, calling forth the manifestations of pious resignation.

The affection is presented to the practitioner in different phases. In some cases the imagination becomes fixed upon a particular disease, and the patient's belief in the existence of the disease defies all efforts to remove it. Disease of the heart, pulmonary consumption, cancer, syphilis, tapeworm, softening of the brain, and diabetes are among the diseases which in different cases are supposed to exist. The physician may strive in vain by assurances and arguments to dispossess the mind of his patient of the delusion. In support of his conviction the patient sometimes not only exaggerates ailments which are present, but insists on the presence of symptoms which are wanting. The pertinacity and ingenuity with which in some cases patients hold to their faith and strive to convince the physician of its truth are remarkable. Not infrequently the conflict between the physician and patient continues until it becomes intolerable, and each is well pleased to break off the professional relation. Hypochondriacs frequently consult many physicians in succession, finding for a time greater attention and apparent interest in a new medical adviser; and, naturally enough in their mental condition, they become dissatisfied when the patience of the physician becomes exhausted and they have given up all expectation of converting him to their belief. The absurdity of the delusion in some instances is truly surprising. I have known two cases in which the patients, women of intelligence and good sense, for a long time could not be persuaded that they were not affected with cancer of the breast, although nothing was to be seen or felt to give color to the delusion. It is a noteworthy fact that hypochondriasis is not confined to the weak-minded. Persons of education and strong mental powers are as liable to the affection as those who are ignorant and of feeble intellectual capacity.

In another phase of the affection there is not a fixed belief in the existence of a particular disease, but a shifting of the delusion from one disease to another. The physician may succeed in convincing the patient of the non-existence of an imagined disease, but no sooner is this done than the patient's imagination runs upon another disease, and so divers diseases are in turn imagined, until at length the physician is discouraged and may give up the contest. Again, patients sometimes keep the house and take to the bed, complaining of ill-health, prostration, and various morbid sensations, without selecting any particular disease. With cases of bedridden patients who have no definite malady, but a host of subjective symptoms, all practitioners of much experience are familiar. Such cases occur much oftener among women than among men, and the phenomena embraced under the name of hysteria are generally more or less involved.

The CAUSES of hypochrondriasis are physical and mental. Some persons are constitutionally liable to morbid apprehensions in this direction. Whenever they are ill with any affection, their mental constitution leads to a sense of danger and to despondency as regards recovery. Not infrequently this state of mind exerts a depressing influence which may interfere seriously with the favorable progress of disease. Persons thus unhappily constituted are likely to exaggerate the permanent consequences of dissipated habits in early life, especially abuses of the sexual function. Delusions with respect to the latter are fostered by publications and lectures designed by unprincipled persons to produce this effect. Apprehensions on this score, often with little or no foundation, are a source of nefarious profit to a host of charlatans. If a person with a hypochondriacal predisposition either have had or imagine that he may have had syphilis, he is liable to fall into the belief of supposing that his system is irretrievably ruined. This delusion also is encouraged by empirics whose business is to prey upon the fears which they foster and excite. There is a class of wretched hypochondriacs whose minds are absorbed with the idea that they are the hopeless victims of the thoughtless errors of youth, and patients of this class, much as they are to be pitied, often tax severely the temper of the physician. It is to be added that the practice of masturbation, excessive sexual indulgence, and the intemperate use of alcoholic stimulants are undoubtedly sometimes involved in the causation, not merely by an effect upon the imagination, but by contributing to produce a morbid condition of the nervous system.

The reading of legitimate medical works is to be mentioned among the causes of hypochondriasis. Want of mental occupation contributes indirectly to the affection. Persons who have relinquished active pursuits often become hypochondriacs. Success in the acquisition of wealth, when it leads to a determination to forego labor and to enjoy leisure, may thus engender a far greater evil than poverty. On the other hand, the loss of fortune or want of success in the pursuits of life, and severe affliction of any kind, are sometimes concerned in the causation of this affection. These various causes may produce the affection in those who are not, as well as in those who are, constitutionally predisposed to it; but of course their efficiency will be marked in proportion as the predisposition is strong.

The morbid physical conditions often existing in cases of hypochondriasis, and more or less concerned in its causation, are various. Among these may be mentioned anæmia, neurasthenia, and dyspeptic disorders. The latter, there is reason to believe, are frequently, to a considerable extent, effects of the morbid condition of the mind.

The DIAGNOSIS of hypochondriasis must rest on an exclusion of the maladies which are apprehended or imagined and of any serious disease. The different organs of the body are to be interrogated by means of symptoms and signs; and it is obvious that the accuracy of the diagnosis will depend on the ability of the physican to exclude the affections to which the different organs are subject. Subjective symptoms are to be admitted with circumspection and a certain amount of distrust. The incongruity of the previous history and the present condition, as described by the patient, with the laws of any definite disease, exclusive of mental disorder, is often at once significant of the affection under consideration, or the subjective symptoms are incongruous with the physical disorders which may be associated with the hypochondriasis. From want of sufficient knowledge or skill to reach the diagnosis by exclusion practitioners are sometimes misled by the statements of patients, and thus contribute unwittingly to establish more firmly existing delusions.

In a certain proportion of the cases in which some serious disease is either apprehended or imagined the positive assurances of the physician suffice to dispel the delusion. In this way the physician who is confident in his ability to interrogate accurately the different organs of the body has the power of doing much good. The fear of the existence of some grave disease oppresses especially those who are dependent for the means of living upon a certain measure of health; and a decided opinion, in which they have confidence, is a great blessing to patients of this class.

In cases of confirmed hypochondriasis the first object with reference to TREATMENT is the removal of any supposed causes. Over-exertion, mental or physical, is to be avoided if possible; and sexual abuses, intemperance, and any violation of the laws of health are to be inquired into and reformed. Aside from the casual indications, the measures of treatment relate to both body and mind. Any coexisting physical disorders are to be remedied as far as practicable. Anæmia and dyspeptic ailments often claim remedial measures. In addition to the physical operation of remedies, they undoubtedly have a useful moral effect within certain limits. Patients often crave remedies, and it is important not to encourage this disposition; but it is often not judicious to withhold remedies altogether, even when the medicinal part of the treatment is comparatively unimportant. Alcoholic stimulants and opiates should not be prescribed, or if indicated for a temporary object they should be given with great circumspection.

The most important part of the treatment is that which relates to the mind. Arguments, assurances, and combined medical opinions may be brought to bear upon the delusions with more or less effect. It is, however, not always judicious to act upon a determination that the patient shall be at once reasoned out of the affection. It is not wise to attempt to dispel the delusions by ridicule or indifference. A fundamental part of the moral management is to divert the patient's attention from himself. This is to be effected by inducing occupation if the patient have been living a life of indolence, and by well-selected recreations if the patient be of industrious habits. It is more difficult to effect this in cases of hypochondriasis among women than among men. Change of scene and new associations are often effectual, and, wherever practicable, should be advised. With respect to the particular plans to be pursued in different cases, the habits, intelligence, education, and social position are to be considered in each case, and the success of the treatment will depend much on the judgment and tact of the physician.

SECTION SIXTH.

DISEASES AFFECTING THE GENITO-URINARY SYSTEM.

CHAPTER I.

CONGESTION OF THE KIDNEYS.—ACUTE AND CHRONIC BRIGHT'S DISEASE.

Active Congestion of the Kidneys.—Passive Congestion of the Kidneys.—Parenchymatous Degeneration of the Kidneys.—Fatty Degeneration of the Kidneys.—Acute Bright's Disease.—Chronic Bright's Disease.

A LARGE proportion of the affections of the genito-urinary system do not fall within the scope of this work. Excluding the affections peculiar to females, diseases of the male generative organs and of the bladder are to be excluded as belonging to surgical rather than to medical practice. The diseases which remain are chiefly those affecting the kidneys. Abnormal changes in the urine are highly important as denoting morbid conditions relating to the kidneys and the blood. These changes constitute important symptoms in various diseases. They are not to be considered as individual affections, and in general it suffices to notice them in connection with other symptomatic events which make up the clinical history of different diseases. In an important disease which, in the present state of our knowledge, it is convenient to regard as a urinary affection, it is certain that the abnormal change in the urine expressed by the name of the disease is merely a symptom. The disease referred to is *glycosuria* or *diabetes mellitus.* This disease will be included among the diseases to be considered in this section. The same statement may probably be applied to the disease distinguished as *diabetes insipidus*, and also to *endemic hæmaturia* and *hæmoglobinuria.*

Active Congestion of the Kidneys.

Active congestion of the kidneys may be due, *first*, to the action of irritants, and *second*, to vaso-motor paralysis. These two forms of active congestion are widely different from each other. The former, when of sufficient intensity to give clinical evidence of its existence, is almost invariably accompanied by inflammatory or degenerative changes in the kidneys; the latter has no tendency to lead to inflammation; the former is accompanied by the excretion of urine similar to that of certain forms of acute nephritis, the urine being albuminous, bloody, and usually diminished in quantity; in the latter the urine is abundant in quantity, non-albuminous, and of low specific gravity.

In the inflammatory variety of active congestion the kidneys are enlarged, of a dark-red color, engorged with blood, and ecchymosed. Certain drugs which exert an irritant effect upon the kidneys are usually assigned as the most important causes of this form of active congestion. Such drugs are cantharides, turpentine, oil of mustard, cubebs, copaiba, nitrate of potash, chlorate of potash, and carbolic acid. Of these drugs, cantharides, taken either internally or applied externally in the form of a blister, provokes the most marked urinary symptoms. In cantharides-poisoning the urine is diminished in quantity and may be suppressed; it is usually, but not always, stained with blood; it is albuminous, and it may contain hyaline and blood-casts as well as epithelial cells. A peculiarity noted in some cases has been the spontaneous coagulation of the urine. Gelatinous fibrinous clots, either pale or red, may form in the bladder and interfere with micturition, or similar coagula may form in the vessel after the urine is voided. It has been shown by experiments that a true diffuse nephritis is produced by cantharides. It is probable that an inflammatory condition likewise exists when similar symptoms follow the ingestion of the other drugs named. The treatment consists in removal of the cause, rest, and the administration of diluent and of mucilaginous drinks. Camphor in doses of two to five grains will often afford relief.

The existence of vaso-motor nerves of the renal arteries has been demonstrated by physiological experiment. These nerves are contained in the splanchnics. Paralysis of these nerves causes dilatation of the arteries in the kidneys and consequent afflux of a larger amount of blood to these organs. Some also assume the existence of vaso-dilator nerves of the kidneys. It is fully in harmony with the results of physiological experiment that this increased flow of blood through the kidneys is attended by the excretion of a large quantity of pale urine. We may assume that renal hyperæmia due to vaso-motor influence may be produced in man by causes acting upon the peripheral nerves as well as by those acting on the nerve-centres. We possess little positive knowledge as to this variety of renal hyperæmia in man. It has been inferred to exist, from the characters of the urine, in certain diseases of the brain. Hyperæmia of the kidneys from vaso-motor influence is assumed to exist in many cases of diabetes insipidus, of hysteria, and of emotional disturbance.

Passive Congestion of the Kidneys.

Passive or venous congestion of the kidneys is one of the characteristic events in the general venous congestion which attends certain pulmonary and cardiac diseases. Passive congestion of the kidneys may also be caused by thrombosis of the vena cava ascendens, but this occurrence is so rare that it does not demand further consideration here. Thrombosis of one of the renal veins has been observed more frequently, but in the cases hitherto recorded no notable congestion of the kidney has been observed. Passive congestion of the kidney, therefore, may be considered as almost invariably accompanied by general venous stasis.

Pulmonary disorders are less important than cardiac diseases in the etiology. In advanced emphysema and in certain cases of fibrous phthisis there is chronic congestion of the kidneys. The various cardiac diseases which lead to general venous congestion—valvular lesions, fatty degeneration, and myocarditis—are the most frequent and important of the causes. Mitral lesions may be especially emphasized in this connection. It will be remembered that the symptoms of venous stasis do not appear until the stage of non-compensation, and are intense in proportion to the failure of heart-power. It is also important to bear in mind that in proportion as the tension in the veins increases the blood-pressure in the arteries sinks.

The appearances presented by the kidneys after prolonged passive congestion are quite characteristic. The condition is sometimes called cyanotic induration of the kidneys. These organs are usually somewhat enlarged, their consistence is extremely firm, the capsule is non-adherent, and the surface is

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smooth. Upon section both the cortical and the medullary substance present a dark bluish-red color, the pyramids being darker than the cortex. The dark-red color and the firm, almost stony-hard, consistence of the kidneys are the most distinctive features. Upon microscopical examination the parenchyma and the interstitial tissue are usually unchanged. Fatty degeneration of the epithelium and increase of the interstitial tissue are found sometimes, but not as a rule.

Chronic passive congestion of the kidneys is accompanied by the symptoms of the primary disease. There are generally evidences of venous congestion in other parts of the body. Œdema of the lower extremities is usually present. The only symptoms which can be referred, with any certainty, to the condition of the kidneys are changes in the urine. The quantity of urine is diminished; its specific gravity is increased; its color is darker than normal; it contains a moderate quantity of albumen, and usually some red blood-corpuscles, a few small hyaline casts, and a few epithelial cells. The urine upon standing often throws down an abundant sediment of the urates. According to Bartels, the excretion of uric acid is greater than in health. The amount of albumen is not great, rarely reaching two parts in a thousand. The quantity of blood does not generally suffice to color the urine perceptibly. The small quantity of urine may be referred to the diminished rapidity with which the blood flows through the glomeruli. The albuminuria is attributed, by some writers, to increased blood-pressure in the glomeruli; and by others to changes either in the capillary walls or in the epithelium of the glomeruli in consequence of an insufficient supply of arterial blood. Passive congestion of the kidneys is never a cause of uræmia. The diminished excretion of urine may perhaps increase the general dropsy. The treatment is directed to the primary condition, and not immediately to the kidneys. The indication is to strengthen the force of the heart's action, thereby increasing the arterial tension and relieving the venous stasis. By the administration of digitalis the urinary symptoms are often improved, and they may entirely disappear as the power of the heart's action increases. The reader is referred to the article on Valvular Diseases of the Heart for a consideration of the treatment applicable to the disturbances of the circulation which cause the manifestations of venous congestion.

Parenchymatous Degeneration of the Kidneys, Fatty Degeneration of the Kidneys.

These degenerations occur in the different forms of Diffuse Nephritis, in connection with which they will be again mentioned. Many of the slighter grades of so-called catarrhal nephritis and of parenchymatous nephritis belong, in reality, to the category of these degenerations. The degenerations may occur independently of any inflammation of the kidney, in which case they are generally without much clinical importance. It is desirable not to include under the name of Bright's disease these degenerations when unattended by any inflammation.

The cortical substance of kidneys in the condition of parenchymatous degeneration or cloudy swelling is somewhat swollen and of an opaque grayish color. The surface of the organ is smooth and the capsule is easily detached. The consistence of the kidney is diminished. Upon microscopical examination the epithelial cells of the convoluted tubes are found swollen and filled with albuminous granules which, in considerable part, dissolve in acetic acid. As these cells are normally very granular, it is not easy to detect with the microscope the slighter grades of this form of degeneration. The macroscopical appearances are often more distinctive than the microscopical. (Vide

Part I. p. 53.) Excluding inflammation of the kidney, the most important causes of parenchymatous degeneration are infectious and febrile diseases and certain mineral poisons. Of the first group of causes may be mentioned pyæmia, septicæmia, diphtheria, and the exanthematous and the continued fevers; of the second group, phosphorus, arsenic, antimony, and the mineral acids. Parenchymatous degeneration also follows extensive burns and irritating applications to a large surface of the skin.

Fatty degeneration is often combined with parenchymatous degeneration (granulo-fatty degeneration). It is a usual sequence when the action of the causes named is long continued or intense. In well-marked cases of fatty degeneration the cortical substance is more swollen than in the preceding degeneration. The enlarged and pale cortex presents yellowish streaks and patches. Hemorrhages are usually present in the fatty degeneration of phosphorus-poisoning. The microscope shows in the epithelial cells of the convoluted tubes an accumulation of fatty molecules larger and more refractive than the albuminous granules of parenchymatous degeneration, from which they furthermore differ by their insolubility in acetic acid and their solubility in ether after treating the specimen previously with alcohol. Both in parenchymatous and in fatty degeneration the glomeruli are unchanged. The causes of fatty degeneration, in addition to those of parenchymatous degeneration, are anæmic conditions, especially pernicious anæmia and leucocythæmia; cachexiæ, especially chronic pulmonary tuberculosis; chronic alcoholism; and, according to Wilks and Moxon, diabetes. In old age and in general obesity a moderate fatty degeneration may be met with. Jaundiced kidneys are likely to be fatty. No attempt is made to discriminate between fatty infiltration and fatty degeneration of the kidney. Many of the causes of these degenerations are also causes of nephritis.

Parenchymatous degeneration of the kidneys is usually attended by a moderate albuminuria. As already remarked, this condition is held by some pathologists to be a mild form of acute Bright's disease or to be the first stage of some of the varieties of that disease. This form of degeneration is generally transitory, and it readily subsides as its causes disappear. It is not certain that the slight albuminuria so often present in simple parenchymatous and fatty degenerations of the kidney in reality depends upon these degenerative changes in the renal epithelium. It is perhaps referable in some other way to the primary condition which causes the degeneration. It is certain that extensive fatty degeneration of the kidney may exist, as in some cases of phosphorus-poisoning, without any albuminuria or other appreciable urinary symptoms. These observations justify the separation from Bright's disease proper of these non-inflammatory degenerative changes.

In all forms of jaundice of sufficient intensity hyaline casts are present in the urine, often without any albuminuria. It is uncertain whether the production of these casts have anything to do with changes in the renal epithelium.

Acute Bright's Disease—Acute Diffuse Nephritis.

Since the memorable researches of Richard Bright, published in 1827, diseases of the kidney characterized by albuminuria and general dropsy have been called Bright's disease. It will be seen that the renal changes which belong to the different forms of this disease are various, although not so many as the names which have been proposed for them. Not every case of albuminuria is to be considered as one of Bright's disease. We do not include under this name the renal changes which have already been considered in this section—namely, simple parenchymatous degeneration, fatty degeneration, and hyperæmia of the kidneys—although each of these alterations may form an important element in Bright's disease. These changes are attended either by moderate albuminuria or by none whatever; and if any dropsy be present, this is not referable, as a rule, to the alteration in the kidneys. By almost universal consent Bright's disease is regarded as a nephritis; that is, as an inflammation of the kidneys. This nephritis affects both organs, and is therefore described as diffuse, in distinction from the circumscribed renal inflammations, such as suppurative nephritis. The great majority of cases of Bright's disease are furthermore appropriately called diffuse nephritis, on the ground that all the constituents of the kidneys—namely, the interstitial tissue, the Malpighian tufts, the uriniferous tubes, and the blood-vessels—are involved in the inflammatory process. But the term "diffuse" must not be understood as implying that the morbid process affects the kidney tissue uniformly. Some parts of the kidney are always more diseased than others. Bright's disease is divided into an acute and a chronic form.

Acute Bright's disease has received various names, such as parenchymatous nephritis, acute tubal nephritis, acute desquamative nephritis, catarrhal nephritis, croupous nephritis. These names are all objectionable, as they imply theories concerning the nature of the disease which are either unproven or erroneous. The designation acute Bright's disease is the simplest, and involves no doubtful hypotheses. If it be desired to employ an anatomical term, acute diffuse nephritis is the one least objectionable.

Anatomical Characters.—The morbid appearances in acute Bright's disease vary considerably in different cases. The kidneys may present so little variation from their normal aspect that the disease can be recognized only by microscopical examination. As a rule, however, these organs are enlarged, moist, and somewhat softened. The surface is smooth, and presents red patches alternating with opaque grayish or yellowish-white portions. The red patches correspond to hemorrhages or to spots of congestion, and the pale portions are either relatively or absolutely anæmic. Upon section the swelling is seen to involve chiefly the cortex, which presents the same mixture of colors. Red points corresponding to congested Malpighian bodies, or more frequently to hemorrhages, are observed. The Malpighian bodies may appear swollen and abnormally pale. In some kidneys the hyperæmic and hemorrhagic portions predominate, and in others the pale opaque appearance prevails. It is thus possible to distinguish red kidneys and pale kidneys of acute Bright's disease, and between these types there is every possible variation. In the majority of cases of acute Bright's disease the kidneys are hyperæmic. If the disease approach a chronic stage, the hyperæmia usually gives place to a whitish or yellowish color, but some hemorrhages usually remain.

Upon microscopical examination the alterations are sometimes found to be astonishingly slight, even when during life the urine was quite albuminous and contained blood and casts. In these cases, which are generally mild and do not terminate fatally from the kidney disease, the inflammatory alteration affects chiefly the capillaries of the glomeruli. These altered capillaries allow the escape of an albuminous fluid and the passage of red and white corpuscles through their walls. These inflammatory products are exuded into the space between the glomerulus and Bowman's capsule, whence they pass into the tubes and escape with the urine. There may be cloudy swelling of the epithelium of the Malpighian tufts and of the convoluted tubes. The interstitial tissue presents no inflammatory changes.

When death occurs from the renal affection itself the changes are generally more marked. Here, too, the affection of the glomeruli is of great importance. To the naked eye the Malpighian bodies often appear swollen and pale.

The capillaries of the glomerulus frequently contain desquamated and proliferating endothelial cells mingled with white and some red blood-corpuscles. Both the epithelium of the glomerulus and that lining the capsule of Bowman swells up, proliferates, and, often with an admixture of white blood-corpuscles, forms a crescentic mass of cells filling up the space between the compressed glomerulus and the capsule. These changes constitute the affection called glomerulo-nephritis, which is an important element in many, and almost the sole alteration in some, cases of acute Bright's disease. A similar accumulation of leucocytes and of desquamated endothelium may occur in the intertubular capillaries. The epithelium of the convoluted tubes is swollen and filled with an abnormally large number of albuminous, mixed with some fatty, granules. Colloid drops, granular material, and desquamated and disintegrating epithelial cells are found in some of the convoluted tubes. Hyaline casts are found here and there in the convoluted tubes, and in still larger number in the loops of Henle and the collecting tubes. Emigrated white blood-corpuscles are found not only in the tubes and the Malpighian bodies, but also in the interstitial tissue. In the latter situation they are generally collected in groups, especially around the veins and the Malpighian bodies. In most cases of acute Bright's disease of any severity hemorrhages are present, partly in the intertubular tissue, but chiefly within the capsules of Bowman and within the convoluted tubes.

It is not possible to draw a sharp line of demarcation between the inflammatory and the purely degenerative changes in the kidney. The same causes may produce both changes. As has already been remarked, in most cases of slight or moderate albuminuria attending various infectious diseases the sole change in the kidney is a parenchymatous or fatty degeneration of the epithelium. These cases are classified by many writers as parenchymatous nephritis. If, however, the symptoms, especially the changes in the urine, are during life sufficiently severe to warrant the diagnosis of Bright's disease, it is believed that in addition to these degenerative changes in the epithelium actual inflammatory lesions are present in the blood-vessels, particularly in the glomeruli.

Anasarca and serous accumulations in the pleural, pericardial, and peritoneal cavities are usually met with to a greater or less degree in the bodies of those dead of acute Bright's disease. Sero-fibrinous pleuritis and pericarditis are not infrequent. Pneumonitis, both in the lobar and in the lobular form, is one of the recognized complications. Increase of the cerebro-spinal fluid with cerebral œdema, or even with meningitis, is likewise to be mentioned in the enumeration of complications.

CLINICAL HISTORY.—Subcutaneous œdema is a very frequent but not a constant symptom. It is usually the first symptom pointing to the existence of this affection. The œdema is generally first observed on the face, particularly on the eyelids and around the eyes, but it is speedily apparent in the lower extremities, and sometimes occurs first in the latter situation. Coincident with the appearance of the dropsy is more or less febrile movement, frequently preceded by chills or shiverings, together with thirst, anorexia, pain and tenderness in the loins. Pain and tenderness over the kidneys are, however, rarely marked and are often wanting. Vomiting is a frequent symptom at the beginning of the affection. The skin is dry and the countenance becomes pallid. The dropsy increases and becomes general, varying considerably in amount in different cases. In some cases the anasarca is great, the limbs, scrotum, penis, or external labia becoming enormously swollen. In other cases it does not exceed a moderate amount. More or less dropsical effusion usually takes place into the peritoneal and the pleural cavity. Hydrothorax sometimes occurs to such an extent as to occasion great suffering from dyspnœa and to endanger life.

The examination of the urine is essential for the diagnosis of acute Bright's disease. The quantity is usually diminished, and it may be very scanty. Suppression sometimes occurs. The specific gravity generally exceeds that of the urine in health. The total quantity of urea excreted in twenty-four hours is much diminished. The urine contains albumen[1] in considerable, and frequently in great, abundance. The amount of albumen is usually between ¼ and 1 per cent. by weight, but it may exceed this proportion. In some cases the urine has a reddish-brown, smoky appearance from the presence of blood. A certain number of red blood-corpuscles are present in nearly all cases, although not always in sufficient number to color the urine. It is possible to distinguish two types of urine in acute Bright's disease—the one bloody, and the other free from blood. As a rule, in this disease a considerable sediment forms in the urine. The microscopical examination of this sediment shows red and white corpuscles in varying proportion; and the one variety may be abundant in the absence of the other. The red corpuscles may appear normal or as clear pale rings, or they may be shrunken. Disintegrated red corpuscles and granules of hæmatoidin may be observed. Epithelium of the urinary passages and of the kidneys may be present. Granular matter and micrococci are also among the frequent constituents of the sediment. The most characteristic objects are cylindrical bodies known as casts. These are formed in the uriniferous tubes of the kidney. Casts vary between $\frac{1}{1000}$ and $\frac{1}{500}$ of an inch in diameter. A number of varieties are distinguished. Those which belong to acute Bright's disease are blood-casts, epithelial casts, and hyaline casts. Granular casts and fatty casts indicate a more advanced change in the kidneys than usually belongs to acute Bright's disease; but in some cases of the acute disease granular, fatty, and even waxy casts are found in the urine. Hyaline casts are pale cylinders with delicate contours, and are so transparent that an inexperienced observer readily overlooks them. The addition of some coloring agent, such as iodine or eosin, is of assistance in searching for them, as they readily stain with most coloring matters. Their chemical composition is not known. They are often described as fibrinous, but they do not conform to the reactions of fibrin. They are probably of an albuminous nature. They vary in size. Small hyaline casts are those most frequently met with in the acute form of the disease. Blood-corpuscles, epithelial cells, and granular material are often attached to these casts. Epithelial casts are composed of desquamated epithelial cells of the renal tubes. Some of the so-called epithelial casts are composed of white blood-corpuscles. Blood-casts are made up of red blood-corpuscles. These last two forms of casts often possess a hyaline basement-substance to which the cells and corpuscles are attached. There may be some oil-globules in the sediment free or attached to the casts or contained in cells. In the majority of cases there are many casts, but sometimes they are so scanty as to require careful search for their detection.

Coma and convulsions occur in a certain proportion of cases. These are symptoms of uræmia. Owing to an insufficient elimination by the kidneys of urinary constituents, these may accumulate in the blood to an extent sufficient for the manifestations of their toxical effects upon the brain. Vomiting and purging may precede, or occur without the cerebral symptoms of uræmia, being due to a vicarious elimination of urea through the gastro-intestinal mucous membrane. Epistaxis and pruritus may occur in uræmia. Impaired vision and amaurosis are among the effects of uræmia occasionally observed. Blindness, which may be complete, when it occurs in acute albuminuria is generally sudden; but it is often of temporary duration, and recovery is complete. The ophthalmoscope, under these circumstances, shows no morbid appearances within the eye. The morbid condition is central. It is otherwise, as will be seen, in amaurosis occurring sometimes in the acute affection, but oftener in cases of chronic Bright's disease. The amaurosis which is not associated with changes appreciable with the ophthalmoscope is called uræmic amaurosis. It occurs especially in cases of acute albuminuria developed in connection with scarlatina and pregnancy.

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[1] Both serum-albumen and globulin are usually present in combination in albuminuria, the former predominating. Other forms of albumen (paralbumen, hemialbuminose) and peptones may be present, but it does not appear that any special diagnostic importance attaches to these latter forms of albumen.

Complications considered as occurring sufficiently often to show not merely coincidence, but a pathological connection with the affection, are bronchitis and inflammation of serous membranes, especially pleuritis and pericarditis. Pulmonary œdema is an occasional complication.

In the course of the affection the symptoms may present considerable variation on different days. The quantity of urine, the amount of albumen, etc. vary, and so with respect to the œdema, the effusion into the cavities, and the febrile movement. If uræmic effects and important complications do not occur, the affection continues for a period varying between a few days and two months, the average duration being about four weeks. The approach of convalescence is denoted by a notable diminution or disappearance of the dropsy, cessation of febrile movement, return of appetite, and an abundant secretion of urine. Albuminuria usually continues, but in a lessened degree, after the dropsy has disappeared. At length the urine becomes normal and casts disappear from the sediment. The latter sometimes continue to be found for some time after the albumen has disappeared. The affection may end in complete recovery or it may eventuate in a chronic affection. The latter is rare. As a rule, if the patient be not cut off by uræmic poisoning or the complications which are liable to occur, the recovery is complete and permanent.

PATHOLOGICAL CHARACTER.—As both kidneys are diffusely inflamed in acute Bright's disease, it is to be inferred that the irritant which excites the inflammation is contained in the circulating blood. In many cases, as in the acute Bright's disease secondary to toxic influences (cantharides, turpentine, etc.) and to infectious diseases, the relation between morbid blood-changes and the local affection is clear. In other cases, as when the disease follows exposure to cold, this relation is obscure. The kidneys are peculiarly exposed to the action of noxious substances, as it is mainly through these organs that these substances are excreted from the body. As it is through the glomeruli that the water and doubtless many of the solid constituents of the urine are excreted, it is believed that inflammatory irritants act first of all upon these structures, and in the mildest cases of the disease only upon them and the renal epithelium, with which also irritants contained in the urine must come into intimate contact. In more severe cases all of the constituent elements of the kidney share in the inflammatory process. The diminished quantity of urine in acute Bright's disease may be explained by the lessened blood-pressure and the slow circulation in the glomeruli, these changes in the vascular system being incident to all acute inflammations. The red and white corpuscles found in the kidney and in the urine are the natural products of inflammation. The red corpuscles doubtless escape, at least in part, by diapedesis (p. 27). The albuminuria may be referred partly to inflammatory alterations in the walls of the renal capillaries, and partly to changes in the epithelium, particularly of that covering the glomeruli. Changes in the blood pressure may also be concerned in the causation of albuminuria.[1] As to the mode of production of hyaline casts, there is discrepancy of opinion. According to one view, they are formed from altered epithelial cells which have coalesced with each other; according to a second, they are produced by secretion of hyaline material from the epithelial cells; according to a third, and the most probable view, they result from a coagulation of albuminous material which has escaped from the blood-vessels into the tubes.

The most important of the effects produced by the renal disease upon the general system are dropsy and uræmia. The dropsy is usually referred to the hydræmia which results from the withdrawal of albumen from the blood and the diminished excretion of water. The relation between hydræmia and general dropsy has been discussed in Part I. p. 63. This explanation of the dropsy is not satisfactory in all cases. There is reason to believe that in some cases the anasarca is best explained upon the assumption that some alteration exists in the walls of the cutaneous and subcutaneous vessels, which renders them more permeable to the passage of albuminous fluids. Cohnheim assumes this alteration to be of an inflammatory nature, and to be produced often by the same causes which excite the nephritis.[2] The uræmia is the result of the accumulation of urinary constituents in the blood. This subject has been considered in Part I. p. 72.

CAUSATION.—This affection may occur at any period of life, and even young infants are not exempt from it. It occurs oftener in males than in females. As already stated, in the majority of cases it is a sequel of scarlatina. It may occur during the progress of scarlatina or follow directly the stage of desquamation, but it is most likely to occur in the second or third week after the date of convalescence. It is an important question whether this sequel proceed exclusively or chiefly from an agency pertaining intrinsically to scarlatina, or whether it depend on extrinsic causes, such as the action of cold. It occurs in cases in which the utmost care is taken to place the patient beyond the agency of extrinsic causes, but it is probable that the latter are frequently involved. It may be developed in a person much exposed to the scarlatinous infection without any affection of the throat or an eruption, the person having already experienced an attack of scarlatina many years previously. I have notes of several cases exemplifying the facts in this statement.

Exclusive of the cases in which it is a sequel of scarlatina, it occurs in various other pathological connections. It is an occasional sequel of diphtheria and of epidemic cholera. It sometimes occurs during the development of pulmonary tuberculosis, in the course of articular rheumatism, in syphilis, in cases of typhoid and typhus fever, yellow fever, erysipelas, relapsing fever, acute lobar pneumonia, acute yellow atrophy of the liver, pyæmia, septicæmia, endocarditis, dysentery, carbuncles and suppurative processes in different situations, smallpox, and measles. Albuminuria and general dropsy occurring in pregnancy depend, in a certain proportion of cases, on this affection, but in many of these cases changes in the kidney are very slight or entirely absent. As a primary affection it occurs especially in persons addicted to intemperance. In these cases it is sometimes difficult to say how much causative agency is to be attributed directly to the action of alcohol and how much to the exposure incident to intemperance. It is observed not infrequently to become developed after lying on the ground in a state of intoxication. In a case which came under my observation it followed excessive indulgence in beer without any exposure to cold. It is sometimes attributable to exposure to cold irrespective of alcoholic indulgence. It may be caused by extensive burns. Other causes are cantharides, turpentine, phosphorus, carbolic acid, iodoform, the mineral acids, arsenic, the nitrate of silver, lead, and mercury, taken internally. Finally, it may be mentioned that acute exacerbations are not uncommon in chronic Bright's disease. It is thus seen that with regard to etiology most cases may be distributed into three groups: 1st, those in which the disease is caused by toxic agents; 2d, cases in which it is incident to infectious diseases; and 3d, cases in which it is referable to causes acting on the skin, as cold and burns.

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[1] Writers are far from being agreed as to the causation of albuminuria. Increased blood-pressure in the glomeruli has usually been considered an important cause. Runeberg has advocated the view that the cause is in all cases diminished pressure. The truth seems to be that the blood-pressure is not an important factor in the production of albuminuria. Diminished rapidity of the circulation in the glomeruli is considered by many to be an efficient cause. The view advocated by Cohnheim is that the usual cause of albuminuria is to be found in changes in the epithelium covering the glomeruli. This view is consistent with the preceding, for slow circulation produces alterations in these cells by depriving them of their proper supply of nutriment. The real difficulty is to explain why albumen is not present normally in the urine. All other capillaries in the body, so far as known, allow an albuminous fluid to transude under normal circumstances. The walls of the glomerular capillaries differ from those of other capillary blood-vessels in the fact that they are not composed of distinct endothelial cells; at least such cells cannot be demonstrated by staining with nitrate of silver. Perhaps structural peculiarities in these walls may explain the absence of albumen in normal urine.

[2] This view must be considered hypothetical. It is elaborated by Cohnheim in the second volume of his *Allgemeine Pathologie*, Berlin, 1882.

DIAGNOSIS.—The existence of albuminuria is not, in itself, adequate proof of either the affection under consideration or of the chronic affections of the kidneys which remain to be considered. Albumen may be present in the urine in a small or moderate quantity, and for a brief period in the course of a great number of diseases. It is found not infrequently in the urine of persons who are apparently in perfect health. Munn found albuminuria in the proportion of 11 per cent. of persons applying for life insurance, this being the only evidence of any disease.[1] In general, the diagnosis is easily made, being based on the occurrence of anasarca developed rapidly, accompanied with febrile symptoms, and an examination of the urine generally showing albumen in abundance. Dropsy is an early symptom in the great majority of cases, but it is sometimes wanting. The diagnosis is then to be based on symptoms pertaining to the urine in conjunction with general symptoms. In addition to the presence of albumen in the urine, the quantity is usually notably lessened; it frequently has a smoky or sooty appearance from the presence of red blood-corpuscles, and it is sometimes distinctly bloody. The sediment of the urine, examined microscopically, is found to contain, in greater or less abundance, the casts which have been described. The presence of these, in conjunction with other characters pertaining to the urine and the general symptoms, render the diagnosis positive. Etiological connections are also to be taken into account in the diagnosis. It is to be borne in mind that acute Bright's disease not infrequently occurs as an intercurrent affection in the course of the chronic form of the disease.

PROGNOSIS.—The danger in cases of this affection is from uræmia and the complications which are liable to occur. Coma and convulsions always denote imminent and great danger to life, but recovery may take place notwithstanding these effects of uræmia. Pleuritis, pericarditis, peritonitis, meningitis, and pneumonitis, developed in the course of the affection, are likely to prove fatal. Œdema of the lungs may prove a cause of death. Dropsical effusion into both pleural cavities may take place to such an extent as to destroy life

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[1] Vide article by Dr. John Munn in the *N. Y. Med. Record*, March, 1879.

An instance of death from this cause has not fallen under my observation, but I have repeatedly met with cases in which the dyspnœa seemed to denote imminent danger to life.

Exclusive of uræmia and serious complications the prognosis is favorable. The affection does not tend to disorganize the kidneys. It is a self-limited affection, seldom continuing more than two months, and in some cases ending in from one to two weeks. It very rarely assumes a chronic form. Cases of the chronic affections which remain to be considered, as a rule, do not originate in an acute attack.

Uræmic poisoning is to be apprehended when the urine is scanty; in other words, when the quantity of urea eliminated by the kidneys is small. Suppression of urine lasting twenty-four or forty-eight hours places the patient in imminent danger, and if it continue is a fatal symptom. Danger from uræmia is not passed, although the albumen may have disappeared from the urine, if the sediment still contain casts. I have known fatal uræmic convulsions to occur under these circumstances.

TREATMENT.—The treatment embraces the following leading objects: 1. Diminution of the intensity of the renal inflammation, promotion of resolution, and restoration of the excretory function of the kidneys. 2. Diminution or removal of dropsical effusion. 3. Elimination of urea through the skin and gastro-intestinal mucous membrane if uræmia exist or be threatened.

With reference to the first of these objects, warmth and rest of the surface are important. The patient should keep the bed in order to maintain both warmth and rest. The diet should be unstimulating. Water and other bland liquids should be allowed as freely as the thirst dictates. Dry cupping over the loins is the best method of revulsion or counter-irritation, and if the patient be not feeble or anæmic the scarificator may be employed. General bloodletting is advisable only in cases in which patients are full-blooded and attacked when in good health. As a rule, after scarlatina, in intemperate persons, and whenever the system is enfeebled from any cause, this measure is inadmissible. Fomentations over the region of the kidneys by means of moistened cloths covered with oiled muslin and several thicknesses of flannel or the spongio-piline are useful. Saline laxatives are useful by way of depletion.

With reference to the dropsy, either saline cathartics or the more active hydragogues are indicated in proportion to the amount and situation of the dropsical effusion. In cases in which suffering and danger are incident to hydrothorax, the fluid may be withdrawn by aspiration. Elaterium is the most prompt and reliable hydragogue. I have repeatedly obtained, by means of this remedy, effectual relief in a few hours when the pleuritic effusion was sufficient to occasion alarming dyspnœa. Gamboge, podophyllin, jalap with the bitartrate of potassa, and sulphate of magnesia or soda are efficient hydragogues. Diuretics are not to be relied upon, and the kidneys will not be likely to respond to them. Moreover, they are considered by some as inadmissible because their action on the kidneys may increase the inflammation. They are, however, recommended as safe and frequently useful by others. A rational indication for their use is afforded by the fact that the renal tubes are obstructed; and a free secretion of urine will be likely to remove the obstruction by washing away the contents of the tubes. So far as my experience goes, digitalis and the saline diuretics do no harm and are often useful.

Measures for the vicarious elimination of urea are indicated whenever the diminished quantity of urine and its low specific gravity show the elimination by the kidneys to be greatly deficient. By timely meeting this indication uræmic poisoning may be forestalled. Of course measures for this object are urgently indicated when symptoms denoting uræmic poisoning are present. Hydragogue cathartics are the most efficient, and next to these the hot-air bath. The pilocarpine, given hypodermically, in some cases produces profuse perspiration and is a potent eliminative remedy. It may be conjoined with the use of the hot-air bath. Both hydragogues and sudorifics are to be employed in alternation if uræmic coma or convulsions have occurred. If the indication be not urgent, salines and sudorific remedies may suffice. An eligible sudorific remedy is the liquor ammoniæ acetatis in half-ounce doses thrice daily. The statement concerning the use of diuretics with reference to dropsy of course is equally applicable to their use for the elimination of urea. They are not to be relied upon for the elimination of urea when uræmia exists or is threatened. Under other circumstances diuretics may be employed, selecting those which do not act as irritants of the kidneys. The bitartrate of potassa, the infusion of digitalis, and the decoction of broom are eligible diuretics. With reference to a diuretic effect pure or distilled water should be taken as a drink as freely as possible without inconvenience. Dickinson says: "Of all diuretics, water is the best;" and my own experience has led me to think that this statement is not too strong.

Uræmic coma and convulsions call for measures of treatment in addition to those which are eliminative in their operation. Venesection may be employed if not contraindicated by the condition of the patient exclusive of the uræmic symptoms. For the arrest of convulsions, either chloroform inhalation or the chloral hydrate given by the mouth or rectum is indicated. Morphia may be administered by subcutaneous injection, but not in toxic doses. These are palliative measures to be pursued until elimination has been effected by hydragogues and sudorifics or as long as the coma and convulsions continue.

During convalescence care as regards diet, exercise, and exposure to cold is important. Tonic remedies, and especially preparations of iron, are generally indicated.

My clinical records furnish illustrations of complete recovery from this affection when the dropsy was excessive, when life was threatened by hydrothorax and œdema of the lungs, and also after the occurrence of uræmic coma and convulsions.

Chronic Bright's Disease—Chronic Diffuse Nephritis.

It is not easy to make a convenient and correct classification of the different forms of chronic Bright's disease. In different cases of this disease the kidneys present a great variety of morbid appearances as regards size, color, character of the surface, etc. Kidneys affected with chronic Bright's disease may be small, of normal size, or enlarged; they may be red, yellow, white, or mottled; their surface may be smooth or granular; waxy degeneration exists in some and not in other cases. The large kidneys usually are pale in color and of smooth surface, and the small kidneys are generally red or mottled and of granular surface; but the exceptions to these rules are many. There are three modes of classification which deserve special mention. There is, first, the classification of Frerichs, adopted in its leading features by Grainger Stewart. This divides Bright's disease into three stages: *first*, a stage of inflammation; *second*, a stage of degeneration; *third*, a stage of atrophy. This classification is founded on theory, and is opposed by clinical and pathological observations. It is certain that in the majority of atrophied kidneys the disease is chronic from the beginning, and there is no reason to suppose that the condition of these kidneys is preceded by either of the other stages;

nor are most cases of the so-called second stage ushered in by an acute attack. There are cases of Bright's disease which pass through the three stages of Frerichs, but this sequence of events is so rare that it cannot serve as a basis of classification. This division of Bright's disease into stages is generally abandoned.

Another classification is based on a view wholly opposed to the preceding. Bright's disease, according to this second view, is a general name for a number of independent diseases. This doctrine is advocated by many English writers, and since Bartels' work on diseases of the kidney it has been widely adopted in Germany. It is without doubt more generally accepted at the present time than any other classification. The distinct affections which are thus recognized as included under the name Bright's disease are: 1, acute Bright's disease; 2, chronic parenchymatous nephritis; 3, chronic interstitial nephritis; 4, waxy degeneration of the kidney. It is admitted that these affections are often combined with each other. This classification is in many respects a convenient one. It is certainly proper to separate acute from chronic Bright's disease; but on pathological as well as on clinical grounds there are serious objections to the sharp distinctions drawn in the foregoing classification between parenchymatous nephritis, interstitial nephritis, and waxy degeneration. In so-called chronic parenchymatous nephritis there exist interstitial changes of the same nature as those found in chronic interstitial nephritis; and in the latter there are also parenchymatous changes. There is no proof of the assertion that the disease begins in one case in the parenchyma and in the other in the interstitial tissue of the kidney. Waxy degeneration of the kidney may occur without inflammatory alterations, but in that case the degeneration is not extensive and is of little clinical importance. The waxy kidney is, in the great majority of cases, accompanied by a true nephritis. In many cases usually described as chronic parenchymatous nephritis there is waxy degeneration in addition to the nephritis.

A third classification is the one adopted in this work. It embraces all of the different forms of chronic Bright's disease under the name chronic diffuse nephritis. This classification recognizes the fact that in all of these forms there are both interstitial and parenchymatous changes. It does not consider the preponderance of parenchymatous alterations in one case and of interstitial alterations in another as a satisfactory reason for distinguishing a parenchymatous from an interstitial nephritis, especially as there may exist every possible degree of combination between these two changes.

It has been fully demonstrated that certain morbid conditions of the kidney in chronic diffuse nephritis are associated with characteristic clinical phenomena. These different morbid conditions are perhaps best designated by indicating the gross appearances of the kidneys in which they are present, as, for example, the small granular kidney, the large waxy kidney, etc. This method of division has disadvantages, but is perhaps as good as any which our present knowledge enables us to make. Whether the varieties of chronic Bright's disease are to be regarded as distinct affections or as different manifestations of one and the same disease is, in a measure, a matter of individual judgment. If chronic diffuse nephritis embraced only typical cases of large white kidney on the one hand and typical cases of small granular kidney on the other hand, there would be no impropriety in regarding these as distinct diseases; but there is every grade of transition between the large and the small kidneys. There are large kidneys with the clinical history of the small, and *vice versâ;* and there are large kidneys which, except as regards size, may present all the pathological changes of the small. For these and other reasons which might be adduced it is better to regard the types of diseased kidney which are to be described not as independent affections, but as different manifestations or varieties of the same disease—namely, chronic diffuse nephritis.

The two influences of greatest importance in determining the character or type of kidney found after death from chronic Bright's disease are the duration of the disease and the presence or absence of waxy degeneration. When the disease has lasted for several years, it is almost certain that the small granular kidneys will be found after death. When the affection terminates in less than a year from the onset (a point by no means easy in all cases to determine), some form of large kidney will usually be found on post-mortem examination. The waxy kidney, however, even when of several years' duration, is, as a rule, large and pale. It may, however, be small and granular. It must not be inferred from the fact that the small kidneys run a more prolonged course than the large that the former represent a late stage of the latter. In the majority of small kidneys the process of contraction or atrophy is to be considered as in operation from the onset of the disease.

The three main forms of chronic diffuse nephritis are: 1. The simple large kidney (red, mottled, or white); 2. The small granular kidney; 3. The waxy kidney. The waxy kidney is usually enlarged. It is therefore convenient to consider two forms of large kidney—one with and the other without waxy degeneration. Mention will also be made of the occurrence of waxy degeneration in small kidneys.

I. *The Large Kidney without Waxy Degeneration.*—This form is less frequent than either the large waxy kidney or the small granular kidney. It is the variety met with in the rare instances in which acute Bright's disease passes into the chronic stage. It is not, however, generally preceded by the acute disease. It runs, as a rule, a more rapid course than any other variety of chronic diffuse nephritis, its duration being rarely more than a year and a half. It presents two modifications according to the color of the cortical portion of the kidney. In one the cortex is red or, more usually, mottled with red and white (large red or mottled kidney). In the other the cortex is pale (large white kidney). The kidneys are enlarged, less frequently of normal size (a normal-sized kidney is reckoned among the large in distinction from the small kidneys). The surface is smooth. The capsule is easily detached. The swelling affects chiefly the cortex, which may present many hemorrhagic and congested portions, with opaque whitish or yellow parts, or may be almost uniformly of a pale yellowish-white color, with only here and there a hemorrhage or a congested patch, or even without any hemorrhages. In not a few cases the surface of the kidney is granular and the capsule is adherent.

Upon microscopical examination small hemorrhages are found in abundance in the red or mottled kidneys, and, although diminished in number, they are usually but not always present in the pale kidneys. The extravasated blood may be in the intertubular tissue, but is more frequently within Bowman's capsules and in the convoluted tubes. The changes in the interstitial tissue are in the form of more or less circumscribed patches scattered throughout the cortex. In these patches the connective tissue between the tubes and around the Malpighian bodies is increased. This new tissue may be rich in cells and indistinctly fibrillated, or it may be poor in cells and fibrous. The epithelium lining the tubes surrounded by the new connective tissue is generally atrophic, and the diameter of these tubes is diminished. In some places the tubes may have entirely disappeared. The glomeruli may be shrunken and fibrous, and the new tissue may be developed, especially around the altered glomeruli. Some of the glomeruli present the lesions of a glomerulonephritis as described in the preceding article (p. 866). There is often a growth of new connective tissue in the walls of the arteries, presenting itself frequently in the form of an obliterating endarteritis. In addition to this development of new connective tissue combined with atrophy of the tubes, the interstitial tissue may present here and there an infiltration with small round cells and with fatty molecules. Outside of the patches of new connective tissue the convoluted tubes, instead of being atrophic, are frequently dilated. In these dilated tubes the epithelium is generally swollen and contains molecules of fat. In other places no change may be noticeable either in the tubes or in the interstitial tissue. The lumina of the tubes contains also blood-corpuscles, hyaline casts, fatty molecules, and granular matter.

In the large kidneys, in distinction from the small granular kidneys, the changes in the epithelial cells (that is, the parenchymatous and the fatty degenerations) exceed in extent the interstitial alterations. Whether or not there be a form of chronic Bright's disease in which the parenchymatous and fatty degenerations constitute the sole changes is a matter of dispute. It is certain that in the great majority of cases there are interstitial alterations, as described, in addition to the changes in the tubes. It is important to observe, however, that both the parenchymatous and the interstitial changes are scattered in patches throughout the cortical portions of both kidneys, and are not uniformly diffused; also that the most extensive swelling and fatty degeneration of the epithelium are found outside of the patches of new connective tissue, in which, on the other hand, the epithelial cells are usually small and atrophied. It is evident from the description that even in large smooth kidneys there may be (microscopically) atrophied portions resembling those in the small granular kidneys, but not so abundant. This atrophy, with increase of fibrous tissue, is evident to the naked eye in those large kidneys which present a granular surface with adherent capsule. These and other considerations which will appear in the course of this article render it clear that, important as it is to distinguish certain general types or varieties of chronic diffuse nephritis, it is not desirable to separate them from each other by sharp boundary-lines. The pale kidneys differ from the red or mottled kidneys chiefly in a greater amount of anæmia and of fatty degeneration.

A considerable number of the large, non-waxy kidneys of chronic Bright's disease are accompanied by hypertrophy of the left ventricle or of the whole heart. Hypertrophy of the heart does not usually develop in connection with the large kidneys when the renal disease occurs in patients much enfeebled, as by phthisis, syphilis, chronic suppurations, etc. It is in connection with such wasting diseases that the greatest number of the large white kidneys are developed. There is reason to believe that many of the kidneys frequently described as of the large white variety are affected by a waxy degeneration which has been overlooked, and which therefore places them in the following variety.

II. *The Large Kidney with Waxy Degeneration.*—This variety and the small granular kidneys constitute the most frequent forms of Bright's disease. This form embraces a large proportion of the cases developed in connection with phthisis, syphilis, chronic suppurations, and severe cachexiæ. As already mentioned, a considerable number of the large white kidneys belong to this variety.[1] Waxy degeneration of the kidneys is, with very few exceptions, accompanied by waxy degeneration in other parts, especially the spleen and the liver. The kidney is one of the favorite seats of this abnormal deposit. The waxy change in the kidney may be so slight as to be recognizable only upon microscopical examination, or it may be so extensive as to give a very characteristic aspect to the organ. In nearly all doubtful cases its presence or absence can be determined by means of the simple iodine test. (See p. 55.) For microscopical examination iodine and sulphuric acid and methyl-violet are to be preferred. A slight or moderate waxy degeneration may occur without further parenchymatous or interstitial changes. This simple waxy degeneration, however, is so rare that its occurrence has even been denied. Waxy degeneration of the kidney, when of clinical importance, may be considered practically as associated always with diffuse nephritis. Whether the waxy change be the cause of the nephritis, or whether both be effects of the same cause, are undecided questions which at present do not admit of profitable discussion.

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[1] Some writers regard the large white kidney as characterized especially by fatty degeneration of the epithelium, as free from waxy degeneration, and as unattended by hypertrophy of the heart. This constitutes only one variety of large white kidney. A part of the confusion arises from the fact that there are different forms of diffuse nephritis which may present the appearances of the large white kidneys—namely, 1. The pale kidney of acute Bright's disease, especially of scarlatinal nephritis. 2. The large pale kidney without waxy degeneration and with hypertrophy of the heart. 3. The large pale kidney of chronic Bright's disease, without waxy degeneration and without hypertrophy of the heart. 4. The large pale kidney with waxy degeneration. Most of the large white kidneys of chronic Bright's disease without hypertrophy of the left ventricle are affected with waxy degeneration.

Kidneys affected with waxy degeneration present a variety of gross appearances according to the degree of degeneration and the extent and character of the parenchymatous and interstitial changes. The waxy change may be so slight as not to influence the appearance of the kidney, which then presents some one of the forms of chronic diffuse nephritis. Usually, however, careful inspection will reveal in the glomeruli, and perhaps elsewhere, the characteristic translucent appearance of the waxy material. When a large amount of the waxy deposit exists, we have the characteristic appearance of the waxy kidney. The organ is then enlarged, pale, and anæmic. The consistence is firm. The surface is smooth and glistening. The capsule is not adherent. Upon section the cortex is found to be the part chiefly swollen. It presents the same pale aspect as the surface. The pyramids are bluish-red, as in most forms of diffuse nephritis. The cortical striæ are obscured. The translucent Malpighian tufts are visible. The cortex presents a combination in irregular patches or in striæ of translucent gray portions with opaque white or yellow portions. The latter correspond to the parts most affected by fatty degeneration, and the former to the waxy districts. The more the translucent gray substance predominates, the greater is the extent of the waxy degeneration and the more characteristic of the waxy kidney is the appearance. The more the yellowish parts predominate, the more closely does the appearance approach that of the large white kidney without waxy degeneration.

By the use of iodine it is evident to the naked eye that the favorite seats for the deposit of the waxy material are the glomeruli and the vasa recta. Microscopical examination shows that the capillaries of the cortex and the membranæ propriæ of the tubes, especially of the straight tubes and the loops of Henle, are also often affected. The deposit, as a rule, is at first in the middle coat of the arterioles and around the capillary walls of the glomeruli. The walls of the affected vessels become swollen and homogeneous. The lumina of the vessels is gradually diminished, and may be obliterated. In a similar way the walls of the tubes may be thickened and homogeneous. The nephritic changes are in most respects the same as those described for the preceding variety, and need not here be repeated. It is only necessary to add that hemorrhages are usually absent in kidneys affected with waxy degeneration. Fatty degeneration of the epithelium and anæmia of the kidneys are prominent features. The waxy glomeruli present a more homogeneous appearance, fewer nuclei, and less shrinkage than those involved in the other forms of chronic diffuse nephritis. There is nothing characteristic of waxy degeneration in the casts found in the tubes.[1]

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[1] These casts, often called waxy, may stain red with methyl-violet, but they do not give the characteristic reaction with iodine and sulphuric acid. Formless clumps presenting this reaction have, however, been found in the tubes (Kyber).

Waxy degeneration is not confined to large smooth kidneys. It is not very rare to find large waxy kidneys with irregular surface and partially adherent capsule. Small granular kidneys may also be waxy. Waxy kidneys are very rarely accompanied by hypertrophy of the heart. The coincidence of waxy spleen and of waxy liver with this form of kidney disease is an important element in the diagnosis. There are cases, however, in which the change in the kidney is well marked, while in the liver and in the spleen it is insignificant. It may always be inferred that some degree of waxy degeneration of the kidney exists when this change can be recognized in the liver and in the spleen.

III. *The Small Granular Kidney.*—At least three varieties of this form of kidney may be recognized—namely, one representing an atrophic stage of the large kidney without waxy degeneration; a second representing the same stage of the large waxy kidney; and a third, being the genuine contracted kidney, of which no stage of enlargement exists. The first two are called secondarily-contracted kidneys. Nothing could be more erroneous than the view which once prevailed that the large kidneys in the natural order of events, if recovery do not take place, necessarily pass into a stage of contraction, and that the majority of atrophied kidneys are preceded by a stage of enlargement. The secondarily-contracted kidneys do not usually become as small as the genuine or primarily-contracted kidneys. It is not rare to find small kidneys with waxy degeneration. Many of the small granular kidneys occurring in gout are waxy. It is probable that in some of these cases the waxy degeneration is superadded to a genuine contracted kidney. Many of the small waxy kidneys, however, are better regarded as representing an atrophic stage of the large waxy variety.

While the existence of secondarily-contracted kidneys is admitted, it is the primarily-contracted form which is referred to in the remainder of this article under the name small granular kidney. Other names for this variety of chronic diffuse nephritis are—genuine contracted kidney, small red kidney, granular atrophy of the kidney, cirrhosis or sclerosis of the kidney, chronic interstitial nephritis, and chronic desquamative nephritis (Johnson). There is no serious objection to any of these names except the last. Of all the forms of chronic diffuse nephritis, this is the one which with most propriety may be regarded as a distinct disease, and this is the view now very generally adopted. There are authorities who hold the opinion that renal cirrhosis is not only to be separated from other forms of chronic Bright's disease, but that it is only one manifestation of a general disease affecting the arteries of the body. This view will receive consideration subsequently.

The kidneys in this form are reduced in volume and weight. As in all forms of Bright's disease, both organs are involved, and, as a rule, to nearly an equal extent. The size may be a third that of the normal kidney or even less. There is usually a compensatory increase of adipose tissue around the kidneys. The organs are abnormally dense and resisting. The capsule is with difficulty detached, and when removed it brings away portions of the torn renal substance. The exterior surface presents little irregular elevations or granulations. The granulations usually have a yellowish and the depressions between them a reddish color. In many cases the name small red kidney is appropriate. Sometimes, however, the prevailing color is whitish or yellowish. Upon section it is observed that the cortical portion especially is diminished. This is reduced to a narrow rim over the pyramids. In marked cases the pyramids extend nearly to the external surface, and their sides are almost in contact. Little cysts, varying in size from a pin's head to a pea or even larger, and filled with a thin fluid or with colloid contents, are frequently observed both on the surface and in the interior. White lines, due to the deposit of urate of soda in the tubes, are not infrequently seen in the pyramidal portion. They are invariably present when the disease of the kidney is associated with gout. Small kidneys with these deposits were called by Todd gouty kidneys. The same deposit of urates may occur independently of gout.

The microscope shows as the most essential change the production in the cortex of a large amount of fibrillated connective tissue. This new tissue is not uniformly distributed, but is in greater amount in some places than in others, and there are usually parts where no thickening of the interstitial tissue exists. Many of the Malpighian tufts are small and fibrous, and are often placed abnormally close to each other in consequence of atrophy of the intervening tubes and retraction of the new fibrous tissue. The rich development of new connective tissue around the shrunken glomeruli may be especially noticeable. An important change in most cases is the growth of fibrous tissue in the walls of the arteries, both in their adventitia (periarteritis) and in their intima (endarteritis). By the obliteration and partial occlusion thus effected of many small arteries and of capillaries in the glomeruli the circulation through the kidneys must be much impeded; but the blood-vessels are not the only parts that suffer by this new growth of fibrous tissue. The tubes are also compressed, and may be in part obliterated. By this constriction of the tubes in certain places the flow of urine through them is obstructed, and, being dammed back, causes cystic dilatations visible to the naked eye. These cysts consist of dilated tubes and of dilated capsules of Bowman. In the latter form of cyst the glomerulus is compressed or entirely obliterated. Although interstitial changes decidedly predominate in this form of kidney, parenchymatous changes are not wanting. Atrophy of the epithelium is constantly met with in tubes enveloped in connective tissue. Fatty degeneration of the epithelium, although less extended than in the large kidneys, will usually be found in some places. Fat-globules may also appear in the interstitial tissue. The tubes within the new fibrous tissue are small and may be entirely obliterated. Outside of this they are frequently dilated. Hyaline and granular casts are often discovered in the tubes, especially those of the pyramids. There may be some increase of the interstitial tissue of the pyramids, but the changes in these are trifling in comparison with the alterations in the cortex.

One of the most constant accompaniments of the small granular kidney is hypertrophy of the left ventricle, unattended usually by valvular lesion. There may be hypertrophy of the whole heart. The hypertrophy is usually attended by more or less dilatation, but it may be simple. Buhl has pointed out the frequent coincidence of fibrous or chronic interstitial myocarditis in connection with this hypertrophy. Another complication of great frequency and of diagnostic value is the form of retinitis called retinitis albuminurica, the ophthalmoscopic appearances of which will be described in connection with the symptoms. Both the retinitis and the cardiac hypertrophy may occur with other forms of chronic Bright's disease, but they are rarely met with in conjunction with the large waxy kidney. Cerebral hemorrhage and inflammation of serous membranes are also to be mentioned as important complications. Bartels calls attention to the frequent occurrence of thickening of the skull-cap and adhesion of the dura mater to the bone. Dropsical effusions are rather the exception than the rule in this form of Bright's disease.

As has already been said, many writers are inclined to regard the small granular kidney as a disease entirely distinct from the other forms of chronic Bright's disease. Even the propriety of considering renal cirrhosis as a nephritis is disputed. Gull and Sutton first advanced the view that renal cirrhosis is only one manifestation of a widespread affection of the whole arterial system, to which affection they give the name arterio-capillary fibrosis. The essential change, according to these authors, is a thickening of the outer coat of the arteries. The disease they regard as mainly one of old age. The hypertrophy of the heart, they urge, is to be explained only by this general arterial disease. There are cases which seem to support Gull and Sutton's theory. Extensive atheroma of the arteries (arterio-sclerosis), such as occurs in old age, is usually attended by partial atrophy of the kidney. This arterio-sclerotic atrophy of the kidney is not, however, usually attended by any symptoms of Bright's disease, and it is not probable that this is the form of disease referred to by Gull and Sutton. Widespread obliterating endarteritis, when it involves the renal arteries, leads to atrophy of the kidneys, which may give rise to grave symptoms; but in many cases, probably in the majority, of small granular kidney no arterial changes have been found which can be assigned with any propriety as the cause of the renal disease. There is not, therefore, sufficient basis for the doctrine that this form of Bright's disease is in all cases the result of primary arterial changes. Moreover, Gull and Sutton have overestimated the influence of old age in the causation, and they seem to have overlooked the instances of cardiac hypertrophy in connection with other forms of Bright's disease.

The view is advanced by Weigert that the primary change in renal cirrhosis is atrophy or necrosis of the epithelial cells (therefore not inflammation), and that in accordance with a pathological law this loss of substance is followed by new growth of connective tissue. The primary atrophy of the epithelial cells may be the result of various toxic substances (such as alcohol, lead, and uric acid) or the result of insufficient nutrition from defective circulation (primary arterial changes). In most cases, however, we are ignorant of the primary cause.

CLINICAL HISTORY.—Chronic Bright's disease sometimes follows the acute affection. This statement applies chiefly to the large kidney without waxy degeneration; but as a rule the disease is developed imperceptibly and is subacute from the first. In a large proportion of cases the event which first directs attention to the existence of renal disease is dropsy. Prior to this event the patient for some time may have been sensible of impaired strength, and the appearance may have denoted anæmia; but not infrequently up to the time when dropsy is noticed the patient is not aware of any deterioration of health. The first appearance of dropsy is œdema either around the eyes or in the lower extremities, or in both situations simultaneously. As regards the amount of dropsy, and the rapidity with which it increases and is diffused, cases differ. It may be present and remain in so slight a degree that its existence is ascertained only by close examination, and it may escape the attention of the patient. On the other hand, in certain cases anasarca occurs either rapidly or slowly, the limbs, body, and especially the genitals, becoming enormously swollen, together with more or less effusion into the serous cavities. A convenient place for determining by pressure with the finger a small amount of œdema is over the sternum. The different forms of disease do not equally give rise to general dropsy. It occurs most constantly and is most marked in connection with the large kidney. It is wanting in a certain proportion of cases, and in these cases the most frequent form of disease is the small granular kidney. It is also frequently wanting for a considerable period during the progress of the waxy form of disease. Of 89 out of 102 recorded cases of chronic Bright's disease under my observation, in which facts respecting dropsy were noticed, it existed in 74, and was absent in 15 cases. The dropsy was slight or moderate in 21, and considerable in 36 cases.

In 17 cases the degree and the extent of the dropsy were not noted.[1] Of 430 cases collected from different authors and analyzed by Frerichs, general dropsy existed in 376 and was wanting in 34. As a rule, general dropsy is marked in the cases in which the albumen in the urine is very abundant, and, *per contra*, general dropsy is either slight or wanting in the cases in which the urine contains little or no albumen.

The examination of the urine is of great importance as regards diagnosis in the different forms of chronic Bright's disease. In the large kidney without waxy degeneration the quantity of urine excreted within twenty-four hours is diminished unless the arterial tension be elevated by hypertrophy of the heart, in which case the quantity is normal or even increased. The diminution is rarely as marked as in acute Bright's disease. The specific gravity, as a rule, is high when the quantity of urine is scanty, and low under opposite circumstances. Generally it does not vary much from the normal standard. The urine of this form of kidney is usually of a yellow color and turbid. The amount of albumen is considerable, and in some cases very large. The daily excretion of urea is lessened. There is generally an abundant sediment which contains casts, epithelial cells, red and white blood-corpuscles, and granular material. Urates may be deposited in abundance. All varieties of casts are found, but the most common are hyaline casts, either narrow or broad, and frequently dotted with oil-globules and granular casts which may also contain fatty molecules. To both varieties of casts frequently adhere red corpuscles, pus-cells, and epithelial cells; and casts composed wholly of epithelium may be observed. The pus-cells usually exceed in number the red corpuscles, but there are exceptions to this rule. Cells filled with fatty globules (so-called granular or exudation corpuscles) and free fatty granules are often present.

The urine excreted by kidneys affected with waxy degeneration presents great variations in its characters, these variations being influenced especially by the degree and the extent of the diffuse nephritis which is present and by the amount of waxy deposit. When the parenchymatous changes predominate the urine presents the characters belonging to the preceding form. In consequence of the absence of cardiac hypertrophy the quantity may be greatly diminished, although rarely in the degree belonging to acute Bright's disease. Red blood-corpuscles are absent or few. In no form of Bright's disease is a larger percentage of albumen met with than in these large waxy kidneys with extensive parenchymatous changes. With the typical waxy kidneys—that is, those containing a large amount of waxy deposit and presenting in most parts a pale translucent appearance—the urine usually is abundant, very pale in color, of low specific gravity, clear, and rich in albumen. The specific gravity may fall as low as 1005 or even lower. The quantity of albumen is usually considerable, but it may be slight. The daily excretion of urea is below the normal, but the poor nutrition of the patients will in a great measure explain this. There is little or no sediment. Casts are not abundant, and they are chiefly hyaline and so-called waxy casts. The term waxy cast is often employed synonymously with hyaline cast. When used in a different sense, it refers to broad homogeneous casts of a yellowish hue and more distinct and refracting than the hyaline casts. These waxy casts are probably similar in structure to the hyaline; and they are not, as the name implies, composed of waxy or amyloid substance. Exceptionally, however, casts are found which give the same reaction with iodine as the waxy material. The so-called waxy casts are not confined to the waxy kidney, but they may be found in the urine of any form of chronic diffuse nephritis. It is this abundant, clear, albuminous, light urine which is considered most distinctive of waxy degeneration of the kidney, but, as already mentioned, its characters are greatly modified by the intensity and the extent of coincident inflammation and by the degree of waxy change. Litten and others have reported cases of even extensive waxy degeneration of the kidneys without albuminuria. In some of these cases dropsy was present during life. We have no satisfactory explanation of the absence of albuminuria in these cases.

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[1] Vide "A Clinical Report on an Analysis of One Hundred and Two Cases of Bright's Disease," by Austin Flint, M. D., one of the Attending Physicians of Bellevue Hospital, etc., published in the *Bellevue and Charity Hospital Reports*, 1870, p. 226.

The small granular kidneys (renal cirrhosis) sometimes secrete urine which in no way differs from the normal. It is particularly to be emphasized that albumen and casts may be absent from this urine. This is one of the reasons why this form of chronic diffuse nephritis so often escapes detection. The quantity of urine excreted by the small granular kidneys usually exceeds the normal. The polyuria may be so great as to lead to the suspicion of diabetes. The specific gravity is low. It averages about 1010, but it may be less. The color is pale greenish-yellow. The urine is clear, there being little or no sediment. The percentage of urea is lessened, as is likewise in most cases the quantity excreted daily. It is not, however, very exceptional to find this quantity equal to that in health. Albumen is usually present in small amount. Its quantity is in striking contrast to that in most other forms of Bright's disease. It may fail altogether, but this is generally only a temporary occurrence. Bartels, however, mentions a case in which albumen was absent throughout the whole course of the disease. Exceptionally, the quantity of albumen is abundant. When a uræmic attack supervenes, the amount of albumen is usually increased. If waxy degeneration be present, there is an increase in the quantity of albumen. Casts may be absent. Careful search will usually reveal a few hyaline casts. Granular casts are much less frequent. Toward the end of life it will usually be found that the quantity of urine is diminished, the amount of albumen is increased, and a sediment containing casts appears. With small kidneys due to secondary atrophy, the urine has qualities similar to those described if hypertrophy of the heart be present.

The characters of the urine belonging to the different varieties of Bright's disease are not sufficiently distinctive to justify a positive diagnosis of the form of kidney in all cases. There is no form of cast which is characteristic of any one variety of the disease. In general, it may be said that hypertrophy of the heart and waxy degeneration tend to increase the quantity of urine, and that low arterial tension and active inflammatory changes diminish the quantity. An abundant sediment containing many casts and blood-corpuscles is indicative of an active inflammatory process, and particularly of that associated with parenchymatous changes. The smaller in quantity the urine, the richer it is in sediment and in albumen as a rule. It is of importance to estimate the amount of urea excreted daily, for which purpose it is of course necessary to determine the percentage of urea in the total quantity of urine passed in twenty-four hours. The specific gravity affords an approximate idea, although far from an accurate estimate, of the percentage of urea. The specific gravity of a single specimen of urine is manifestly of no value in forming an idea as to the daily excretion of urea. It is necessary to take into consideration at the same time the quantity of urine voided in the twenty-four hours. A low specific gravity may be compensated by a large daily excretion of urine.

Pallor of the countenance, due to anæmia, is generally more or less marked. The anæmia is measurably, if not chiefly, owing to the loss of albumen, and it is most marked in cases in which albuminuria and general dropsy are prominent. It may be an effect of defective nutrition from anorexia or persistent vomiting. In cases of cirrhotic kidney in which albuminuria and dropsy are slight or wanting pallor is not usually a marked symptom. Œdema and pallor combined give to the face an appearance highly characteristic of Bright's disease.

Symptoms referable to the digestive system are more or less prominent. Indigestion or dyspeptic disorder is common. Flatulence of the stomach is a frequent symptom. Vomiting is a prominent and persistent symptom in certain cases. It is referable to the vicarious elimination of urinary principles by the gastric mucous membrane. It may be preceded or accompanied by other symptoms denoting uræmia, but it is sometimes the chief uræmic manifestation. The uræmic vomiting occurs when the stomach is empty, and often early in the morning. The vomited matter or eructated gas sometimes has an ammoniacal odor. The acts of vomiting often occur suddenly and violently without much previous nausea. These circumstances connected with vomiting should always excite suspicion of renal disease, although no other symptoms pointing to the kidneys be apparent. Uræmic vomiting not associated with general dropsy denotes the small granular kidney. The vomiting is sometimes so persistent as to interfere with alimentation and thus induce alarming prostration.

Diarrhœa is a less frequent symptom. It is, however, a prominent and persistent symptom in some cases, either with or without vomiting, or, like vomiting, it may be a uræmic symptom. It may proceed from uræmic intestinal ulceration. It is especially frequent in cases of the waxy kidney combined with waxy disease of the intestine.

Urea is also eliminated vicariously by the skin. The perspiration emits distinctly the odor of urine in some cases. An intense characteristic odor is given off from the skin by some patients who do not pay any attention to cleanliness. Crystals of urea are sometimes found on the skin. Bartels observed a case in which the full beard of a patient had a frosted appearance from an abundant deposit of these crystals. An occasional symptom is an intense itching of the skin.

Symptoms referable to the respiratory system may be produced by pleural effusion or hydrothorax, by pulmonary œdema, and by œdema of the glottis. Pleuritic effusion and œdema of the lungs sufficient to occasion notable embarrassment of respiration occur, especially when disease of the heart, involving mitral contraction or regurgitation, or both, coexists with the renal disease. Under these circumstances the pleural cavities may become nearly filled with serum. Œdema of the lungs under these circumstances is sometimes the immediate cause of death. The occurrence of pulmonary œdema is sometimes very sudden, and it may prove quickly fatal. Not infrequently, however, the œdema disappears, even when life has been placed in imminent danger. Œdema of the glottis is of rare occurrence (according to the statistics of Frerichs and of Rosenstein, in only 4 of 292 cases). It may occur when general dropsy is moderate or slight or wanting.

Dyspnœa may exist in a marked degree when not referable to any of the conditions just named, but proceeding apparently from a toxical effect upon the nervous centre presiding over respiration. In these cases the moist râles incident to pulmonary œdema and the dry râles of asthma are wanting. Auscultation and percussion give no signs denoting any pulmonary affection. I have met with several cases in which this nervous dyspnœa was the most prominent symptom; and I have known it to exist when no other symptom was present to direct attention to the kidneys as the seat of disease. The patients suffer from a feeling of apprehension of "losing the breath," and are afraid to sleep on this account, an exertion of the will being required for respiration. Disturbance of the rhythm of respiration characterized by the frequent occurrence of a long interval between the respiratory acts, followed by series of rapid respirations (Cheyne-Stokes respiration), is not infrequent, occurring in connection with uræmic stupor and coma. Enlargement of the heart with predominant dilatation is another cause of dyspnœa. Especially in cases of contracted kidney dyspnœa from this cause may occur before other events have directed attention to the kidneys as the seat of disease.

Symptoms referable to the nervous system are among the most important of those belonging to the clinical history of the disease. Pain in the back is rarely prominent, and is generally wanting. Cephalalgia is a frequent symptom. It may precede as well as accompany other symptoms which point to the existence of renal disease. Neuralgic pains in other parts of the body are common; and attacks of vertigo are not infrequent. Insomnia and disturbed sleep are frequent symptoms. Troubles relating to vision are in certain cases important symptoms. Diplopia or double vision, hemeralopia or night-blindness, myopia, and presbyopia are attributed, and perhaps are fairly attributable, to the disease. Including these forms of disorder, vision is more or less affected in a pretty large proportion of cases; but exclusive of these, either obscurity or loss of sight—that is, amaurosis complete or incomplete—is not of extremely rare occurrence. The amaurosis affects both eyes. When not complete—and it rarely is so—the degree varies in different cases and in different periods of the same case. In chronic as in acute Bright's disease the impairment of vision may depend on an intracranial morbid condition, the ophthalmoscope showing no morbid appearances within the eye. This condition is called uræmic amaurosis. Under these circumstances it is temporary, and is liable to recur from time to time. I have known it to occur repeatedly in the same case as a precursor of uræmic coma and convulsions. More frequently, however, it is connected with changes within the eye, which by means of the ophthalmoscope have been brought under clinical observation. In this so-called *retinitis albuminurica* the optic papilla is swollen, clouded, and indistinctly bounded, the retinal veins are tortuous, and white patches appear in different parts of the retina. The most characteristic appearance, however, is the presence of white dots and streaks in the periphery of the macula lutea. "These fine white dottings and radiating lines give the impression of having been splashed on with a feather." The appearances are so distinctive that a practised ophthalmoscopist is led by them to suspect renal disease. It has happened in several cases which have come under my observation that disease of the kidney was discovered by means of ophthalmoscopic appearances, the patients having first consulted oculists for defect of vision. Amaurosis dependent on neuro-retinitis may diminish, and recovery is possible; but it is almost always persistent, either progressively increasing or remaining stationary after it has reached a certain degree. The development is usually gradual, whereas the so-called uræmic amaurosis occurs suddenly. The patient describes the defect of vision as like that of a mist before the eyes. Deafness, hemiplegia, paraplegia, and paralysis limited to a single member have been observed to follow or accompany uræmic coma and convulsions. Muscular cramps are sometimes a source of suffering, and I have known them to constitute the chief ailment for some time before other symptoms directed attention to the kidneys.

Coma and convulsions are the most important of the symptoms referable to the nervous system. They occur in a pretty large proportion of cases and are often forerunners of a fatal termination. In chronic Bright's disease, as in the acute affection, they proceed from uræmic poisoning; they are therefore among the ulterior effects of interference with the secretory functions of the kidneys. They are consequently most likely to occur when the secretory structure of the organs is extensively destroyed, and hence especially in connection with the small granular kidney. In acute Bright's disease they always denote great danger, but in chronic Bright's disease the danger is much greater.

Coma occurs in some cases, and death takes place without the occurrence of convulsions. Occasionally coma occurs suddenly, but generally it is developed gradually, the patient having been more or less somnolent, dull, or lethargic for several days before becoming profoundly comatose. The coma is sometimes quiet, and in some cases accompanied by stertor. Marked dilatation of the pupils is sometimes observed. The face is congested, sometimes becoming livid, and death is preceded by tracheal râles. The convulsions are epileptiform in character, varying much in degree and extent in different cases. They occur in paroxysms which may be repeated at short intervals, the patient in the intervals being completely comatose, the convulsions at length ceasing, and the coma continuing until death; or after a paroxysm or a series of paroxysms the patient recovers intelligence, and hours, days, weeks, or months elapse before a recurrence takes place. Convulsions not following coma are generally, but not always, preceded by symptoms referable to the head, such as cephalalgia, vertigo, and sometimes delirium. Contraction or rigidity of certain muscles has been observed to precede convulsions. Of 70 cases among those which I have analyzed, selecting those only the histories of which embrace either death or recovery, coma or convulsions were noted in 26.

The uræmic phenomena, if neither an acute inflammation nor an essential fever exist, are not, as a rule, accompanied by notably increased frequency of the pulse; the action of the heart is sometimes diminished in frequency; and I have known the pulse to fall to 32 per minute. Pyrexia is wanting except in a certain proportion of the cases in which coma and convulsions occur. In some instances, in connection with these events, there is a considerable rise of temperature (105° F.); in other instances the temperature is lowered, and it may fall considerably below the minimum of health. The circumstances which occasion these variations are not as yet ascertained. Further study of the temperature in the different varieties of chronic Bright's disease is a desideratum.[1]

Delirium, which has just been mentioned as sometimes preceding convulsions, is occasionally a prominent feature in cases of uræmic poisoning occurring in connection with both acute and chronic Bright's disease. It is characterized in some cases by remarkable hallucinations. The delirium may be quiet or gay or sad, and it is sometimes furious or maniacal. Uræmic poisoning, as manifested by delirium, coma, and convulsions, may occur in cases of Bright's disease with abundant general dropsy, but it often occurs in cases in which dropsy is slight or wanting. Of the 26 cases above referred to in which coma or convulsions occurred, in 11 there was no dropsy. It may follow copious vomiting and purging, but it occurs oftener in cases in which these symptoms are slight or wanting. The symptoms of uræmic poisoning are sometimes the first to direct attention to the kidneys as the seat of disease.

After an attack of uræmic coma and convulsions there is sometimes complete absence of the symptoms of renal disease, inclusive of the morbid characters of the urine, the disease remaining completely latent and another outbreak occurring after an intermission of a greater or less duration.[2]

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[1] Vide Bourneville, *Études cliniques et thermomètriques sur les Maladies du Système nerveux*, 1872; also, article by McBride in the *Archives of Medicine*, New York, Feb., 1880; also, Bartels in *Ziemssen's Cyclopædia*, vol. xv.

[2] For cases illustrating the latency of renal disease, vide *Bellevue and Charity* Hospital *Reports*, 1870, p. 256.

As regards uræmic phenomena, different cases present a striking difference in the amount of lesions with which their occurrence is connected. Coma, convulsions, and death may take place when the kidneys are not greatly diseased, and, *per contra*, the amount of disease sometimes becomes very great before any uræmic phenomena are manifested. So also there is a great difference in different cases with respect to the relation between the diminution of the excretion of urea and the effects of its accumulation in the blood. The occurrence of uræmic phenomena depends much on the rapidity with which the accumulation of urea in the blood takes place. *Cæteris paribus*, uræmia is quickly induced if the urea accumulate rapidly. On the other hand, if the accumulation take place slowly, the system acquires a tolerance of urea, as of other toxic substances, and hence fatal uræmic effects may be postponed for a long period. Another explanation has reference to the production of urea in the system. This will vary within wide limits according as the ingestion of nitrogenized food is small or abundant, and also according to the amount of muscular exercise. Different cases differ also as regards the vicarious elimination of urinary constituents through the skin and alimentary canal. It is a matter of clinical observation that uræmic manifestations are less frequent with than without dropsy. Bartels finds an explanation of this fact, in a measure, in the amount of urea contained in the dropsical effusions. The elimination of the urea renders the dropsy to a certain extent protective against uræmia.

The complications of the disease form an important part of the clinical history. Several affections included among the symptomatic events might be considered in the light of complications—namely, œdema of the lungs, œdema of the glottis, and amaurosis. Other secondary affections are pericarditis, peritonitis, pleuritis, and cerebral meningitis. Of these, pericarditis and pleuritis occur oftener than inflammations of other serous membranes. They are secondary especially to the small granular kidney.

Hypertrophy of the left ventricle of the heart is developed especially in connection with the small kidney. It may be a secondary affection in cases of the large kidney, but is rarely so in cases of the waxy kidney. If not associated with valvular lesions, hypertrophy of the left ventricle renders the existence of renal disease extremely probable. The hypertrophy may not give rise to any subjective symptoms, and it will therefore be overlooked if the rule be not adopted of always making a physical examination of the chest. With hypertrophy of the heart is associated a hard, resistant pulse and increased intensity of the aortic second sound, indicating high arterial tension. In my analysis, out of 26 fatal cases in which the condition of the heart was noted, in 11 there was more or less enlargement without valvular lesions. Cirrhosis of the liver and fatty liver are not infrequently associated with Bright's disease, but they are to be considered as concomitant, not secondary, affections. This statement is applicable to waxy liver and waxy spleen, which in a very large proportion of cases accompany the waxy form of disease of the kidneys. Waxy disease of the alimentary canal coexists in a certain proportion of cases. Cerebral hemorrhage, although not of frequent occurrence, occurs sufficiently often to show a pathological connection. It is a complication of the small granular kidney oftener than of the other forms of renal disease. Hemorrhagic pachymeningitis is an occasional complication. Epistaxis, the frequent recurrence of which in itself, in a person in middle life, should excite suspicion of renal disease, may be explained by disease of the blood-vessels and hypertrophy of the heart. A relatively greater weakness of the left than of the right ventricle is causative of pulmonary œdema. Pulmonary phthisis is a complication of the waxy kidney sufficiently often to show a pathological connection; but this does not hold true of the other forms of renal disease. The researches of Fenwick and Wilson Fox show that the appearances after death often denote gastritis. This may be attributed to the presence of decomposed urea in the stomach. Gastritis occurs especially as a complication in cases of the small granular kidney. Epistaxis and hemorrhages in other situations may be here mentioned as of not infrequent occurrence.

Chronic rheumatism is sometimes associated with chronic Bright's disease. It may, however, be doubted whether it occur often enough to establish any pathological connection. Todd and other British writers have observed the frequent association of gout and the small kidney. This form of Bright's disease was called by Todd the *gouty kidney*. Gout in this country is, comparatively speaking, a rare disease, but the small kidney is by no means extremely rare. The union of these diseases is not of frequent occurrence with us.

PATHOLOGICAL CHARACTER.—With the description of the anatomical appearances the grounds for regarding chronic diffuse inflammation of the kidneys as the basis of all forms of chronic Bright's disease have been given.

Albuminuria, dropsy, and uræmia are common to both acute and chronic Bright's disease. Reference has been made to the mode of their production under the pathological characters of the former disease. Although a certain amount of hypertrophy of the heart may occur with acute Bright's disease, especially in the scarlatinal form, this symptom becomes prominent only in cases of chronic Bright's disease. There are many theories and much controversy as to the cause of this hypertrophy. Perhaps the most plausible of these theories is that which was advocated by Bright, which refers this hypertrophy to the accumulation of urinary constituents in the blood, especially urea. In some way, it is believed, these constituents increase the blood-pressure, but whether by irritating the vaso-motor centres, by acting directly upon the vascular walls, or by rendering the blood more difficult of circulation, is uncertain. In a certain number of cases, particularly of the atrophied kidneys, the cardiac hypertrophy may be due to structural changes in the arterial walls.

However produced, the hypertrophy of the heart is an important conservative process. It compensates in a remarkable degree for the disturbance of the renal functions incident to the small granular kidneys. The fact that notwithstanding the destruction of a large amount of renal parenchyma these kidneys secrete a normal or even increased amount of water, and not infrequently a nearly normal quantity of solids, is explained by the high arterial tension in the diseased organs; and this tension could not be maintained without increased force of the heart. The compensation is, however, less complete for the excretion of the solids than for that of the water of the urine. The beneficial effects of this compensatory hypertrophy are made evident when from any cause (dilatation or fatty degeneration) the cardiac muscle becomes insufficient. Then the quantity of urine becomes small, the excretion of solids is still further diminished, and the danger of uræmia is great.

CAUSATION.—In a small proportion of cases chronic Bright's disease follows the acute affection. The causes in these cases are those which give rise to the latter. Exclusive of these cases the causation is often obscure. In a considerable proportion of cases there are no symptoms denoting impaired health prior to the manifestations of the renal disease; that is, prior to either dropsy or uræmic symptoms. This was true in 30 of 53 cases among those which I have analyzed, information on this point being contained in the histories of these 53 cases. In 6 of these 30 cases uræmic coma or convulsions occurred either when the patients were supposed to be in good health or when these events were preceded by other ailments only for a period not longer than three or four days. In the 23 cases in which ailments preceded the manifestations of renal disease, excluding the cases in which other diseases existed, the patients had complained of general debility more or less marked for a period ranging from a few weeks to several years. It is probable that in many of the cases in which the manifestations of renal disease occurred in persons apparently in good health the urine would have been found for weeks, months, or even years, to furnish proof that the kidneys were not healthy. The disease occurs very rarely in infancy and childhood. The disease is also infrequent in old age. In the vast majority of cases the patients are either young or in middle life. My analysis of 102 recorded cases showed not a single case at less than fourteen or more than seventy years of age. One patient only was seventy years of age. The mean age was thirty-four years. A difference relating to age exists between the different forms of renal disease. The large kidney occurs especially in early life, although it is extremely rare at less than two years of age. On the other hand, the small kidney is seldom developed in early life. It is most liable to occur in the neighborhood of fifty. Men are much oftener affected than women. Of my 102 cases, 67 were males and 35 females. As regards habits of intemperance, of the 102 cases which I have analyzed, in 38 the histories contained information on this point. In nearly all these cases the patients were males. In 18 cases the habits, as regards the use of alcoholic stimulants, were good; in 13 cases the patients were spirit-drinkers and intemperate; 3 patients were moderate drinkers; 4 drank malt liquors freely; and in one case it was simply noted that the patient was a "free liver." So far as these facts go, they certainly do not show any special causative influence pertaining to the use of alcoholic stimulants. It is probable that the abuse of alcoholic beverages contributes to the production of the disease, but it is also probable that their direct agency has been much over-estimated. Clinical observation in this country certainly does not warrant the name *spirit kidney*, as applied to the small kidney by Goodfellow. The disease is much oftener observed in hospitals than in private practice among the better classes of society—a fact going to show that the exposures, hardships, and deprivations incident to the lives of those who seek for hospital relief in illness are involved in the causation. Persons whose occupations involve exposure to cold and wet are more liable to be affected than those who are protected against the vicissitudes of the weather. Cases are more frequent in humid, marshy climates and on the sea-coast than in dry, elevated, and inland situations. Malaria is supposed to be a causative agent, but of 78 cases observed in situations in which intermittent fever was of frequent occurrence, this disease had existed antecedently to the renal affection in only 10. Dickinson shows by statistical data that chronic Bright's disease, especially the small granular kidney, is of more frequent occurrence in temperate than in either cold or tropical countries, and that variableness as regards temperature and humidity favors its production.

Chronic Bright's disease may be developed in the course of various other diseases. It is sometimes developed during the progress of pulmonary tuberculosis, but exclusive of the waxy kidney the proportion of the cases in which after death tubercle in the lungs or elsewhere is found to be associated is perhaps not greater than in fatal cases of other diseases. Of the cases of the waxy kidney, pulmonary tuberculosis exists in a considerable proportion, and it precedes the renal disease sufficiently often to denote a causative influence. The waxy kidney is developed secondarily to syphilis in such a proportion of cases as to show that the latter has a causative agency. Caries and necrosis of bones ulcers of the intestine and in other situations, and chronic suppuration in any part, have a relation of causation to this form of renal disease. Todd, Garrod, and other British authors have observed the frequent occurrence of the small kidney in persons affected with gout. That the latter affection is often involved in the causation is doubtless true, but the connection is by no means sufficiently constant to warrant, as a distinctive title of this form of renal disease, the name "gouty kidney." Facts show that the absorption of lead into the system may lead to the development of the small kidney.

Chronic Bright's disease not infrequently supervenes in protracted cases of diabetes mellitus. In these cases the latter is probably causative. In some of these cases there is a remarkable tolerance of the disease of the kidneys, but in some cases the renal affection has a rapid course, life being speedily destroyed by uræmic coma.

In some instances an hereditary element seems to be involved in the causation of chronic Bright's disease. A number of members of the same family may be affected with the disease.

DIAGNOSIS.—The diagnosis of chronic as of acute Bright's disease is to be based on the evidence afforded by examinations of the urine. It is always to be borne in mind that transient albuminuria is a symptom occurring in the course of many diseases. Albumen in the urine is found in cases of all the essential fevers, inclusive of lobar pneumonia, diphtheria, erysipelas, etc. etc. In these connections it denotes hyperæmia of the kidneys or parenchymatous degeneration. Generally the amount of albumen is small. On the other hand, it is to be borne in mind that albuminuria is not an invariable symptom in cases of chronic Bright's disease, nor is the presence of a few casts a sure sign of Bright's disease. These may be found in other conditions, both with and without albuminuria. In general, the diagnosis is rendered sufficiently easy and positive by persistent albuminuria and casts of the different kinds which have been described, together with dropsy and symptoms pointing to uræmia; but the occasional latency of the disease, exclusive of symptoms referable to the urine, is not to be lost sight of. Not very infrequently albuminuria and casts show disease to exist when dropsy and other diagnostic symptoms are wanting. The disease is always to be suspected in cases of coma or convulsions, of cephalalgia or neuralgia in other situations, of repeated attacks of epistaxis, of cerebral hemorrhage, especially in persons before the middle period of life, of vertigo and other forms of cerebral disorder, of amaurosis and other forms of paralysis, of serous inflammations, of persistent vomiting or purging, and of anæmia and impaired muscular strength, whenever the etiology is not otherwise intelligible. In short, it is a good rule in practice to examine the urine in all cases, whether symptoms point to the kidneys or not, and repeated examinations should be made whenever there are any grounds for a suspicion of the existence of renal disease. To determine the coexistence of Bright's disease or to exclude this disease in all affections is of much importance. Of course the urine is to be examined repeatedly in order to ascertain the persistency of albumen or casts, together with other morbid changes.

When acute diffuse nephritis supervenes upon chronic Bright's disease, it may be difficult to determine the prior existence of the chronic affection, and such cases may lead to the erroneous supposition that the chronic is a sequel of the acute disease.

A difficulty in the way of determining the particular forms of chronic disease represented in individual cases is the fact that the different forms are not infrequently combined. In typical cases of each form, however, the diagnostic points are sufficient for the differential diagnosis, but even then mistakes in diagnosis cannot always be avoided. These points, which have been already presented in the Clinical History and Causation, are as follows:

1. *The Large Kidney without Waxy Degeneration.*—The existence of this form is probable whenever the chronic follows the acute disease. In trusting exclusively to this diagnostic point, however, there would be a liability to error arising from the fact that the acute disease may occur in a patient already affected with either the small or the waxy kidney. Much or considerable general dropsy, assuming that cardiac lesions are not involved in its causation, denotes the large kidney. A scanty secretion of urine, with a large proportion of albumen, abundant sediment, and the specific gravity not low, has the same significance. Hypertrophy of the left ventricle of the heart without valvular lesions may occur in this form of Bright's disease when the affection is of sufficient duration. The amount of urine is then increased. Uræmic phenomena—namely, coma and convulsions, cephalalgia, impaired vision, vomiting, diarrhœa, and inflammation of serous membranes—are of less frequent occurrence than in cases of the small kidney; and when they occur they are preceded by the symptoms which are diagnostic of the large kidney. Anæmia is constant, and often exists to a marked degree. The youth of the patient is a point in favor of the large as contrasted with the small kidney. The absence of phthisis and gout is a point of some weight.

2. *The Small Granular Kidney.*—In this form the disease not infrequently is latent as regards any obvious symptoms prior to uræmic manifestations. The minor manifestations—namely, headache, vomiting, impaired vision, etc. —may be the first to occur, or those of a graver character—namely, coma and convulsions, pulmonary œdema, and pericarditis—may occur without any antecedent events pointing to renal disease. Uræmic phenomena are much more frequent in this than in the other forms. Dropsy is often wanting, and it rarely exists to much extent. The urine is usually increased considerably in quantity (polyuria); its specific gravity is low; albuminuria is often slight, and at times may be wanting. The characters of the pulse and increased intensity of the aortic second sound of the heart, which denote arterial tension and hypertrophy of the left ventricle, are so common in this form, and comparatively so infrequent in the other forms, that they have much weight in the differential diagnosis. The age of the patient has diagnostic weight. In the great majority of cases the age is between forty and sixty. The coexistence of gout or of lead-poisoning has a bearing upon the differential diagnosis.

3. *Waxy Kidney.*—Important diagnostic points relate to causative and coexisting diseases. Antecedent to the renal affection are phthisis, disease of bone, suppurations, ulcers, or syphilis. Enlargement of the liver and spleen from waxy disease is often associated. The urine exceeds the normal quantity. It is pale, clear, and of low specific gravity. Albuminuria is rarely absent, but it is variable as regards its degree, the quantity of albumen being usually less than in cases of the simple large kidney, and more than in cases of the small granular kidney. Anasarca is more frequent and marked than in the small granular kidney, but less so than in the large kidney. Hydroperitoneum may be out of proportion to the general dropsy, owing to coexisting disease of the liver. Diarrhœa is a frequent symptom, pointing to waxy disease of the intestine. Hypertrophy of the left ventricle does not occur as secondary to the renal affection. Both the minor and the grave manifestations of uræmia are less frequent than in cases of large kidney without waxy degeneration, and much less frequent than in cases of the small granular kidney. This form of disease may occur at any age, but it is rare in very early and in advanced life. The diagnostic points which have been mentioned apply to the typical waxy kidney. In cases in which parenchymatous changes

predominate over the waxy the urine may be scanty, very albuminous, and may contain a large number of casts. Dropsy is then usually very marked. In other words, some of the large kidneys with waxy degeneration give the clinical history usually ascribed to large white kidneys without waxy degeneration.

Uræmic coma, with or without convulsions, sometimes occurs suddenly in persons not known to be affected with renal disease. To determine that the attack is due to uræmia in these cases the urine is to be examined with reference to albumen, the presence of casts, the specific gravity, and the quantity, as soon as practicable; and the catheter should be resorted to, if necessary, in order to obtain a specimen with as little delay as possible. Apoplexy, congestive or from extravasation of blood, meningitis, epilepsy, narcotism, and alcoholism are the affections to be excluded in cases of uræmic coma occurring suddenly or when the previous history cannot be ascertained.

PROGNOSIS.—In a large proportion of cases the different forms of chronic Bright's disease destroy life sooner or later if the patient be not cut off by some intercurrent affection. The duration in fatal cases varies greatly in different cases. It is difficult in many instances to determine the actual duration, because the disease is usually developed imperceptibly and has existed for a greater or less period before its manifestations are observed. Dating from the time when its existence is declared, the fatal termination may take place in a few hours on the one hand, and on the other hand the disease may continue many years. Although recovery does not take place, not infrequently the progress of the disease is stayed or it progresses imperceptibly. The prominent symptoms may disappear, and the patient may have comfortable health for an indefinite period, albumen and casts sometimes disappearing from the urine. The kidneys, like other important organs, such as the heart, liver, lungs, etc., may be damaged to a considerable extent and still be competent to fulfil their functions sufficiently for the continuance of life and health.

A fatal termination is frequently due to accidents or secondary affections, such as pneumonia, pericarditis, œdema of the glottis; cerebral hemorrhage, etc. Coexisting disease of the heart, of the liver, or of other important organs often hastens the fatal termination. The prospects of improvement will also depend much on the general condition of the system. As regards the mode of dying, it is by apnœa in some cases, and in some cases purely by asthenia. In the former cases uræmic coma, hydrothorax, pulmonary œdema, or œdema of the glottis is the immediate cause of death. In the latter cases long-continued and excessive general dropsy, persistent vomiting, or complete loss of appetite and digestion precede death, symptoms of uræmic poisoning being absent. Of 45 fatal cases among those which I have analyzed, in 18 the death was attributable to uræmic coma or convulsions; in 6 cases the death was due to exhaustion irrespective of complications; and in 21 cases the cause of death related, measurably or entirely, to complications or intercurrent affections.

Examinations of the urine furnish important prognostics. If the urine continue to be loaded with albumen, this, in conjunction with persisting dropsy and marked anæmia, denotes that the disease will probably not be of very long duration. Danger from uræmia is to be apprehended in proportion to the deficiency of urea in the urine. A scanty secretion of urine and a low specific gravity point to imminent danger in that direction. Uræmia may, however, develop when the patient is passing a fair amount of urine.

The prognosis is not the same in the different forms of chronic Bright's disease. It is most unfavorable in cases of the small granular kidney; yet it is certain that with the kidneys considerably damaged from this form life may continue with comfortable health for many years. The form of disease represented by the large kidney without waxy degeneration may exist to a greater or less extent and recovery take place. Bartels cites cases in which there was complete recovery from the waxy kidney. The changes in the small granular kidney do not admit of restoration. Whatever may be the form of disease, it is to be borne in mind that the kidneys may be damaged to an extent equivalent to the loss of the whole of one of the organs, and yet the remaining portion be quite sufficient for life and health.

Of my 102 recorded cases, in 77 the histories extended to either death or a condition which was considered as denoting recovery, and of these 77 cases, in 22 the latter termination was noted. In a few of these cases the affection was acute, and in some, instead of a permanent recovery, there may have been only a temporary suspension of the symptomatic phenomena. Making due allowance for these cases, the result of the analysis affords encouragement to hope for recovery in not an insignificant proportion of cases of chronic Bright's disease; and in many cases the hope may be entertained of an arrest of further progress of the disease.

TREATMENT.—In certain cases of chronic as well as of acute Bright's disease therapeutical indications relate to general dropsy and uræmia. General dropsy, when great or considerable, calls for the same measures as in cases of the acute disease. In general, hydragogue cathartics constitute the most efficient and reliable means of affording prompt relief. If the dropsy be slight or moderate, the saline cathartics suffice—namely, the sulphate of magnesia or soda, especially the so-called bitter waters containing these salts (Friedrichshall, Hunyadi Janos, etc.) or the citrate of magnesia; but if the dropsical effusion be large more active hydragogues are required—namely, elaterium, gamboge, or the bitartrate of potassa with jalap. The acrid or drastic cathartics are to be avoided. The hydragogue may be given in doses sufficient to produce free purging, and repeated after intervals of two or three days, or it may be continued in small doses daily, so as to keep up a moderate draining away of liquid. Elaterium is sometimes well borne in doses sufficient to produce a few watery evacuations daily for a considerable period. When it is important to obtain an efficient hydragogue effect as promptly as possible the elaterium is invaluable. It may be prescribed in doses of from one-sixteenth to one-fourth of a grain hourly until the cathartic operation begins. It should then be at once suspended, in order to avoid vomiting. There is an unnecessary apprehension in the minds of many physicians of dangerous prostration from the use of this drug. I have been led by a pretty large experience to consider it safe when employed in the manner just stated. The sulphate of magnesia in half-ounce doses repeated at intervals of three or four hours is an efficient hydragogue cathartic, and is generally well tolerated by the patient.

Diuretics are much less reliable than hydragogue cathartics. Frequently the kidneys will not respond to them, but in some cases they act efficiently. Experience shows that they may be tried without risk of doing harm. The bitartrate of potassa in doses not large enough to purge, or the acetate of potassa, may be given in conjunction with infusion of digitalis, the infusion of broom, or other vegetable diuretics. I have known the infusion of parsley-root (petroselinum) to be remarkably effective as a diuretic remedy. If the state of the stomach and bowels preclude the internal use of diuretics, their external application may be tried after the plan proposed by Christison—namely, an infusion of digitalis ʒj, in water ℥xx, applied to the abdomen by cloths soaked in it or by the spongio-piline. I have known this application to be followed by copious diuresis and disappearance of the general dropsy. A liniment of digitalis, iodine, and squill rubbed over the abdomen and limbs will in some instances increase notably the quantity of urine. If continued, however, it soon excites soreness of the skin. As regards digitalis, it may be useful in certain cases not only as a diuretic, but by limiting the production of urea. Experimental observations by Mégevaud and Rabuteau, under the direction of Robin of Paris, showed a marked diminution of urea in the urine as an effect of this drug, this effect being greater in proportion to the retardation of the circulation which the drug occasions. The inhalation of the vapor of juniper has proved useful as a diuretic in some cases under my observation when remedies were not tolerated by the stomach. The vapor may be produced by dropping thirty minims of the oil on a sponge dipped in hot water and squeezed. The inhalation may be repeated several times daily. The fluid extract of convallaria is useful both in strengthening the heart's action and in increasing the flow of urine.

Sudorifics must be relied upon if hydragogue cathartics be not borne and if the kidneys do not respond to diuretics. The hot-air bath—that is, heated air introduced under the bedclothes—is the most efficient mode of producing diaphoresis. This may be used in alternation with cathartics or diuretics. Patients often express a sense of marked relief after perspiring freely under the use of the hot-air bath, but in some cases it occasions a disagreeable heating of the surface without free perspiration, and if carried too far or repeated too often it produces prostration. Experience in individual cases must be the guide as to its utility and the extent to which it should be carried. The warm bath, perspiration being kept up for some time afterward by covering the patient with an abundance of clothing, may be employed as a substitute for the hot-air bath when the latter cannot be conveniently employed or when its operation is unsatisfactory. Jaborandi or its alkaloid, pilocarpine, sometimes acts efficiently as a diaphoretic remedy, but it is less reliable than the sudorific measures which act directly upon the skin.

In cases of excessive distension of the integument of the lower limbs and genitals temporary relief may be obtained by making a number of minute punctures of the skin with a pin, the punctures being so superficial as not to draw blood. The amount of water which drains away through these imperceptible punctures is surprising. This method is to be preferred to deep punctures or incisions, which may lead to erysipelas and gangrene. Removal of the dropsical effusion in this way has the advantage over measures which cause its removal by absorption, in that the excrementitious principles contained in the effusion do not re-enter the blood. Uræmic effects have repeatedly been observed to follow the rapid absorption of dropsical effusions.

Hydragogue cathartics, diuretics, and sudorifics are also the means for the elimination of urea in the cases in which symptoms denoting uræmia are present. If uræmic poisoning be manifested by coma or convulsions, diuretics are too uncertain to be depended upon. Active hydragogues and the hot-air bath are indicated. Impending death is to be warded off only by the prompt and efficient action of these measures. We have no knowledge of remedies which will neutralize the toxical properties of urea accumulating in the blood. Elimination, so as to reduce the quantity below the amount requisite to produce poisoning effects, is the only resource. It is a therapeutical object to forestall the development of uræmia sufficient to give rise to poisoning effects in the cases in which an examination of the urine shows a notable deficiency of urea or an examination of the blood-serum shows an excess of this excrementitious principle. A moderate hydragogue action may be maintained for this object, or the hot-air bath may be employed once or twice daily. Under these circumstances diuretics may be adequate if the kidneys respond to them. It might be supposed that diuretics are objectionable on the score of their increasing the excretion of albumen in the urine. Experience shows that they do not produce this effect. Although the quantity of urine is augmented, the amount of albumen is generally not greater than before, and it may even be diminished.

An important caution is not to subject patients to the effects of sudorifics, hydragogue cathartics, and diuretics except when these are indicated by uræmic manifestations or deficient elimination of excrementitious principles. The employment of these measures when these indications for them are not present will tend to disturb the functions of the body and thereby diminish tolerance of the disease. Patients may be injured by active treatment in anticipation of future consequences. The eliminative functions of the kidneys may be adequately performed although albumen and casts be present in the urine in large quantity. On the other hand, eliminative treatment may be urgently indicated when the urine contains little or no albumen and few or no casts.

Certain remedies have been supposed to have a curative effect—namely, the tincture of cantharides, nitric acid, and preparations of mercury. Experience has failed to establish the efficacy of these or other remedies addressed directly to the kidneys. Remedies, however, indicated by morbid conditions associated with the renal disease are often of much utility. Anæmia, which is especially associated in cases of the large kidney, calls for chalybeates and tonics, having reference to improvement of appetite and digestion. Dyspeptic disorders claim appropriate remedies. Antecedent and coexisting affections which may have caused the renal disease or contributed to its progress furnish therapeutical indications. This statement applies particularly to the waxy kidney, but measurably to the other forms. The maxim that to improve to the utmost possible extent the general health of the patient is to promote the restoration from, and the tolerance of, a local lesion which may be irremediable, is important in its application to chronic Bright's disease.

Hygienic measures are of the first importance. Alimentation as abundant and nutritious as the powers of digestion will admit of, clothing to secure uniform warmth and activity of the functions of the skin, and such an amount of outdoor life as the strength of the patient will bear, constitute the hygienic management. In all cases, taking into view the correlation of the cutaneous and renal functions, attention to the skin is of special importance. Travelling, and especially long sea-voyages, have been found highly useful. The fact that renal disease is infrequent in tropical and also in cold climates renders it desirable in certain cases for patients to exchange for these climates one which is unfavorable from its variability and humidity. Experience has shown the utility of this measure.[1] In general, warm climates are to be preferred. It is hardly necessary to add that all causes of debility or disorder are as far as possible to be removed. Overtasking the body or mind, exposure to vicissitudes of weather, the use of alcoholic stimulants (save as a means of aiding appetite and digestion, and taken for these objects, if at all, very sparingly), venereal indulgences, and dietetic imprudences are to be interdicted.

Symptoms other than those already referred to in certain cases call for treatment. Cephalalgia and neuralgic pains in different situations call for palliative measures. With regard to the use of opium to relieve pain or procure rest, some writers have inculcated the importance of great circumspection, under the belief that even in moderate doses this drug is liable to produce undue, and even fatal, narcotism if the blood be surcharged with urea. Apprehensions on this score have been greatly exaggerated, if, indeed, there be any foundation for them. I have known opium to be taken habitually in large doses for several months for vomiting and diarrhœa, the urine being loaded with albumen and the dropsy very great, and, so far from its being injurious, life was apparently prolonged by its use. There is reason to believe that opium renders the nervous system more tolerant of the uræmic poison. Opium is indicated by uræmic dyspnœa.

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[1] Vide Dickinson, *op. cit.*

Vomiting and purging, if excessive, claim interference, but it is to be recollected that generally these symptoms denote the vicarious elimination of urea through the alimentary canal. It would be injudicious to attempt to arrest the eliminatory process in this direction, but as excessive vomiting and purging preclude adequate alimentation, palliative measures are to be employed. Relief may be procured indirectly by favoring elimination through the cutaneous surface by means of the hot-air bath. Direct palliation may be obtained by pieces of ice taken into the stomach, by rubefacients or dry cups applied to the abdomen, and by the internal use of hydrocyanic acid, creasote, the oxalate of cerium, and the preparations of bismuth. The latter are often highly useful. The dilute sulphuric or the hydrochloric acid may be given with a view to neutralize the ammonia.

Astringent remedies have been much used with a view to restrain the excretion of albumen in cases in which the urine is highly albuminous. Gallic acid, the acetate of lead, and the astringent preparations of iron have been used for this purpose. I have repeatedly made trial of gallic acid for the end just stated, but with little or no effect.

It has been claimed that restriction of the diet to skim milk effects a cure in cases of the large kidney, and that marked benefit is derived from it in the other forms. These claims in behalf of this treatment have not been substantiated, but there is evidence of its utility in certain cases. It may be tried, but if the symptoms do not within a short period show its utility, it should not be persisted in. Milk, however, may with advantage enter largely into the diet in the different forms of Bright's disease. Buttermilk is sometimes preferred by patients. Owing to the loss of the albumen in the blood from its elimination by the kidney, it is important, if the degree of albuminuria be great or considerable, to make up this loss by a highly albuminous diet.

The treatment of complications, such as serous inflammations, or of affections which may be accidentally associated with chronic Bright's disease, is to be modified by the coexistence of the latter. Active therapeutic measures which, under other circumstances, might be admissible are contraindicated. General or local bloodletting, mercury, and other depressing measures, as a rule, are injudicious. If there be exceptions to this rule, they are of rare occurrence.

In conclusion, the management, as has been seen, does not embrace special medication, but in addition to remedies for particular symptoms or effects and the treatment of concomitant affections it consists, in a great measure, in the removal of morbific causes and in hygienic regulations. The object is not to effect recovery from damage that has been done, but to prevent further injury of the diseased organs, recollecting that the existence of a certain amount of disease is not incompatible with the continuance of life and comfortable health for an indefinite period. The advantage of an early diagnosis is very great in this disease. The same consideration is applicable here as in cases of pulmonary tuberculosis—namely, inasmuch as the object to be hoped for is an arrest of the progress of the disease, the earlier its existence is ascertained the less will be the amount of damage before the disease is stayed.

CHAPTER II.

ACUTE INTERSTITIAL OR SUPPURATIVE NEPHRITIS.—DISEASES OF THE URINARY PASSAGES.—PERINEPHRITIC ABSCESS.

Acute Interstitial or Suppurative Nephritis.—Pyelo-nephritis: Anatomical Characters: Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Pyelitis.—Pyonephrosis.—Hydronephrosis.—Renal Colic and Nephro-lithiasis.—Perinephritic Abscess.

Acute Interstitial or Suppurative Nephritis—Pyelo-nephritis.

THE most common form of suppurative nephritis is that in which a number of small abscesses are present in one or both kidneys. This disease is called also acute interstitial nephritis and (when there is coincident inflammation of the renal pelvis) pyelo-nephritis. Large collections of pus in the substance of the kidney, constituting a single abscess or several abscesses, are rare, and are generally due to traumatism or to extension of suppurative inflammation from adjoining parts.

Suppurative nephritis is caused by the presence in the kidney of micro-organisms capable of exciting suppuration. These organisms usually are micrococci, and can generally be demonstrated without difficulty. The bacteria gain access to the kidney either by the blood-vessels or by the urinary passages.

ANATOMICAL CHARACTERS.—When the bacteria are brought by the blood to the kidneys, the abscesses are called pyæmic or metastatic. Such abscesses are embolic in origin. In this form of acute interstitial nephritis there usually are abscesses also in other organs. Both kidneys are involved. Small, yellowish-white elevated spots, often surrounded by a hemorrhagic zone, are generally seen upon the surface of the kidney after removing the capsule. Such purulent foci are present, as a rule, in both cortex and pyramids. There may be present wedge-shaped white infarctions due to larger emboli. Upon microscopical examination are found foci of pus in which the renal tissue has been entirely destroyed. It is not always easy to demonstrate microscopically the presence of bacteria in the fully-formed abscesses. In an earlier stage of the process colonies of micrococci can be found in the capillaries. Around these colonies the tissue may appear normal or it may be necrotic for a certain distance. Around such necrotic spots, containing colonies of micrococci, a zone of pus-cells makes its appearance. Thus, all stages in the formation of the abscesses can frequently be demonstrated.

The appearances when the bacteria make their way to the kidney through the urinary passages are somewhat different. In these cases the pelvis of the kidney and the bladder are usually inflamed. The affection is a pyelo-nephritis. The abscesses appear as yellowish-white streaks, and they often extend from the papillæ through the pyramidal and cortical portions. The kidneys are much swollen. Purulent foci are generally evident upon the surface of the kidney after stripping off the capsule. Colonies of micrococci are found in the urinary tubes. Pus-cells accumulate in the interstitial tissue and in the tubes, and lead to a destruction of the renal tissue.

In most cases of acute interstitial nephritis the suppurative inflammation occurs in a large number of more or less circumscribed foci. In rare instances there is a diffuse suppurative inflammation of the whole kidney.

Large renal abscesses are not common in the form of disease which has been described. Many cases which have been described as renal abscess are in reality examples of pyonephrosis, an affection which will be considered in the next article. That large collections of pus may, however, in rare instances be formed primarily in the substance of the kidney cannot be doubted.

CLINICAL HISTORY.—The miliary abscesses in the kidneys which occur in pyæmia give rise to no marked local symptoms. The urine undergoes no constant distinctive modifications. It may, however, contain pus-corpuscles. Pyæmic renal abscesses are less common than those in the lungs, liver, and spleen if the pyæmia caused by acute ulcerative endocarditis be excluded.

Acute interstitial nephritis supervening upon disease of the urinary passages is of grave import. The symptoms are those of severe constitutional disturbance—namely, repeated chills, irregular fever, delirium, somnolence, coma, etc. These symptoms denote the presence of some toxic agent in the blood. They are attributed by some to uræmia, by some to ammoniæmia, and by some to septicæmia or pyæmia. (Vide p. 75.) The urine, usually diminished in quantity, is often ammoniacal. It may be albuminous and may contain casts. The alterations in the urine, however, are not diagnostic of this disease. The symptoms distinctly point to a blood disease. The nephritic disease is to be suspected when these symptoms follow the causes which give rise to acute interstitial nephritis. This disease is developed in many of the cases in which death takes place within a few days after an operation for stone in the bladder or for stricture of the urethra. The diagnosis cannot be made with certainty, as similar symptoms and a fatal issue may follow without the kidneys becoming involved. The prognosis is grave—as much so as in cases of pyæmia, with which the condition seems to be identical. Abscesses may be present in other parts of the body, but they are usually confined to the kidneys.

The consideration of large renal abscesses, usually of traumatic origin, belongs to surgery. These cases are clearly distinct from those of acute interstitial nephritis, as just described. These renal abscesses are sometimes due to suppuration in adjacent parts, and they sometimes occur without any apparent causation; that is, idiopathically. They are to be distinguished from pyonephrosis and perinephritic abscesses, and, clinically, this is not always easy. In renal abscess the constitutional symptoms may be either slight or marked. An essential diagnostic point is a recognizable tumor, sometimes fluctuating, referable to the kidney.

CAUSATION.—Suppurative nephritis caused by infectious emboli occurs in many cases of pyæmia and in acute ulcerative endocarditis. It is a rare complication of dysentery and of other diseases. Suppurative nephritis has been observed in cases of actino-mycosis.

As already mentioned, when suppurative nephritis is caused by the entrance of the infectious agent from the urinary passages into the uriniferous tubules, there usually is coincident inflammation of the pelvis of the kidney and of the bladder. This pyelo-nephritis occurs in connection with stricture of the urethra, enlarged prostate, gonorrhœa, stone in the bladder, operations on the genito-urinary tract, and cystitis from disease of the spinal cord or from other causes.

Abscess of the kidney from traumatism is likely to be associated with perinephritic abscess.

Finally, renal abscess may occur as an apparently primary or idiopathic disease without evident cause.

DIAGNOSIS.—Miliary abscesses in the kidney due to pyæmia give rise to no diagnostic symptoms. Pyelo-nephritis consecutive to surgical operations on the urinary tract and to cystitis is to be suspected when the symptoms which have been mentioned in the clinical history are present, these symptoms denoting increased gravity of the disease. Suppurative nephritis of traumatic origin may be diagnosticated by means of unilateral pain and tenderness, together with rigors, more or less fever, and other symptoms of constitutional disturbance. The diagnostic evidence of a large renal abscess is the presence of a fluctuating tumor referable to the kidney. In making this diagnosis pyonephrosis and perinephritic abscess are to be excluded, which generally is not easy. An exploratory puncture may be employed to reveal the presence of pus.

PROGNOSIS.—The prognosis in suppurative nephritis secondary to pyæmia, to acute ulcerative endocarditis, and to affections of the urinary passsages is fatal. Idiopathic abscess of the kidney and abscess secondary to traumatism may discharge in various directions, as into the renal pelvis, into the peritoneal or the pleural cavity, into the stomach or the intestine, or externally. Recovery has taken place in some of the cases in which the abscess opened externally, and also when the opening was into the pelvis of the kidney or into the intestine. A certain number of cases have been cured by appropriate surgical treatment. In all cases, however, the prognosis is grave.

TREATMENT.—The treatment in pyæmic abscesses of the kidney and in suppurative pyelo-nephritis is directed to the general symptoms. In cases of cystitis, or of conditions likely to occasion cystitis, measures to prevent the development of pyelo-nephritis are of great importance. These measures relate to the prevention of the accumulation and of the decomposition of urine in the bladder, to the cleansing of the bladder, and to the treatment of the cystitis. Great care is to be exercised lest infection should occur through the use of unclean catheters. The treatment of traumatic and of large renal abscesses is mainly surgical. The abscess is to be opened, and, if necessary, the diseased kidney to be extirpated, according to established surgical principles.

Pyelitis—Pyonephrosis.

Inflammation of the mucous membrane of the renal pelvis is called pyelitis. In the majority of cases pyelitis is due either to a calculus in the pelvis of the kidney or to some disease of the lower urinary passages, particularly cystitis. A moderate simple pyelitis is not uncommon as a complication of various infectious diseases or as a result of the elimination of toxic substances by the kidney, but in these cases the affection is not of much clinical importance.

ANATOMICAL CHARACTERS.—The slighter grades of simple pyelitis (often called catarrhal pyelitis) are characterized by hyperæmia of the mucous membrane and the exudation of a little mucus and pus. In more severe cases the formation of pus is greater, and may produce a considerable sediment in the urine. In many of these cases with profuse production of pus there is an obstacle to the passage of the urine, and the pus is retained in the dilated pelvis, causing the condition known as pyonephrosis. In pyonephrosis the kidney may take part in the suppurative process or it may simply undergo atrophy from pressure. In extreme cases a large sac containing pus is formed, the walls of the sac consisting of the atrophied renal tissue and the coats of the renal pelvis. In the so-called diphtheritic pyelitis discolored sloughs and ulcers are present in the mucous membrane in connection with fibrinous exudation. The term diphtheritic is used in this connection in its anatomical sense, and not on account of any relation between this disease and diphtheria. In chronic pyelitis the mucous membrane usually is thickened in places and pigmented, and the cavity of the pelvis contains pus. As has been mentioned in the preceding article, pyelitis is often accompanied with suppurative nephritis, constituting pyelo-nephritis. Chronic pyelitis with obstruction to the flow of urine generally causes a genuine chronic interstitial inflammation of the kidney, with lesions like those in the atrophied kidneys of chronic Bright's disease.

CLINICAL HISTORY.—The symptoms of pyelitis vary according to the variety and the cause of the disease. The simple pyelitis complicating many infectious diseases does not generally give rise to any characteristic symptoms. The leading symptoms of calculous pyelitis are the existence or the history of attacks of renal colic, pain in one of the loins, and the occurrence of mucus, pus, and blood in the urine. Calculous pyelitis more frequently than any other form causes hæmaturia, which may be more or less constant or paroxysmal. As a calculus may be lodged in the renal pelvis without the passage of other calculi along the ureter, there may be no history of attacks of renal colic. The lumbar pain, although not a constant symptom, is of considerable value in diagnosis.

When pyelitis is the result of the extension of inflammation from the lower urinary passages, there is usually more or less pain in the lumbar region. The pain is not generally severe, and it may be slight. There may be some tenderness over the region of the kidney. In severe cases, especially in those complicated with suppurative nephritis and in diphtheritic pyelitis, there is fever, which may be of a pyæmic type, and grave constitutional disturbance. In chronic pyelitis with secondary atrophy of the kidney the urine is often greatly increased in quantity, and the symptoms of the small granular kidney may be developed.

When there is marked pyonephrosis a fluctuating tumor may be appreciated in the region of the kidney. Exploratory puncture with a hypodermic needle will reveal the presence of the pus.

CAUSATION.—It is doubtful whether pyelitis ever occur as a purely idiopathic affection, although in some cases it may not be possible to discover the cause. Pyelitis may be produced by wounds penetrating the pelvis of the kidney, but rarely, if ever, by contusions. One of the most frequent causes of this affection is the local action of calculi. Such calculi may occupy a large part of the renal pelvis and send offshoots into the calices. Other foreign bodies, such as parasites (strongylus gigas, distoma hæmatobium, and hydatids) are rare causes. An important cause of pyelitis is obstruction to the flow of urine, leading to the accumulation and decomposition of the urine in the renal pelvis. In these cases some inflammatory irritant is present. The obstruction may be in the ureter, the bladder, or the urethra. Such obstructions are furnished by calculi, tumors, enlarged prostate, stricture of the urethra, phimosis, and paralysis of the bladder in cases of paraplegia. An inflammatory irritant may gain access to the bladder, and thence to the pelvis of the kidney, without any obstruction to the flow of urine. Here belong the cases of pyelitis secondary to gonorrhœa, to specific vaginitis and urethritis in females, and to surgical operations in the genito-urinary tract.

In these cases cystitis is nearly always present. Sometimes pyelitis is secondary to disease of the kidney. The pyelitis secondary to smallpox, scarlet fever, and other infectious diseases, and that secondary to the elimination of cantharides and other toxic substances from the kidney, is of comparatively little practical importance. Tuberculous and cancerous pyelitis will be described subsequently.

DIAGNOSIS.—The diagnosis of pyelitis is to be based on characters pertaining to the urine in conjunction with more or less pain and constitutional disturbance, cystitis being excluded.

Pus-cells and (especially in calculous pyelitis) red blood-corpuscles are found in the urine. To determine that these cells are derived from the renal pelvis it is necessary to exclude inflammatory affections of the bladder and of the urethra. This can sometimes be done by the symptoms and by physical exploration. It is not, however, in all cases possible to exclude cystitis. In uncomplicated pyelitis the urine is generally acid, whereas in cystitis it is usually alkaline, but this rule has less diagnostic value than is often assigned to it. On the one hand the urine may undergo ammoniacal decomposition in pyelitis, and on the other hand the acid reaction does not of necessity exclude cystitis. Moreover, an uncomplicated pyelitis is rare. In many cases there is coincident inflammation of the bladder. It is a prevalent view that there are characteristic epithelial cells lining the renal pelvis, and that when these cells appear in the urine the diagnosis of pyelitis is established. It is admitted that these cells are not present in all cases, and that they are found more frequently at the onset than in the later stages of the disease. The cells considered characteristic of pyelitis are described as oval, pyriform, fusiform, and caudate cells. The presence of these cells in the urine is not pathognomonic of pyelitis. It is generally admitted by histologists that no essential differences exist between the epithelial cells of the bladder and those of the pelvis of the kidney. Both belong to the so-called transitional type and are characterized by the variety of forms which they present. The cell-forms which are described as characteristic of pyelitis may also be derived from the bladder.

The occurrence of paroxysms of pain characteristic of the passage of a calculus along the ureter, and the expulsion of calculi or gravel from the bladder, are important diagnostic events. The sudden discharge of pus in more or less abundance with the urine shows that an accumulation in the pelvis of the kidney has escaped through the ureter into the bladder. Sometimes the ureter on the diseased side becomes obstructed by the entrance of a calculus, and in this case the urine which is voided, being excreted only by the healthy kidney, may be clear and normal in appearance.

Pyelitis caused by calculi will be likely to be limited to one kidney, but if it follow cystitis or retention of urine from an obstruction beyond the ureter, both kidneys will be likely to be affected. Complete or very great obstruction, causing an accumulation of urine within the pelves of both kidneys, leads speedily to uræmia. Complete obstruction, however, limited to one kidney, may exist for an indefinite period without leading to uræmia, the other kidney being sufficient for the excretion of the urine. The accumulation of pus in the pelvis of one kidney may be sufficient to occasion a fluctuating tumor which may be felt through the abdominal walls. This tumor may attain a large size, and in women it may simulate an ovarian cyst. It is to be differentiated from perinephritic abscess.

An accumulation of purulent matter within the pelvis and calices, if the ureter remain permanently obstructed, generally eventuates in perforation and the discharge of the pus in some direction, provided the life of the patient be sufficiently prolonged. The source of the pus is to be determined by the antecedent and coexisting symptoms which point to the seat of the affection. The pus may make its way in an outward direction, giving rise to a subcutaneous purulent collection. It may be discharged into the peritoneal cavity, causing a fatal peritonitis. A case in which these two events occurred has fallen under my observation. Not infrequently the pus has escaped by perforation into the intestine. It has been known to pass into the pleural cavity and into the bronchial tubes.

PROGNOSIS.—The gravity of pyelitis depends much on its causes. If produced by an extension of inflammation in cystitis, and if there be no permanent obstruction to the flow of urine and pus into the bladder, a favorable termination may be looked for. If due to the presence of calculi, and if these make their way into the bladder before destruction of the kidney ensues, recovery will be likely to take place. If from the size of the calculi their escape be impossible, the inflammation will probably continue and the prognosis is unfavorable. Cases have been observed of pyelitis caused by a large calculus in which, after having led to complete destruction of the kidneys, the inflammation has ceased and the calculus has remained encysted by the renal capsule, giving rise to little or no inconvenience, the remaining kidney doing all the work of renal elimination. Permanent obstruction to the flow of urine from the pelvis and calices of both kidneys, dependent on cystitis or retention of urine in the bladder from whatever cause, leads to destruction of the renal parenchyma and death from uræmia. An accumulation of urine and pus in the pelvis and calices of one kidney may prove fatal by perforation into the peritoneal cavity. If the discharge take place either into the intestinal canal or externally, the patient is likely to be worn out by the prolonged drain and constitutional disturbance.

The TREATMENT in cases of pyelitis must have reference to the causation. If dependent on retention of urine from stricture of the urethra, enlarged prostate, or paralysis, measures to relieve the bladder are of prime importance. If consecutive to cystitis, the latter claims appropriate treatment. Quietude is to be enjoined, especially in cases in which the affection depends on calculi. Soothing applications to the loins and anodyne remedies are indicated in proportion to the amount of pain. If the affection become chronic, the balsam of copaiba and other balsamic remedies, also boric acid and benzoic acid, are sometimes useful. Liquids should be taken freely in order to dilute the urine. If the accumulation of pus (pyonephrosis) form a tumor which presents itself in an accessible situation, it should be opened after an exploratory puncture. Tonic and supporting measures are required after the opening of the abscess either externally or into the alimentary canal.

When a calculus is lodged in the pelvis of one kidney it may be justifiable to make an incision and extract the calculus or even to extirpate the kidney. Surgical works are to be consulted for the principles guiding the treatment in such cases.

Hydronephrosis.

Hydronephrosis is the name given to dilatation of the pelvis and calices of the kidney by an accumulation of urine in consequence of some obstruction in the urinary passages. This dilatation leads to atrophy of the renal tissue. The obstruction may be seated in the pelvis, ureter, bladder, or urethra, and consequently the dilatation may involve not only the pelvis, but also other parts of the urinary passages. Thus, if the impediment be in the urethra, this canal behind the obstruction, together with the bladder and ureters, will be distended if hydronephrosis be produced.

ANATOMICAL CHARACTERS.—Hydronephrosis, especially if the distension be extreme, in the majority of cases is unilateral. It may, however, be bilateral, and it then constitutes a graver affection. The degree of distension varies extremely. It may be insignificant, or the dilatation may lead to the formation of an enormous sac filling most of the abdominal cavity and containing several gallons of fluid. From the pressure of the liquid in the dilated pelvis and calices the kidney-substance wastes and gradually disappears. At first the papillæ are flattened, and afterward the pyramids are further encroached upon, and finally disappear. The cortical substance also undergoes atrophy. This wasting of the kidney is accompanied usually by increase of its interstitial tissue. It is very rare to find the hydronephrosis and the atrophy of the kidney so extreme that no remnant of the renal parenchyma can be found. Even in large hydronephrotic cysts it is the rule to find a thin border, or at least islands, of renal tissue upon the cyst-wall. A very large hydronephrotic sac sometimes consists of a single cavity. Usually, however, a number of membranous septa project from the wall into the cavity of the sac and give to its external surface a lobulated appearance. These septa partially enclose smaller cavities or sacculi which represent the dilated calices, and which, therefore, communicate by wide mouths with the main cavity or dilated pelvis. These septa may be prominent or may be reduced to narrow ridges on the inner wall of the sac. The walls of a hydronephrotic sac consist, in addition to atrophic kidney-substance, of dense vascular fibrous tissue. The outer surface may be roughened by adhesions to adjoining parts. According to the seat of obstruction one or both ureters may be dilated. A dilated ureter may reach the size and present the appearance of a coil of small intestine. With unilateral hydronephrosis and considerable atrophy of the kidney the other kidney is often hypertrophied.

The liquid contained within a hydronephrotic cyst varies in its composition. It usually has the appearance of watery urine. It is clear in most cases, but it may be discolored and rendered somewhat turbid by admixture with blood, pus, or epithelium. The specific gravity is low, and the reaction often slightly alkaline. The urinary constituents are usually present, but urea and uric acid may be in small amount or even entirely absent. A small quantity of albumen is almost invariably found. In a few cases the contents have consisted of a gelatinous, colloid substance. Paralbumen, the modified form of albumen present in ovarian cysts, has also been discovered in the fluid of hydronephrosis.

CAUSATION.—The causes of hydronephrosis are conveniently divided into *congenital* and *acquired*. Of 52 cases of hydronephrosis collected by Sir William Roberts, 20 were congenital. *Congenital* causes are the following: 1, A supernumerary renal artery crossing and compressing the ureter near its origin; 2, narrowing or obliteration of the orifices or of the lumen of the ureter; 3, a valve-like impediment produced by an oblique insertion of the ureter into the pelvis; 4, the insertion of the ureter into the upper instead of the lower part of the pelvis; 5, certain abnormalities of the bladder or of the urethra, as vesical diverticula, atresia of the urethra, etc. Congenital hydronephrosis is often associated with malformations of other organs, as imperforate anus, hare-lip, etc.

The causes of *acquired* hydronephrosis may be classified as follows: 1. The impaction of substances in the renal pelvis or in the ureter. Calculi impacted in these parts are among the most frequent causes of this affection.

Much less frequently the lumen of the ureter is obstructed by coagula of blood or by parasites (echinococcus). 2. Disease of the walls of the ureter, causing partial or complete obliteration of its lumen. To this group of causes belong inflammatory thickening, cicatricial stenosis from ulcers, and tumors, especially cancer. 3. Pressure upon the ureter from without. The causes of pressure include pelvic tumors, inflammation of the pelvic connective tissue or of the pelvic peritoneum, prolapse of the uterus, retroflexion of the pregnant or of the non-pregnant uterus, and distension of the rectum. Cancer of the cervix uteri is very frequently complicated by hydronephrosis. It is exceptional for hydronephrosis to be caused by the pressure of an ovarian cyst. The newly-formed fibrous tissue resulting from parametritis and from pelvic peritonitis may compress the ureter. Some of the causes named produce hydronephrosis—not by pressure, but by exerting traction upon the ureter and so causing bends and distortions in its course. 4. Diseases of the bladder which involve one or both of the orifices of the ureters. This class of causes comprises hypertrophy of the coats of the bladder, abscess in the vesical wall, neoplasms, particularly cancer, and large calculi. 5. Affections narrowing the calibre of the urethra. These causes are stricture and the so-called hypertrophy of the prostate. In a few cases hydronephrosis has been attributed to phimosis. Hydronephrosis does not follow these causes until after the bladder has become incapable of overcoming the urethral obstacle. In cases of urethral obstruction, therefore, hydronephrosis is due to a secondary affection of the bladder. 6. In not a few cases no cause of the hydronephrosis has been found. With some of the largest hydronephrotic sacs the autopsy failed to reveal any impediment to the flow of urine. In some of these cases it is believed that a calculus, once impacted in the ureter, has been dissolved or dislodged. In other cases it is probable that some not very obvious cause escaped detection. The largest hydronephrotic cysts are found more frequently with partial than with complete obstruction in the ureter.

CLINICAL HISTORY.—With double congenital hydronephrosis the fœtus is generally not viable. There are cases, however, in which life has been continued in apparent health for many years. A moderate degree of hydronephrosis, whether unilateral or bilateral, is unattended by recognizable symptoms. One kidney may be destroyed by the development of a hydronephrotic sac without any symptoms referable to this condition. In this case the other kidney, which is usually hypertrophied, excretes the normal quantity of urine. If, however, the ureter of the remaining kidney become permanently obstructed, death ensues within a few days with symptoms of uræmia. In cases of double hydronephrosis the condition is manifestly grave if the renal tissue be sufficiently impaired to interfere with its functions. Under these circumstances the urinary constituents are imperfectly eliminated from the body, and death ultimately ensues, usually with the manifestations of uræmia. An important diagnostic sign of hydronephrosis is the discovery of a tumor in the lumbar region. It is only when the hydronephrotic sac is of considerable size that a tumor can be detected. The tumor is fluctuating and presents in some cases a lobulated surface. It has the characteristics of a tumor taking its origin from the kidney, among the most important of which is the existence of the tympanitic resonance of the colon over its anterior surface. This sign, however, may be wanting, the colon being pushed to one side by the tumor. A symptom of almost pathognomonic value is the disappearance of the tumor coincident with the discharge of a large quantity of pale liquid by the bladder. This symptom is not common. It is, of course, present only when the obstacle to the flow of urine is removed or when there is only incomplete obstruction, which latter is more frequent. A hydronephrotic tumor is not painful. It may give rise to symptoms by compression of other parts. Thus obstinate constipation may be due to compression of the colon. The urine presents no abnormal characteristics in most cases of hydronephrosis. As already mentioned, it may be suddenly increased in amount by discharge of the contents of the cyst. If both kidneys be so much involved that their function is interfered with, the urine is usually diminished in quantity. Important symptoms may be referable to the conditions causing hydronephrosis, such as those of renal colic, uterine cancer, etc.

DIAGNOSIS.—If the accumulation of liquid in the renal pelvis be small, the disease cannot be diagnosticated. The recognition of one or more of the various causes of hydronephrosis which have been mentioned is of great importance in the diagnosis. The presence of a tumor and its subsidence or diminution in size, coincident with the discharge of a large quantity of urine, are symptoms which are sometimes available in making the diagnosis. Large hydronephrotic sacs filling the greater part of the abdominal cavity may be mistaken for ascites or for ovarian cysts. Such sacs have repeatedly been mistaken for ovarian cysts, and have been punctured on this supposition. The attempt has even been made to remove hydronephrotic cysts by laparotomy under the belief that an ovarian cyst was present. Some of the characteristic signs of an ovarian cyst have been mentioned on p. 591. It must be admitted that in some cases the diagnosis is very difficult. The presence or absence of urea in liquid withdrawn by an exploratory puncture cannot serve as a diagnostic point. Urea may be present in fluid from an ovarian cyst. It is frequently small in amount, and may be absent in hydronephrosis. The colon is often present over a hydronephrotic cyst, and it is found very exceptionally in front of an ovarian cyst. (The reader is referred to the article on Ascites for the symptoms and physical signs of this condition, p. 587 *et seq.*) Hydronephrosis may be confounded with pyonephrosis. With the latter condition there is more constitutional disturbance with rigors and hectic fever. The presence of pus in the fluid removed by exploratory puncture renders the diagnosis of pyonephrosis positive if other sources of pus can be excluded. The diagnosis also involves the exclusion of hydatid cysts of the kidney, perinephritic abscess, and large renal cysts.

PROGNOSIS.—The prognosis of hydronephrosis depends upon the extent to which the functions of the kidneys are interfered with and the consequent danger of uræmia. Unilateral hydronephrosis is unattended with danger so long as the remaining kidney does its work adequately; but if this kidney become diseased or if its ureter become obstructed, life is endangered. Double hydronephrosis is grave in proportion to the liability to uræmia. The liability to rupture of a hydronephrotic sac is so slight that it hardly enters into the prognosis.

TREATMENT.—An important object in the treatment is the removal of the cause of obstruction. This cannot often be accomplished. The measures to be pursued for that object in individual cases are to be determined by the nature and seat of the obstructing cause. In some instances, by means of rubbing and kneading over the sac or along the course of the ureter, an obstruction has been overcome or the urine has been made to pass beyond the obstructed part. These manipulations should not, of course, be so forcible as to cause pain or to incur any risk of rupture. Removal of the liquid by aspiration has proved successful. Using a small aspirating needle or trocar, this measure is attended with little if any danger of the escape of the liquid into the peritoneal cavity. For this reason it is to be preferred to tapping without aspirating. The treatment in other respects is to be governed by the local and general symptoms in each case. Free opening of the sac and drainage and the entire removal of the tumor are surgical procedures which have thus far been attended by favorable results.

Renal Colic and Nephro-lithiasis.

Urinary concretions or calculi are generally formed within the renal cavities. Calculi of small size may pass from the kidneys to the bladder, giving rise to little or no inconvenience, and, having reached the bladder, they are either discharged with the urine, or, remaining in the bladder, they increase in size and may require surgical interference for their removal. In some cases calculi within the renal cavities, increasing in size, become too large to pass along the ureter, and are consequently retained. As has been seen, retention of urine within the pelvis and calices of the kidney, pyelitis, renal abscess, and destruction of the renal parenchyma are sometimes attributable to the presence of calculi or a calculus. A calculus retained in the pelvis of the kidney may continue to increase in size, and, leading to atrophy of the parenchyma, it may acquire a bulk nearly as great as, or perhaps even greater than, that of the healthy organ. Calculi not too large to pass through the ureter, and not small enough to pass with facility, occasion during their passage more or less pain and constitutional disturbance. The amount and duration of the pain caused by the passage of a calculus are in proportion to its size and the roughness of its surfaces. The pain begins when the calculus enters the duct, and ends when it reaches the bladder. The paroxysm is analogous to that occasioned by the passage of a gall-stone from the gall-bladder to the duodenum. The latter is commonly known as hepatic colic, and the paroxysm of pain occasioned by the passage of a renal calculus is called *renal colic.* In rare instances attacks of renal colic are produced by the passage of coagulated fibrin and of an hydatid vesicle through the ureter.

A paroxysm of renal colic is usually developed suddenly. The attack, however, may be preceded by more or less pain or a sense of uneasiness due to irritation or perhaps inflammation caused by the presence of calculi in the pelvis of the kidney. The paroxysm quickly attains its maximum of intensity. The pain in some cases becomes exceedingly intense. It is referred to the situation of the kidney on one side; that is, by the side of the last dorsal and the first lumbar vertebræ. The pain radiates from this point in different directions, but especially along the course of the ureter, extending frequently to the groin and thigh. In the male, pain is generally felt in the testicle, which is often, but not always, drawn upward by the contraction of the cremaster muscle. Frequently the pain is described as lancinating or tearing. It is sometimes so excessive as to force the patient to groan and cry aloud; he tries a variety of positions to obtain relief, walks about the chamber, and compresses the abdomen with the hands as in intestinal colic. The pain continues without intermission, but there occur exacerbations and remissions. The urine in most cases is notably diminished. The patient experiences a frequent or constant desire to micturate, passing only a few drops at a time. The sensation is well expressed by the term *tenesmus of the bladder*. Not infrequently the urine is bloody. If it contain pus or muco-pus, either pyelitis or cystitis coexists.

With these local symptoms are associated those denoting more or less constitutional disturbance—namely, anorexia, thirst, nausea, and vomiting, coldness of the surface with sweating, and feebleness of the circulation. The countenance is pallid and expressive of anguish. Suddenly, after a duration varying considerably in different cases, the paroxysm ends. The pain abruptly ceases. An abundant discharge of urine takes place. The calculus has reached the bladder, and if care be taken for some time afterward to examine the urine it may be found at the bottom of the vessel. As the passage of a calculus by the urethra, even of one which has caused severe renal colic, may occasion no pain, it is no proof that a calculus is retained in the bladder that its escape with the urine has not been noticed. The general symptoms rapidly disappear, and in one or two days the recovery may be complete. In some cases, however, one or more additional paroxysms follow, other calculi contained within the pelvis of the kidneys entering the ureter in consequence of the dilatation caused by the calculus which has just passed. A person who has experienced one paroxysm is liable to recurrences at periods more or less remote. Not infrequently, however, there is no recurrence of a paroxysm.

The duration of an attack of renal colic varies in different cases, but it is usually short. In the majority of cases it lasts but a few hours, but in some cases it continues for one, two, or three days. Although the suffering is often extreme, it very rarely proves fatal. A persistent impaction of a calculus in the ureter, causing obstruction, is followed by hydronephrosis, and following this pyelitis and pyelo-nephritis may become developed. Ulceration may take place at the point of the obstruction, and fatal peritonitis may be produced by the escape of urine into the peritoneal cavity. These are sources of danger happily of rare occurrence. Simultaneous obstruction of both ureters by calculi has been known to occur, causing anuria and uræmic poisoning.

The diagnostic characters of renal colic are—the suddenness of the attack and termination, the situation of the pain and its extension to the groin, thigh, and testicle, the retraction of the testicle, the diminution of urine, the tenesmus of the bladder, hæmaturia in some cases, and the discovery of the calculus. The fact of antecedent attacks having occurred will assist in the diagnosis. The affections to be excluded are—the form of muscular rheumatism called lumbago, lumbo-abdominal neuralgia, enteralgia, and hepatic colic. In lumbago the pain is less severe, is provoked especially by movements of the body, and is felt on both sides. Lumbo-abdominal neuralgia is characterized by tenderness at certain points. (Vide p. 805.) In enteralgia the seat of the pain is within the abdomen. This statement also applies to hepatic colic. In all these affections the characteristic features of nephritic colic are wanting.

The TREATMENT of an attack of renal colic consists of palliative measures. Complete relief depends on the escape of the calculus into the bladder, and there are no means of aiding directly in effecting its passage. The propelling force is derived from the accumulation of urine behind the calculus and from the peristalsis of the muscular coat of the ureter. The copious ingestion of liquids may, by increasing the urinary secretion, hasten its progress. Fomentations to the loins, the warm bath, morphia given hypodermically, and, if the suffering be excessive, the inhalation of chloroform, constitute the measures for palliation.

The discharge of urinary concretions from the body, either with or without the symptoms of nephritic colic, constitutes *lithiasis* or *gravel*. The term "gravel" is properly applied only to concretions formed within the body, not to those which form in the urine after its emission. Not infrequently concretions varying in size from that of a pin's head to a small pea pass through the ureter without giving rise to sufficient pain to constitute an attack of renal colic. The composition of the concretions varies in different cases. In a large majority of cases (five-sixths) they consist of uric acid. The urine in these cases is always acid. The concretions may consist of the earthy salts—namely, the phosphate of ammonia and magnesia, the phosphate of lime, and the carbonate of lime. Concretions of carbonate of lime are extremely rare in human urine. Ammonio-magnesian calculi are liable to form in cases of cystitis. These concretions are generally not renal but vesical. The urine in these cases is usually alkaline. In another variety the concretions consist of the oxalate of lime. In frequency these come next to the uric-acid calculi. They are known as mulberry calculi. In cases of renal colic the composition of the calculi which occasion the paroxysms may be inferred from the knowledge of concretions which have been already passed and from an examination of the sediment which the urine deposits. If the urine contain uric-acid crystals or abound in the urates or have an acid reaction, the calculus is probably composed of uric acid; and the urine will be likely to contain the phosphates in more or less abundance when the calculi consist of the earthy salts. Oxalate-of-lime or mulberry calculi will be likely to be preceded or accompanied by oxaluria. Oxaluria is not, as was formerly supposed, attended by any characteristic symptoms. It may happen, however, that the calculi giving rise to renal colic are not of recent formation, and the condition of the urine giving rise to their formation may not exist at the time when the attack of colic occurs. A variety of calculus, so rare as to be a curiosity, consists of xanthine. Another rare variety is the cystine calculus. The condition of the urine in which the latter occurs is called cystinuria. In this condition the urine contains hexagonal tablets of cystine. This, more than any other form of lithiasis, has been shown to be hereditary. Cystinuria may exist without the formation of calculi. It lasts for a long time, and sometimes continues indefinitely.

The TREATMENT of nephro-lithiasis, irrespective of attacks of renal colic, will depend on the nature of the concretions or the character of the urine. In cases of uric-acid gravel alkaline remedies are indicated. The liquor potassæ, in half-drachm doses thrice daily, may be given, but either the bicarbonate or the citrate of potassa, a drachm twice daily, is preferable. The Vichy water and the waters of other alkaline springs are well suited to this variety of gravel. The phosphate of soda and ammonia, introduced by Dr. Buckler of Baltimore, is considered as having the power of keeping uric acid in solution in the urine, given in doses of ten grains thrice daily. The phosphate of soda in doses of from a drachm to six drachms is recommended by Heller. The benzoic acid is another remedy, given in doses of eight or ten grains thrice daily. Lithia in the form of the carbonate and the benzoate was introduced by Garrod as a remedy which promised to be especially valuable from its forming with uric acid a highly soluble compound. It may be given in doses of five grains three times daily. All these remedies act as solvents and as preventing uric-acid concretions. In addition to these remedies attention to the digestive organs, the skin, and the functions generally constitutes an important part of the treatment. Liquids should be taken freely between meal-times in order to dilute the urine. The diet should consist mainly of the white meats, milk, eggs, and farinaceous articles.

In cases in which the concretions are composed of the earthy salts the mineral acids are indicated, either the sulphuric or hydrochloric. The nitro-hydrochloric acid is especially indicated in cases of oxalate-of-lime gravel. Diuretic remedies and the ingestion of pure spring, river, rain, or distilled water abundantly form a highly important part of the treatment in all cases of gravel. The Hygeia water may be especially recommended for its purity. The phosphatic and oxalate-of-lime concretions often occur in persons suffering from *nervous asthenia*, and the latter condition claims appropriate management. Abstinence from articles of diet containing oxalic acid is to be enjoined if the concretions consist of the oxalate of lime.

As already stated, the sedimentary deposits which occur in urine after its emission are not properly called gravel. It is important, however, to take cognizance of these as constituting evidence of disorder and as involving a liability to the formation of concretions within the kidney or bladder. The conditions of the urine known as *lithuria*, *oxaluria*, and *phosphoruria*, standing respectively in relation to the different varieties of gravel just noticed, claim appropriate treatment, although concretions within the body have not taken place. (For further details respecting these and other disordered conditions of the urine, the reader is referred to works devoted to urinary affections.[1])

Perinephritic Abscess.

Inflammation of the connective tissue surrounding the kidney is called perinephritis. The tissue involved is the lax adipose tissue in which the kidney is imbedded (the so-called adipose capsule of the kidney) and the neighboring retro-peritoneal tissue. It is possible to distinguish an adhesive and a suppurative variety of perinephritis. The former is characterized by the development of new connective tissue. It accompanies tumors and some other diseases of the kidney. Adhesive perinephritis has no clinical importance, and will receive no further consideration in this article. Suppurative perinephritis or perinephritic abscess is not a frequent disease, but it is attended by characteristic symptoms and merits separate consideration. On post-mortem examination a collection of pus is found occupying the situation of the adipose and connective tissue around the kidney. There may be a number of circumscribed abscesses, but, as a rule, there is a single abscess-cavity. The walls of the abscess may consist of shreddy necrotic tissue, or in old cases of tough fibrous tissue. The abscess varies as regards size and the direction in which it extends. It may be confined to the tissue immediately surrounding the kidney, but not infrequently the pus burrows in various directions. In the majority of cases it extends backward and opens externally in the lumbar region. The pus may make its way into the sheath of the psoas muscle and extend along the course of this muscle to Poupart's ligament, below which it may be discharged. The abscess may extend in the retro-peritoneal tissue toward the diaphragm, and may open by perforation into the pleural cavity or into the lung. The abscess may make its way behind the peritoneum into the pelvis. The peritoneum over the abscess is usually thickened. It is rare for the abscess to open into the peritoneal cavity. More frequently the colon in some part of its course is perforated and the pus is discharged into this viscus. Perinephritic abscess has been known to open also into other parts of the intestine, into the bladder, and into the pelvis of the kidney; and it may discharge its contents externally in directions other than those named, as in the gluteal region and in various parts of the abdominal parietes. When allowed to discharge its contents spontaneously, the abscess most frequently opens externally in the lumbar region. The pus in perinephritic abscess often contains shreds of necrotic tissue. It may be good and laudable or it may be thin and ichorous. The latter is particularly likely to be the case when the abscess is accompanied by infiltration of urine. In very rare instances the abscess has a gangrenous character. When the abscess is secondary to calculous pyelitis calculi may be found in the abscess-cavity if ulceration of the pelvic walls or of the kidney have occurred.

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[1] Thudichum on the *Pathology of the Urine*, and Roberts on *Urinary and Renal Diseases*, may be especially mentioned.

Of 166 cases of perinephritis collected by Nieden,[1] 70 per cent. were in males and 30 per cent. in females; 26 cases were in children. From an etiological point of view it is important to distinguish between primary and secondary perinephritic abscess. Secondary perinephritis is much more common than the primary form. The causes of primary perinephritis are traumatism (especially contusions and wounds in or about the kidney), muscular strain, exposure to cold, and severe acute general diseases. Wounds of the kidney are by no means necessarily followed by suppurative inflammation. Perinephritis has been known to develop in the course of pyæmia, of smallpox, and of typhus and of typhoid fever, but this complication is extremely rare. Secondary perinephritis is due to disease of neighboring organs. The most important of this second group of causes are affections of the kidney; above all, suppurative pyelitis and pyelo-nephritis in consequence of the irritation of calculi. Tuberculous inflammation of the kidney has also been complicated in a few cases by perinephritic abscess. Tumors of the kidney, the rupture of renal cysts, and the presence of parasites in the renal pelvis are among the rare causes of this disease. Perinephritis has been attributed to the extension of inflammation from an inflamed bladder. It has been observed more frequently to follow pelvic cellulitis, especially inflammation of the connective tissue about the uterus. Psoas abscess, which so often accompanies caries of the vertebræ, may give rise to perinephritis, but in most cases the thickened sheath of the psoas is an effectual barrier to this extension of the inflammation. Perinephritis may follow typhlitis and perityphlitis; or, in fact, any suppurative process involving the retroperitoneal tissue. Finally, there are cases of perinephritis for which no cause is discoverable.

Pain, increased by motion, is one of the prominent SYMPTOMS. It is referred to the seat of the inflammation, and shoots downward to the lower limb. It may extend to the testicle, but there is no retraction of the testicle if the kidney be not affected. There is tenderness on deep pressure. The patient is confined to the bed and lies with the limbs flexed. There is more or less increase of body-heat, with the usual concomitants of fever. Rigors and perspirations are likely to occur. After a variable period a tumor is felt in or a little below the lumbar region. Fluctuation over the site of the tumor may be felt, but this for some time is not readily appreciable. There is subcutaneous œdema over the tumor. The urine is unaffected unless the kidney be involved. With reference to the local symptoms and the diagnosis the liability of the purulent collection to descend below Poupart's ligament, to open into the intestine, the bladder, or the vagina, and in some instances into the pleural cavity and the bronchial tubes, is to be borne in mind.

The DIAGNOSIS is to be based on the presence and situation of the tumor, in conjunction with the local and general symptoms denoting inflammation. The symptoms resemble those of suppurative nephritis and pyelitis. These affections are to be excluded by examinations of the urine. In suppurative nephritis the quantity of urine is notably diminished, whereas in perinephritic abscess there is no interference with the renal secretion. The urine in the latter affection contains neither blood nor casts, and but little if any albumen. In pyelitis, with which suppurative nephritis is often associated, the urine contains pus, and often blood.

As soon as the nature of the affection is ascertained or rendered probable the tumor should be explored, and in this way the presence of pus is demonstrated. There should then be no delay in opening the abscess in order to forestall a spontaneous opening in an unfavorable direction. Bowditch has reported 3 cases in which an external opening was made, all ending in recovery. A considerable hemorrhage followed the opening in 1 of the cases. In 2 of the cases the kidney was felt by the finger at the bottom of the abscess.[1] Gibney has reported 28 cases of perinephritis in children, suppuration more or less extensive occurring in 16. In 14 of these cases the abscess was opened by incision, and in 2 cases the opening was spontaneous. With a single exception, in all of the 16 cases recovery was complete. A little lameness remained in the excepted case. Gibney is of the opinion that in a certain proportion of the cases of reputed recovery from hip-joint-disease without lameness or deformity the affection is in reality a perinephritis.[2]

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[1] "Ueber Perinephritis," *Deutsches Archiv für klin. Med.*, Bd. 22, p. 451, 1878.

CHAPTER III.

STRUCTURAL DISEASES OF THE KIDNEY.—HÆMATURIA.—HÆMOGLOBINURIA.—DIABETES INSIPIDUS AND DIABETES MELLITUS.—SEXUAL DISORDERS.

Renal Cysts.—Cystic Degeneration of the Kidneys.—Hydatids of the Kidney.—Renal Tuberculosis.—Carcinoma of the Kidney.—Movable Kidney.—Renal Hæmaturia.—Endemic Hæmaturia.—Hæmoglobinuria.—Malarial Hæmaturia.—Chyluria.—Diabetes Insipidus.—Diabetes Mellitus: Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Involuntary Seminal Emissions.—Spermatorrhœa.—Impotence.

THE kidneys are sometimes the seat of affections involving structural changes other than those already considered. Carcinoma and the tuberculous affection occasionally occur in these organs. Hydatids and cysts are additional affections in this category. Some of the structural affections of the kidney are of greater pathological than clinical interest. These will receive only brief consideration.

Renal Cysts—Cystic Degeneration of the Kidneys.

The occurrence of cysts in chronic Bright's disease, especially in the small granular kidneys, has already been mentioned. These cysts are rarely larger than a pea. They frequently contain a colloid substance. They are developed chiefly in the cortical substance. They are attributable to dilatation of Bowman's capsules and of portions of the uriniferous tubes. Similar cysts are often found in senile atrophy of the kidney. Cysts may be formed in otherwise healthy kidneys. These are of pathological rather than of clinical interest.

Sometimes the kidney is converted into a mass of cysts between which little or no renal parenchyma can be discovered. This is the *cystic kidney* or *cystic degeneration of the kidney.* Sometimes only one, but usually both kidneys are affected, although not necessarily in the same degree. Cystic degeneration of the kidney may be a congenital or an acquired condition.

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[1] *Boston Medical and Surgical Journal*, July 9, 1868.

[2] Vide articles by Dr. V. B. Gibney in the *American Journal of Obstetrics*, April, 1876; in the *American Journal of the Medical Sciences*, April, 1877, and Oct., 1878; and in the *Chicago Medical Journal*, June, 1880. To the last of these articles is added a full bibliography of this disease.

Cystic kidneys developed during intra-uterine life may attain an enormous size, so as to prove a serious mechanical obstacle to the birth of the fœtus. The severe grades of this degeneration render the fœtus non-viable, but the lighter grades can be tolerated after birth. In many cases there is some other malformation in the body. Cystic degeneration of the kidney is less frequent in adults than in the fœtus. The causes and the symptoms of this condition are not well understood. An advanced degree of cystic degeneration is sometimes found at autopsies of persons who had presented no symptoms referable to the kidneys during life. There is no doubt, however, that the functions of the kidneys may be so impaired by the cystic formations that death may result therefrom, usually with uræmic symptoms. Sometimes uræmia develops rapidly. In the recorded cases the urine has been rather increased than diminished in quantity. The specific gravity is low. Albuminuria is a frequent but not a constant symptom. The development of a tumor is an important symptom in some cases. Without this symptom the diagnosis is impossible, and with it the existence of cystic degeneration is not easily determined. Hypertrophy of the heart has been known to result from cystic metamorphosis of the kidneys. In most cases in which renal symptoms developed the affection had been considered as Bright's disease prior to the autopsy. No other mode of formation of renal cysts than by dilatation of the tubes and of Bowman's capsules has been proven.

Echinococcus or Hydatids of the Kidney.

Hydatids in the kidney are in all respects similar to those in other organs—for example, the liver (vide p. 618 *et seq.*)—and a description of them need not be here repeated. They occur in the kidney more frequently than in any other organ except the liver and the lungs, but according to Davaine they are twelve times less frequent in the kidney than in the liver. Men are oftener affected than women. The disease is rare in this country. One kidney only is affected as a rule to which there are very few exceptions. They do not, therefore, diminish the excretion of urine sufficiently to occasion uræmia. If they interfere with the excretory functions of the affected kidney, the augmented functional activity of its fellow makes up for the deficiency. So long as an hydatid cyst remains intact within the renal parenchyma, it gives rise to no well-marked symptoms, and it may remain completely latent for an indefinite period. It has been found after death when no renal trouble had been manifested during life. It may attain a sufficient size to form a tumor perceptible by palpation. The symptoms to which it gives rise depend on inflammation, ulceration, and the discharge of echinococci in different directions.

If hydatid cysts open into the pelvis of the kidney, the echinococci pass through the ureter into the bladder and are discharged with the urine. In their passage along the ureter they may give rise to the same train of symptoms as the passage of a renal calculus; in other words, to renal colic. They may also give rise to more or less trouble in their passage through the urethra. Owing to their elasticity, echinococci of considerable size may pass into the bladder and through the urethra without great difficulty and without much pain. Cases have been reported in which they were discharged in great numbers. Their passage may be accompanied by hæmaturia and pyuria. The latter is due to suppuration of the hydatid cyst or to pyelitis. The opening of the cyst is most frequently into the pelvis of the kidney. Echinococci may form the nuclei of calculi in the pelvis of the kidney or in the bladder.

The hydatid cyst sometimes develops in a direction toward the surface of the kidneys and opens into the circumrenal areolar tissue. Suppuration takes place and an abscess forms in the lumbar region, which when opened or allowed to open spontaneously gives exit to echinococci together with pus in greater or less quantity. Hydatid cysts in the kidneys have been known to open into the stomach, intestinal canal, and even into the lungs, the echinococci in the latter case being expectorated. It is possible that here, as in other situations, the parasites sometimes die, and the cyst contracts and becomes obliterated, or contains a certain amount of calcareous matter, a spontaneous cure in this way taking place. Instances of this, however, are extremely rare, and their occurrence has been denied.

The DIAGNOSIS, prior to the discharge of the contents of the hydatid cyst in some one of the situations named, can be made positive only by a microscopical examination of the liquid contained in the cyst, obtained by means of an exploratory puncture and aspiration. The diagnostic characters pertaining to the liquid are the same as in hydatids of the liver. (Vide p. 621.) The hydatid fremitus is a sign rarely available in cases of renal echinococcus. Hydatids in the kidney are sometimes not readily differentiated from hydatids in the liver or the spleen. Simon recommends an exploration by means of the hand introduced within the large intestine, in order to determine whether the hydatid tumor be renal or not.[1]

The TREATMENT is chiefly surgical. An attempt should first be made to destroy the parasite. For this simple aspiration sometimes suffices. Electrolysis is sometimes effectual. The more effective measure is opening the cyst. Simon's operation is as follows: He thrusts in two canulæ, and allows them to remain until adhesions have formed, and then he connects by incision the two openings made by the canulæ. When the tumor is large enough to cause projection of the abdominal parietes it is time to resort to surgical interference. Simon advises first to make trial of aspiration and electrolysis. These are harmless measures. If at the end of six months they be found not to have succeeded, the cyst should be opened in the manner just described.

Renal Tuberculosis—Renal Phthisis.

Tubercles appear in the kidneys in the following conditions: 1, In acute miliary tuberculosis; 2, as a secondary event in pulmonary phthisis; 3, in primary tuberculosis of the genito-urinary organs. Miliary tubercles are usually present in the kidneys in acute miliary tuberculosis. They give rise to no symptoms, and therefore do not claim clinical consideration. So, too, the few miliary tubercles or somewhat larger caseous nodules which are frequently found in the kidneys in pulmonary phthisis are without clinical significance.

There may be formed in the kidney large and small cheesy masses, accompanied usually by ulcerative destruction of the affected tissues and by similar caseous deposits in the renal pelvis, the ureter, and other parts of the genito-urinary apparatus. This condition is called renal phthisis, nephro-phthisis, or genito-urinary tuberculosis. Extensive tuberculous infiltration of the kidneys and other parts of the genito-urinary system may be secondary to pulmonary tuberculosis, to tuberculosis of bone, or to tuberculosis in other situations, but it is most frequently a primary disease. Primary genito-urinary tuberculosis is far more common in males than in females. In females primary tuberculosis of the uterus and of the Fallopian tubes is more common than primary tuberculosis of the urinary organs. The primary tuberculous deposit in genito-urinary tuberculosis in males is most frequently in the testicles, epididymis, seminal vesicles, or prostate. From these organs the tuberculosis may extend to the renal pelvis and kidney, involving usually, although not always, the bladder and ureter. Less frequently the tuberculosis extends downward from the kidney to the bladder and genital glands. In rare instances the affection is confined to the kidney and renal pelvis.

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[1] *Die Echinococcuscysten der Nieren*, Stuttgart, 1877.

In renal phthisis caseous masses usually first involve the pyramids and then extend to the cortex. These cheesy masses generally soften and form cavities, which usually, but not necessarily, communicate with the renal pelvis. The whole organ may be destroyed by this process of softening and of excavation. Sometimes only one, but usually both kidneys, are affected, although often in unequal degree. The diseased kidney is usually enlarged, but it may be shrunken. The mucous membrane of the pelvis and of the ureter is usually thickened from infiltration with fresh and caseous tuberculous material, and it presents ulcerations in some places. As in other situations, inflammatory processes are combined with the tuberculous. The lumen of the ureter, as a rule, in certain places is diminished, and as a consequence the pelvis of the kidney is dilated, so that pyonephrosis often complicates the disease. Tuberculous nodules and ulcers may be present in the bladder. There usually are tuberculous nodules in the testicles or in the prostate.

The tubercle bacilli can generally be demonstrated in the tuberculous deposits. In secondary genito-urinary tuberculosis the bacilli may be carried to one of the genito-urinary organs by the blood. It is probable that the ordinary mode of infection in primary genito-urinary tuberculosis is by the entrance of the bacilli through the urethra.

Acute miliary tuberculosis and pulmonary tuberculosis may be sequences of primary genito-urinary tuberculosis.

The SYMPTOMS of renal phthisis are generally those of a severe pyelitis and cystitis combined. In most cases there is pain in the region of one or of both kidneys. The pain is usually dull, but there may be paroxysms of severe pain. Attacks of renal colic may be caused by the passage of blood or of caseous masses along the ureter. The urine contains pus, and at times blood. The hæmaturia is generally moderate in degree, but it may be severe. The reaction of the urine is acid unless a severe cystitis be developed. Detritus consisting of cheesy particles and of connective-tissue fibres is occasionally but not frequently found in the urine. Of capital importance in the diagnosis, is the discovery of tubercle bacilli in the urine. By careful search these can be found in the majority of cases. The method for detecting them is similar to that employed in examining the sputum (p. 199). When they are discovered the diagnosis of tuberculosis involving some part of the genito-urinary tract is established, but the exact location of the tuberculosis remains to be determined by symptoms. Inoculation in the anterior chamber of a rabbit's eye with urine from cases of genito-urinary tuberculosis produces tuberculosis in the animal operated upon. This procedure has been recommended for diagnostic purposes. Physical examination of the region of the kidney will sometimes reveal a tumor, but often this examination yields only negative results. It is important to examine the testicles and the prostate gland for tuberculous infiltration. The lungs and other organs are also to be examined for tuberculous lesions. Constitutional symptoms develop sooner or later. These are—hectic fever, chills, night-sweats, progressive emaciation, and loss of strength. There may be diarrhœa with or without tuberculous intestinal ulceration. Waxy degeneration of the organs is an occasional result of renal phthisis. Uræmic symptoms do not usually develop in the course of renal tuberculosis. Bright's disease may, however, occur as an intercurrent affection.

Renal phthisis terminates fatally in the great majority of cases. The duration generally is not longer than one or two years. If the affection be localized in one kidney, recovery may take place.

The TREATMENT is directed to the general condition of the patient and to the palliation of local symptoms. The general health is to be kept up and improved as far as possible. The treatment of the local symptoms is similar to that in other cases of pyelitis and of cystitis. The general indications for treatment are similar to those in cases of pulmonary phthisis.

Carcinoma of the Kidney.

A great variety of tumors form in the kidney, but only cancer and sarcoma are of much clinical importance. Sarcoma of the kidney is infrequent, and when it gives rise to symptoms it cannot usually be distinguished from cancer. Cancer may occur in the kidney as a primary or as a secondary growth. It develops secondarily, either in the form of metastases or as a continuous growth from adjoining parts. Secondary renal cancer, although more frequent than the primary form, rarely attains a large size or gives rise to symptoms drawing attention to the kidneys. It has much less clinical importance than primary cancer, and the latter is the form to be considered in this article. Primary cancer of the kidneys is a rare disease, although less infrequent than was formerly believed. It occurs oftener in the first decade of life than in any succeeding decade, and in this it differs from cancer in other situations. Rohrer has collected 115 cases of primary renal cancer. Over one-third of the cases occurred within the first ten years of life. The next largest number of cases were those between fifty and sixty years. Notwithstanding its infrequency, cancer of the kidney is the cancer oftenest met with in childhood. Of the cases of renal cancer in children, by far the largest number fall within the first five years of life.[1] In very rare instances it has been observed as a congenital affection. In the majority of cases primary cancer of the kidney is unilateral. It may, however, develop as a primary affection in both kidneys.[2] Males are somewhat more frequently affected than females.

Renal cancer, in the great majority of cases, belongs to the soft or medullary variety. Cancer in this situation may, however, present all grades of transition between the medullary and the scirrhous forms. Colloid renal cancer has also been described. The new growth may appear either as a diffuse infiltration of the kidney or in a nodular form. There may be remnants of intact renal tissue or the entire parenchyma may be destroyed. The size of the tumor is in many cases enormous, and it is a remarkable fact that relatively, and almost absolutely, the largest tumors are met with in children. According to Sir William Roberts, the average weight in children is 8½ pounds and in adults 9½ pounds. The maximum weight was 31 pounds, and this was observed in a child. In the most frequent form of renal cancer the consistence is soft. Not infrequently cavities form containing soft, pulpy, degenerated cancerous material. From the fluctuating feel afforded by these softened masses renal cancer has often been punctured during life under the supposition that a cyst or an abscess was present. The color is not uniform. In most cases a section of the tumor presents a whitish or yellowish appearance mottled with red and brown portions. The red portions are due to dilated blood-vessels and to hemorrhages. When their number is large the red patches give a hemorrhagic aspect to the tumor, whence it derives the name fungus hæmatodes. The brown portions result from pigmentary changes in blood-extravasations. Sometimes the tumor is confined within the capsule, and at other times it invades surrounding parts. In some cases the appearance is as if the tumor took its origin in the connective tissue or the lymphatic glands about the kidney, and secondarily invaded this organ. Those who maintain the epithelial origin of cancer are unwilling to admit that it ever develops primarily in the perinephritic tissue. The tumor usually contracts adhesions to surrounding parts, but sometimes a cancerous kidney is movable. In nearly all cases the colon lies in front of the tumor. Secondary cancerous growths are found in other organs in rather less than half the cases of cancer of the kidney. It is a peculiarity of primary renal cancer, especially in children, that it gives rise to metastases less readily than cancer in other situations.[1] The secondary deposits are met with most frequently in the retro-peritoneal glands, the lungs, and the liver. The frequency with which the renal vein is involved in the new growth explains the common occurrence of secondary cancerous deposits in the lungs, as by this implication of the veins cancerous material may gain access to the blood-current. Metastases are rare in the lower urinary passages, but cancer of the kidney frequently grows continuously into the renal pelvis, and it may invade the ureter. It is not rare to meet with calculi in the pelvis of a cancerous kidney. It is exceptional for renal carcinoma to cause ulceration of the superjacent integument. The intestine, especially the overlying colon, may be perforated by the cancerous growth. Intestinal fistulæ opening externally have been caused by this disease. The vertebræ in some cases are involved in the malignant growth. Pressure upon the inferior vena cava may cause œdema of the lower extremities. Ascites is rare. Displacements and distortions of the abdominal organs are often the result of pressure from cancerous tumors of the kidney. The diaphragm may be pushed upward and the lungs compressed. If the disease be unilateral the kidney unaffected by cancer is generally hyperæmic. Amyloid degeneration of the kidney, as well as of other parts, may be caused by renal cancer.

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[1] Rohrer, *Das primäre Nierencarcinom*, Inaug. Diss., Zurich, 1874. Of 107 cases in which the age of the patient was stated, 37 were less than ten years, 4 were between ten and twenty, 5 between twenty and thirty, 10 between thirty and forty, 10 between forty and fifty, 17 between fifty and sixty, 10 between sixty and seventy, 3 between seventy and eighty, and 11 were described simply as adults. Of 29 cases of cancer in children collected by Hirschsprung (1868), 15 were of the kidney: 82 per cent. of all renal cancers in childhood fall within the first four years of life (Rohrer).

[2] Of 114 cases, the cancer was 52 times on the right side, 50 times on the left, and 12 times bilateral. In children the left kidney was oftener affected than the right (2½ : 2). In adults, the right oftener than the left (5 : 4). Bilateral cancer was of equal frequency in children and in adults (Rohrer).

Upon microscopical examination the tumor presents the arrangement of cells in alveoli with intervening stroma, which was described as characteristic of cancer in Part I. p. 44 *et seq.* The microscopical appearances as regards the size of the alveoli, the shape of the cancer-cells, the amount and character of the stroma, the degree of vascularity, vary in different cases. The designation of the tumor as medullary or as scirrhous depends upon these variations, as has also been explained in the article on Carcinoma in Part I. In the soft renal cancers, which is the most frequent form, the alveoli are separated by a small amount of stroma and are usually large. It is a peculiarity of soft cancer of the kidney that the alveoli may be separated from each other in some places by nothing but blood-vessels, the appearance being that blood-vessels actually penetrate the alveoli. The stroma of these cancers in its scanty amount of fibrillated connective tissue resembles the normal interstitial tissue of the kidney. The stroma is not in all places as thin as has been described, and it may be abundant. In some cases the cancer-cells resemble the epithelium of the convoluted tubes, being very granular and indistinctly separated from each other. They are, however, always irregular in shape. Cylindrical-celled epithelioma of the kidney has been described by Wagner. Waldeyer, Klebs, Pereverseff, and others have described with much detail the development of cancerous alveoli from the uriniferous tubes, and of cancer-cells from the epithelium of these tubes, the tubes becoming dilated and irregular in shape, and giving off processes; and within these dilated branching tubes the epithelial cells proliferate. In the immediate neighborhood of the cancerous growth the interstitial tissue of the kidney is often increased in amount, and the tubes are in some places compressed and in others dilated. Cancer seems to take its origin more frequently from the cortical than from the medullary tubes.

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[1] According to Kühn, secondary growths are found in 28 per cent. of the cases in children, and in 44 per cent. in adults (*loc. cit.*).

In a clinical point of view, as already stated, secondary cancer of the kidneys has little interest. It is generally latent as regards diagnostic symptoms. Both kidneys are affected, as the rule. The disease does not advance sufficiently to form appreciable tumors, and there may be no symptoms denoting renal disturbance. Primary cancer, on the other hand, is generally unilateral, and in a large proportion of cases it gives rise to symptoms which direct attention to the kidney. Lumbar pain on the affected side, extending down the ureter to the thigh, may be a more or less prominent symptom, but it is not so except in a certain proportion of cases; and this symptom for a long time may be wanting. There are loss of appetite, progressive emaciation, and other symptoms which belong to the cancerous cachexia and denote an inroad upon the constitution, but they may be long deferred. The diagnosis hinges upon the discovery of a renal tumor.

The tumor is felt between the ribs and the pelvis latero-anteriorly. It varies greatly in size and form in different cases. It may be as large as the closed hand or a child's head, and in young subjects it sometimes attains enormous dimensions, as in the case cited by Roberts, in which the weight of the tumor was 31 pounds. In the pictorial representation of that case the enormously enlarged abdomen contrasts strikingly with the attenuated chest and limbs. The tumor may be smooth or lobulated. The aortic impulses may cause it to pulsate, resembling in this respect an aneurismal tumor. It may be hard and resisting to the touch or it may give a sense of fluctuation. It is sometimes hard to the touch in some and soft in other parts. The tumor sometimes retains the shape of the kidney, although much enlarged, but oftener there is so much distortion that the shape is of no value in the diagnosis. The cancerous disease may extend to the neighboring lymphatic glands, and these may form a mass which may exceed in size the enlarged kidney.

The first point with reference to the DIAGNOSIS is to determine that the tumor is renal. It is to be discriminated from enlargement of the liver and spleen, from an ovarian and from a fecal tumor. It is shown not to be connected with the liver or the spleen if the fingers can be pressed between the lower margin of these organs and the tumor. The evidence afforded by percussion must not be too confidently relied upon. The renal tumor is usually behind the intestine, and therefore a tympanitic resonance is often obtained by percussing over it; but it is common to find this resonance over the liver and the spleen, owing to its being conducted from the colon and stomach upward to a greater or less height. Hence the resonance over the liver and over the spleen may be tympanitic, although no part of the intestine is situated over these organs. On the other hand, the portion of intestine situated over a renal tumor may not at all times contain gas, and there may then be flatness on percussion. Too much reliance on a tympanitic resonance over an abdominal tumor as a differential point is likely to lead to error in the diagnosis. A cancerous kidney usually becomes closely adherent to adjacent parts, and it is not, like the liver and spleen, lowered by the descent of the diaphragm in inspiration. An ovarian tumor differs in being found at first in the iliac fossa, and as it increases in size it extends upward, whereas an enlarged kidney is at first felt in the hypochondrium, and as it enlarges it descends. For further diagnostic evidence of ovarian tumors the reader is referred to works on gynæcology. Fecal tumors have their characteristics which have been considered (p. 506).

A renal cancer is to be discriminated from other affections of the kidney —namely, hydronephrosis, pyonephrosis, renal abscess, hydatid disease, and cystic degeneration. Relying upon palpable characters of the tumor this is by no means easy. With reference to this differentiation the antecedent and concomitant symptoms are to be considered, and, as bearing on both the seat and the character of the tumor, hæmaturia is pre-eminently a diagnostic symptom.

Hæmaturia and the existence of a tumor referable to the kidney render the diagnosis of renal cancer almost positive. The analysis, however, of 59 cases by Sir William Roberts gave 28 in which hæmaturia did not occur, and of the larger collection of cases by Rohrer this symptom was present in only 34 per cent. The absence of hæmaturia therefore by no means excludes cancer. Hæmaturia, too, without tumor is not evidence of renal cancer. It is the conjunction of the hæmaturia and the tumor which establishes the diagnosis, and a positive diagnosis is not possible unless these two events be conjoined.[1]

The hæmaturia may occur for the first time early or late in the progress of the disease. It often recurs more or less frequently after irregular intervals. The hemorrhage is sometimes profuse, and sometimes it is small. Clots formed in the calices or the pelvis of the kidney in passing through the ureter may give rise to all the symptoms which attend renal colic. The coagula in some instances become moulded in the ureter, and appear in the urine in the form of casts resembling lumbricoid worms. Many of these were passed in a case which came under my observation. The coagula may obstruct the ureter and give rise to hydronephrosis. The blood sometimes coagulates in the bladder, and the coagula are with more or less difficulty expelled through the urethra.

The mistake is often made of supposing that the urine may contain cells distinctive of cancer. It is perhaps true that cells from the tumor may be contained in the urine, but these cells have no characters by which they can be distinguished as cancer-cells. The normal epithelial cells lining the urinary bladder present such a great variety of forms that they are often taken as the physiological type or paradigm of cancerous cells. There is no instance of the diagnosis having been based upon the recognition, in the urine, of masses of cancerous tissue of sufficient size to enable an alveolar structure to be determined. If blood or pus be contained in the urine, there is of course a certain amount of albuminuria; but if these elements be absent renal cancer may exist without any trace of albumen in the urine. The disease may, however, be attended by changes in the kidney which give rise to considerable albuminuria.

The TERMINATION in cases of renal cancer is inevitably in death, sooner or later. The duration of the disease in children is shorter than in adults. Sir

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[1] With reference to these two diagnostic events—namely, tumor and hæmaturia—Rohrer divides his 115 cases into four classes, as follows: 1, in 36 cases there was neither tumor nor hæmaturia, and the disease was therefore unsuspected; 2, in 12 cases there was hæmaturia without tumor; 3, in 25 cases there were both tumor and hæmaturia; 4, in 42 cases there was tumor without hæmaturia.

William Roberts found the average duration in children to be about seven months, the extremes being ten weeks and over a year. In adults the average duration was two and a half years, and the extremes five months and seven years. Rohrer from his statistics reaches similar results. It is to be observed that in the cases embraced in these statistical analyses the latent period of the disease when there were no symptoms present cannot be taken into account, and there is reason to believe that this latent period may be of long duration. Wharry mentions a case which proved to be one of renal cancer in which a tumor, gradually increasing in size, had been ascertained fourteen years before death.[1] Dunlop has reported two cases, the duration having been ascertained to have been in one between ten and twelve years and in the other sixteen years.[2] Rohrer cites a case which lasted seventeen years. These are, of course, exceptional cases, but it is a noteworthy fact that cancer in this situation is tolerated in some instances for a very long period, and a great length of time may elapse without much constitutional disturbance. On the other hand, in some cases, especially in children, the tumor grows with astonishing rapidity.

It is a peculiar feature of primary cancer seated in the kidney that the tendency is much less here than in other situations to extend to neighboring parts or to give rise to metastatic cancerous disease in situations more or less remote. Heredity seems not to be involved in the causation of the disease in this situation. Respecting the causation nothing is known.

The indications for TREATMENT have reference in some cases to lumbar pain, to the occurrence of renal colic, to urethral obstruction from clots, to the removal of coagula from the bladder, to hæmaturia when the hemorrhage is profuse, and to other effects, symptoms, or complications which are liable to occur during the progress of the disease. Aside from meeting these indications, the object of treatment is to render the system tolerant of the disease as completely and as long as possible. The measures for this object are the same as in other diseases in the management of which all that is to be hoped for is retardation of their progress and of their inroads upon the constitution. These measures embrace tonic remedies, palliatives, nutritious diet, and favorable hygienic influences, moral and physical, in the place of those which tend to impair the ability to endure an incurable disease. The only hope of recovery is to be sought in extirpation of the tumor. This operation has been performed a number of times, but thus far not with favorable results.

Movable Kidney.

One of the kidneys is sometimes loosened so as to form a movable tumor which may be felt through the abdominal walls. This condition is called movable kidney, floating kidney, wandering kidney, or ectopia renis. The degree of mobility varies much in different cases. The kidney is normally held in position by the layer of peritoneum which is attached to the anterior surface of its adipose capsule. In movable kidney the adipose tissue in which the normal kidney is imbedded partly or wholly disappears. The overlying peritoneum becomes lax and may form a partial or even complete investment of the kidney. In extreme cases the mobility of the organ is restrained only by its vessels and the ureter. Unusual length of the renal vessels is thought to be a predisposing cause of displacement of the kidney. In nearly all cases the intestine is interposed between a movable kidney and the abdominal walls. In very exceptional cases the organ has been found in front of the intestine.

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[1] *The Lancet*, Feb. 10, 1877.

[2] *Ibid.*, April 21, 1877.

A displaced kidney may become fixed in its abnormal position by the formation of adhesions.

It is difficult to form an estimate of the frequency of this abnormality of the kidneys, as the condition doubtless often exists without being recognized. It is probable that abnormal mobility of these organs is not very uncommon. It is far more frequent in women than in men. Of 290 cases collected by Newman, 252 were in women and 38 in men.[1] The right kidney is movable oftener than the left. Both organs may be affected. Of 91 cases brought together by Ebstein, the right kidney was affected 65 times, the left 14 times, and both kidneys 12 times.[2] The affection may be congenital or acquired. It is usually acquired. In most of the cases the abnormality developed between the twentieth and the forty-fifth year, or, in other words, during the child-bearing period in women. The affection may, however, appear at any age. Mobility of the kidney is attributable to relaxation of the abdominal walls from pregnancies, to the use of corsets and tight girdles about the waist, to violence, to increased weight of the organ from disease, to the pressure of tumors growing in the neighborhood of the kidney, and to the traction of hernias. The disappearance of the fat from the adipose capsule of the kidney, as happens in wasting diseases, has also been assigned as a cause, but its efficacy is doubtful. Unusual laxity of the abdominal walls from causes other than pregnancy may be an element in the causation. In congenital floating kidney there is usually some abnormality in the development of the peritoneum covering this organ or in the length, origin, or distribution of the renal vessels.

Movable kidney may exist without causing any noticeable SYMPTOMS. In several cases it has been discovered accidentally by the physician while making a physical examination of the abdomen. Usually, however, the condition gives rise to more or less uneasiness. The patient often experiences a sense of weight and dragging pain, especially when walking or standing. There may be severe neuralgic pain radiating from the affected side. If the tumor be first discovered by the patient, it becomes often a source of great mental anxiety. In some cases the movable kidney gives rise to the paroxysmal development of symptoms of considerable severity to which the name "symptoms of incarceration" was applied by Dietl. These symptoms are chilliness, nausea, vomiting, prostration, intense pain, and anxiety. The affected kidney and surrounding tissues are swollen and sensitive to pressure. These symptoms usually subside within a few days under the influence of rest and opiates. They are relieved if the kidney be replaced. The explanation of the symptoms of incarceration is not satisfactorily established. The symptoms have been ascribed to twisting of the ureter and to retention of the urine in the pelvis of the organ from the temporary obstruction thereby occasioned. In this way it is claimed that acute hydronephrosis and pyelitis may develop. Hydronephrosis may coexist with movable kidney, but this explanation does not seem to be applicable to all cases. The symptoms have more frequently been attributed to inflammation of the peritoneum and connective tissue surrounding the kidney. Another possible explanation is that the symptoms are due to partial strangulation of the kidney from compression or from twisting of its blood-vessels. It is claimed that a movable right kidney pressing on the duodenum may be a cause of dilatation of the stomach. Constipation may be a result of compression of some part of the intestine, most frequently of the colon, by a movable kidney. Œdema of the lower extremities has been described as a result of compression of the inferior vena cava by a displaced kidney. According to Roberts, epigastric pulsation is in some cases a prominent phenomenon in connection with movable kidney. Various symptoms resembling hysteria have been frequently observed in women suffering from this disorder.

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[1] *Glasgow Medical Journal*, Aug., 1883.

[2] Of 65 cases collected by Roberts, in 42 the right alone was movable, in 9 the left alone, and in 14 both kidneys.

The occurrence of movable kidney is to be recollected in determining the nature of obscure tumors within the abdomen. The recognition of this variety of tumor is to be based on the following diagnostic points: It is situated in the hypochondriac region. It has the size and the shape of the normal kidney, and this may be determinable by palpation, which is most advantageously employed by placing one hand over the lumbar region and the other in front on the abdominal walls, and then making counter-pressure from one hand to the other. It is generally movable, and in some cases the organ can be restored to its proper situation. Over the normal site of the organ on the affected side a depression is sometimes observed, with a tympanitic resonance on percussion derived from the portion of the intestine which is situated in the place which the kidney should have occupied. These two latter diagnostic points are available in so few cases that they are of more theoretical than practical interest. Other tumors are to be excluded by the absence of their diagnostic characters.

The inconvenience, and sometimes suffering, attending this anomaly may often be obviated, in a measure at least, by mechanical compression of the abdomen by means of a bandage or belt or abdominal supporter. In attacks of pain the organ should if possible be replaced; rest and recumbency are to be enjoined; emollient applications should be made over the abdomen; and if these measures do not afford relief opiates are indicated. It very rarely happens that the symptoms are so severe as to justify extirpation of a movable kidney, although this operation has been repeatedly performed. An operation for fixation of the kidney by means of sutures has been devised (nephroraphy), but this also is open to many objections.

Renal Hæmaturia.

Hæmaturia, or bloody urine, occurs in various diseases, as well as after wounds and other injuries of different parts of the urinary apparatus. The blood may come from the kidney, the pelvis of the kidney, the ureter, the bladder, or the urethra. When it takes place from the bladder or the urethra, it comes within the province of surgery; but the means of determining whether the hemorrhage be either cystic or urethral are of importance to the physician. Urethral hemorrhage is easily discriminated. The blood escapes without any effort of micturition or it immediately precedes the first escape of urine. Moreover, the seat of the disease or injury may be ascertained by surgical exploration. Vesical hemorrhage is discriminated by attention to the following points: The last part of the urine discharged is often more bloody than the first part; there usually are blood-clots in the urine, and these may prove an obstacle to the free escape of the urine; there can frequently be found some cause of vesical hemorrhage, such as a calculus, a tumor, or some wound or injury of the bladder; and ammoniacal decomposition of the urine and other symptoms of cystitis are often present.

Hemorrhage from the ureter generally involves the presence of a calculus. Hemorrhage from the pelvis of the kidney is most frequently due either to calculus or to tuberculous pyelitis, and occasionally to infectious diseases with a hemorrhagic tendency. Sometimes coagulated blood is moulded in its passage through the ureter so as to form casts of this tube, compared in their appearance to leeches.

When the bleeding is from the kidney the blood is thoroughly mixed with the urine, which often presents a smoky or sooty appearance. Blood derived from renal hemorrhage is very rarely coagulated. Of great importance in the detection of renal hemorrhage is the recognition of changes in the urine indicative of disease of the kidney, especially casts and a much larger proportion of albumen than can be explained by the quantity of blood present. The presence of blood-casts in the urinary sediment is proof that the hemorrhage is renal. The reaction of the urine is generally acid. It is to be borne in mind that blood-corpuscles may not be discoverable in urine which has become ammoniacal, as under these circumstances they may be speedily destroyed. In urine of low specific gravity the blood-corpuscles appear very pale, or only the so-called shadows of blood-corpuscles may be present. If, owing to the absence of blood-corpuscles, there be doubt as to the presence of blood-coloring matter in the urine, chemical tests[1] or the spectroscope may be employed. Bloody urine is always more or less albuminous.

Bloody urine should not be confounded with any of the following conditions: the high-colored, saturated urine of fevers, the urine of jaundice, the dark urine from carbolic-acid poisoning, and urine stained by ingestion of rhubarb, senna, logwood, madder, beet-root, the prickly pear, and fuchsin.

The causes of renal hemorrhage are injuries to the kidney, as from wounds or from a fall or a blow; the absorption of various toxic substances, such as cantharides, turpentine, or oil of mustard; congestion of the kidney due to these or other causes; acute, less frequently chronic, Bright's disease; rarely embolism, thrombosis, or aneurism of the renal blood-vessels; abscess, echinococcus, tuberculosis, and cancer (rarely other tumors) of the kidney; occasionally acute infectious diseases; and the hemorrhagic diathesis, including purpura and scurvy. Sometimes hæmaturia, apparently of renal origin, occurs without any apparent cause. The influence of malaria and of parasites in the production of hæmaturia will be considered in the following articles.

In cases of renal hæmaturia, if the hemorrhage be considerable, the application of cold over the kidneys by means of compresses wet in cold water or the ice-bag is indicated. Internal hæmostatics which may be prescribed are tannic or gallic acid, ergot, the muriate and other astringent preparations of iron, alum, and acetate of lead. Hypodermic injections of ergotin may be employed. Aside from the treatment having reference to the hemorrhage, indications are to be derived from the diseases or morbid conditions with which it is connected as a symptom.

Endemic Hæmaturia.

This name is applied to a form of hæmaturia occurring in Brazil, Egypt, the Cape of Good Hope, and some other tropical countries. The hæmaturia is caused by the distoma hæmatobium, a parasite which is common in these countries, and which was discovered by Bilharz in 1851. This parasite, which is one of the suctorial worms or trematodes, is about half an inch in length, and is found especially in the portal and other abdominal veins. The parasite is thought to be ingested with the drinking-water. It produces a multitude of eggs, which are found in the liver, the intestine, the bladder, ureter, renal pelvis, kidney, and other organs. The lodgment of these ova produces symptoms referable especially to the liver, the intestine, and the urinary organs. The mucous and submucous coats of the bladder, and of the ureter and renal pelvis are thickened, inflamed, and ulcerated. The local effects of the parasite and its ova upon the urinary organs are manifested in hæmaturia, combined generally with symptoms of cystitis and of pyelitis. There frequently is diarrhœa, and there may be pyrexia with symptoms of typhoid fever or septicæmia. The ova often form the nucleus of a renal or a vesical calculus.

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[1] Teichmann's blood-test is convenient. Some of the urinary sediment or a portion of the dried urine on a slide is treated with glacial acetic acid after the addition of a few minute crystals of common salt. The specimen is covered with a cover-glass and heated until it begins to boil. After cooling (more glacial acetic acid can be added if necessary) the characteristic brownish rhombic hæmin crystals appear if blood be present.

The diagnosis is based upon finding the eggs in the urine. The eggs are from $\frac{1}{180}$ to $\frac{1}{160}$ of an inch in length, and are either sharply pointed or furnished with a projecting spine near one end.

The disease is a severe one and may terminate fatally. Recovery, if it take place, is slow, generally extending over several years.

No remedy has been discovered which will destroy the parasite. The indications are to remove the patient from the endemic influence, to support the strength by tonics and good hygiene, and to treat the cystitis. For the latter purpose the bladder should be cleansed, and such drugs as boric acid, benzoic acid, bicarbonate of potash, and infusion of buchu may be employed.

According to observations in China and in India, hæmaturia may be caused by the filaria sanguinis hominis.

The strongylus gigas is a long, cylindrical, red worm found sometimes in the pelvis of the dog's and of the horse's kidney, but is so extremely rare in human beings that it does not require consideration.

Hæmoglobinuria—Hæmatinuria.

If from any cause the red blood-corpuscles be dissolved in the blood, the hæmoglobin which is thus set free is excreted by the kidney. The condition of blood thus produced is called hæmoglobinæmia, and that of the urine hæmoglobinuria. The urine in hæmoglobinuria contains, therefore, dissolved blood-coloring matter, but usually no red blood-corpuscles. The urine has a reddish-brown color, which in extreme cases is compared to the color of porter.

Hæmoglobinæmia and the consequent hæmoglobinuria may be produced by a variety of causes. A large number of substances when taken into the blood cause a dissolution of the red blood-corpuscles. Such substances are pyrogallic acid, naphthol, arseniuretted hydrogen, glycerin, hydrochloric acid, sulphuric acid, chlorate of potash, and the biliary salts. The ingestion of certain poisonous fungi has this effect, as well as the transfusion of the blood of one species of animal into the blood-vessels of another species. Hæmoglobinuria has been observed in the course of various infectious diseases, such as scarlet fever, typhoid fever, and malaria. It is also produced by extensive superficial burns, and in one form of paroxysmal hæmoglobinuria exposure to cold is an important cause.

In all cases of hæmoglobinuria which have terminated fatally nephritis has been found. This nephritis is to be regarded as secondary. There are glomerulo-nephritis, degeneration and necrosis of the epithelial cells of the tubes, small-celled infiltration, and especially characteristic casts in the tubes composed of reddish-brown pigment-granules.

Hæmoglobinuria is distinguished from genuine hæmaturia by microscopical examination, which shows the absence of red blood-corpuscles. The presence of blood-coloring matter is made evident by spectroscopic or chemical examination, as mentioned in the article on Hæmaturia. The urinary sediment usually contains formless, granular (less frequently crystalline) reddish-brown pigment, and casts of which the most characteristic are those containing or composed of similar pigment. The urine is always albuminous. Of course care must be taken not to mistake for hæmoglobinuria urine in which the blood-corpuscles have been dissolved in consequence of ammoniacal decomposition.

Experiments have shown that fragments of red blood-corpuscles are not only excreted by the kidney, but are also taken up by the spleen, the liver, and the marrow of the bones. The spleen becomes thereby swollen, and the liver excretes an increased amount of bile, which may have an abnormally thick consistence. The jaundice which is a frequent attendant of hæmoglobinæmia is probably to be regarded as hæmatogenous, although the changes in the liver which have been mentioned might be adduced in favor of its hepatogenous origin.

There is a form of hæmoglobinuria which deserves consideration as an independent disease. This is the so-called paroxysmal hæmoglobinuria. Some of these cases are of malarial origin and others are not. On account of its importance malarial hæmoglobinuria will receive separate consideration.

Paroxysmal Hæmoglobinuria.

Exclusive of its causation by malaria in certain regions, this affection is not common. It may occur in persons otherwise apparently healthy. As the name implies, the disease appears in paroxysms. A typical paroxysm is ushered in by pain in the back, the limbs, and the head, sometimes by nausea and vomiting and a sensation of coldness in the extremities. Frequent yawning has been noted as a premonitory symptom in several cases. There is usually a distinct chill, followed by rise of temperature and sweating. During this attack, and generally for a few hours afterward, the urine acquires a dark reddish-brown, sometimes almost black, color, is generally of acid reaction, and is of rather low specific gravity. While the spectroscope reveals the absorption-bands of hæmoglobin and of meth-hæmoglobin, the microscope shows the absence of red blood-corpuscles. There is generally a chocolate-colored sediment containing pigment-granules and hyaline, epithelial, and pigment casts. There is at the same time albuminuria. Severe colicky pain in the region of the liver is sometimes experienced during the attack. A more or less distinct icteric hue of the skin is present. Urticaria in some cases, and exceptionally purpuric patches, have been noted. After the paroxysm the urine gradually becomes clearer, and in from twelve to twenty-four hours it becomes normal. Meanwhile, the other symptoms diminish and disappear, and on the day following that of the paroxysm, except the weakness due to the latter, the usual health of the patient is restored.

With a light paroxysm there is only chilliness, followed by little or no fever, and the symptoms may not be sufficient to interrupt the usual occupation of the patient. Rarely the hæmoglobinuria appears without marked premonitory symptoms.

The paroxysms may occur with some regularity, but more frequently they recur irregularly at variable intervals. Two or more daily sometimes occur, or the intervals may be days, weeks, or months. In proportion as the paroxysms are frequent and severe patients present a pale, cachectic aspect.

Examination of the blood withdrawn during the paroxysm by wet cups has shown a reddish color of the serum, indicating solution of the hæmoglobin. The red blood-corpuscles have been found sometimes altered in shape and size, and the so-called shadows of the corpuscles have been detected in the blood. The number of red blood-corpuscles is diminished. There is less tendency than normal in the red corpuscles to form rouleaux.

In a large number of cases the causation of the paroxysm has been attributed directly to exposure to cold, but of the *modus operandi* of this cause we possess no definite knowledge. In persons subject to the disease attacks have been repeatedly produced experimentally by plunging the feet or hands into cold water. It is not improbable that the red blood-corpuscles in these individuals possess some peculiarity in composition which renders them abnormally susceptible to the action of cold.[1] All cases of paroxysmal hæmoglobinuria, however, do not seem to possess this peculiar susceptibility to cold. Some cases are referred to muscular exercise. The disease is more common in men than in women. Most of the paroxysms occur in winter. A large proportion of the patients have had syphilis. That malaria has an important influence in the causation of periodical hæmoglobinuria in this country will be made evident in the following article.

Exclusive of malarial cases, paroxysmal hæmoglobinuria does not involve danger to life. Paroxysms may recur for an indefinite period, but recovery may be expected to take place sooner or later.

The TREATMENT during the paroxysm consists in placing the patient in bed, applying heat to the surface, and giving hot drinks internally, with the addition of some form of alcoholic stimulant. After a paroxysm measures to maintain warmth of the surface are of prime importance. A rough, changeable climate may be exchanged with advantage for one which is mild and uniform. Only in a few cases, in which a syphilitic history has existed, has mercurial treatment been found effective. Even in non-malarial cases quinine is often beneficial. Chalybeates and other tonics, with good alimentation and the moderate use of wine, are to be prescribed according to the indications in individual cases.

Malarial Hæmaturia—Malarial Hæmoglobinuria.

Both hæmaturia and hæmoglobinuria may be caused by malarial poisoning. In the Northern and Middle States of this country only the milder forms of malarial hæmaturia are observed, and these are not common, whereas in parts of the Southern and South-western States and in tropical countries malignant malarial hæmoglobinuria is not uncommon.

In the *milder* cases the affection may be either hæmaturia or hæmoglobinuria. The diagnosis is based upon the history of malaria. The attacks of hæmaturia may occur with the regularity of malarial paroxysms, but more frequently they are variable in the time of their appearance. In some instances the hæmaturia becomes continuous, although fluctuating in intensity. The ordinary symptoms of intermittent fever may or may not be present at the time of the hæmaturia. The paroxysms may resemble those described in the preceding article, and they may likewise be caused by exposure to cold.

Malignant malarial hæmoglobinuria is one of the manifestations of pernicious malarial fever, and is a disease of great gravity. During the last twenty-five years a large number of cases have been observed in parts of the South and South-west of this country. The disease may be preceded by one or more paroxysms of intermittent fever, or the hæmoglobinuria may be developed suddenly. There is always a history of exposure to malarial influences. The urine is of a dark reddish-brown, sometimes almost black, color. It contains few or no intact red blood-corpuscles, but an abundance of hæmoglobin and meth-hæmoglobin, as is shown by the production of hæmin crystals or by spectroscopic examination. The sediment contains hyaline and epithelial casts, granular matter, and granules of blood-pigment, both free and in casts. The urine is albuminous and generally acid. At first it is usually excessive in amount, and later it is diminished. In addition to the hæmoglobinuria, there are other symptoms of gravity. After a few hours the patient becomes jaundiced. Usually at the onset of the attack there are nausea and vomiting, and often black vomit makes its appearance. There may be bleeding from the nose, from the bowels, from the uterus, from blistered surfaces, etc. Pain in the back and in the epigastric region is often complained of. The temperature is elevated, sometimes as high as 106°; the pulse is rapid, small, and feeble; and there is great prostration. There may be delirium, and frequently the patient passes into a state of somnolence and coma. The mind may, however, remain unaffected. Death may occur during the first attack, or the symptoms may be relieved, and then recur after one or two days, attended in all cases with great danger to life.

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[1] This view is supported by the experiment of Ehrlich and of Boas, who, in a case of paroxysmal hæmoglobinuria, after applying an elastic ligature around the root of one of the fingers, plunged this finger into ice-water for a quarter of an hour. In blood withdrawn then from this finger they observed the changes in the serum and red blood-corpuscles above noted, while blood taken from other parts of the body did not present these changes.

Malignant malarial hæmoglobinuria seems to be more common in males than in females. It is said that negroes are exempt from this form of the disease.

The PROGNOSIS is grave, but under appropriate treatment recovery may take place.

The TREATMENT of the malignant form consists in the administration of quinine in large doses and hypodermically. Of great importance is the administration of stimulants to maintain the vital forces, which are often at a low ebb. Morphine may be employed to relieve the gastric symptoms. Physicians in the South consider stimulants and restoratives of equal importance with quinine in the treatment of this affection.

The milder forms of malarial hæmaturia are often relieved or cured by quinine. The ordinary astringent remedies may be employed in conjunction with quinine. All severe muscular exertion should be avoided. For cases which are rebellious to treatment—and such cases are not very rare—Tyson recommends the use of chalybeate and alum springs.[1] Iron and appropriate hygienic treatment are to be used to counteract the anæmia attending or following malarial hæmaturia.

Chyluria.

This disease is characterized by the passage of urine which resembles milk. The urine has sometimes a rose or pink tinge, due to the presence of a little blood, and the disease is sometimes associated distinctly with hæmaturia. In many instances the urine has the opaque white appearance of milk, without any admixture with blood. After its passage the urine generally forms a jelly-like coagulum which disintegrates in a few hours. The microscope shows the presence of fatty matter in the form of very minute granules. The fatty matter is dissolved by ether, and the urine which remains assumes a normal appearance. The urine contains albumen, usually in large amount. The abnormal chemical constituents of the urine, therefore, in chyluria are fat, albumen, and fibrin. To these blood is frequently added.

Chyluria prevails in certain tropical and subtropical countries. It is observed in China, the East and West Indies, South America, Africa, Australia, and some tropical islands. In the cases which have fallen under observation in temperate climates the disease, as a rule, has been contracted in a hot climate. Chyluria, however, has sometimes appeared in persons in this country and Canada, as well as in England and Germany, who have never visited tropical regions.[1]

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[1] James Tyson, article "Malarial Hæmaturia and Hæmoglobinuria," in *Pepper's System of Practical Medicine, by American Authors*, Philadelphia, 1886, vol. iv. p. 111.

The milky urine in most cases appears without any premonition or apparent exciting cause. All of the urine may be chylous, or it may part of the time be normal. Chyluria usually occurs intermittingly, the intervals in different cases varying from days to months or years. There is no regularity as regards either the duration of the attacks or their recurrence. In the intervals between the attacks there is no evidence of urinary disease.

In severe cases there are lumbar pains and symptoms denoting exhaustion. Dysuria from coagula in the bladder sometimes occurs. Milder cases are not only exempt from grave symptoms, but there may be nothing which points to any disease, aside from the passage, after irregular intervals, of milky urine.

Tropical chyluria is caused by the presence of the parasite called *filaria sanguinis hominis*, first accurately described by Timothy Lewis in 1872. The adult or parent worms are cylindrical, thin, smooth nematode worms three or four inches in length. The sexes are distinct. The female gives birth to an enormous number of living embryos without sexual distinction. In the disease under consideration the parent worms are lodged somewhere in the body, probably in the lymphatics. The embryos make excursions into the blood and lymphatic vessels, where they are found, sometimes in enormous numbers.[2] These embryos are from $\frac{1}{125}$ to $\frac{1}{70}$ of an inch long, and about as broad as a red blood-corpuscle. They are elongated, generally enclosed in a membranous sheath, and endowed with the property of a rapid coiling and twisting motion. In a drop of blood they can often be detected with a low power of the microscope by the motion which their lashing imparts to the red blood-corpuscles. In chyluria these embryonic filariæ are found in the urine and also in the blood. They have the singular habit of disappearing from the blood during the daytime, and appearing in large numbers at night, the maximum number being usually found about midnight. What becomes of them in the daytime is not known. By reversing the patient's habits—that is, by making him sleep during the day and eat and move about during the night—the migrations of the parasites become diurnal instead of nocturnal.

In what way chyluria is produced in general, and what the relations of the filariæ are to this disease, are problems which have not been satisfactorily solved. The common theory is that chyluria is caused by some abnormal communication between the lymph-vessels and the urinary passages. It is suspected that the filarial worms are often lodged in the thoracic duct or abdominal lymphatics, and thus cause a damming back of lymph and consequent distension of the lymphatics.[3] Some think that the blood contains at times in chyluria an excess of chyle.

Chyluria is not the only affection which may be caused by the filaria sanguinis hominis. This parasite may be the cause also of hæmaturia, chylous hydrocele, chylous ascites, elephantiasis, milk scrotum, and nævus-like dilatations of the lymphatics in various situations, particularly upon the external surface of the body, from which a chylo-serous fluid may escape. Abscesses are sometimes produced in which the parent worms have been found. These various affections may be combined with chyluria.

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[1] Dr. Guiteras has found the filaria sanguinis hominis in two persons in Charleston, South Carolina. In one case the patient had always lived in Charleston or its neighborhood, and in the other case the patient had always lived in Charleston with the exception of five years in Augusta, Ga. One was a case of chyluria, the other one of chylocele (John Guiteras, *The Medical News*, Apr. 10, 1886).

[2] Stephen Mackenzie estimated that there were from 36,000,000 to 40,000,000 embryo filariæ in the blood of a patient whom he observed.

[3] In Mackenzie's case an enormous dilatation of the lower part of the thoracic duct and of the renal and some other abdominal lymphatic vessels was found after death, which occurred from pyæmia, the parent worms having died some time before and not being found at the autopsy. In the few other reported autopsies no changes have been found in the urinary organs or lymphatics to explain the chyluria.

According to Manson of China, the embryonic filariæ are sucked up with the blood from patients affected with the filaria disease by mosquitos, in which the parasite undergoes further development, and, being discharged into water, is prepared to again enter the human body, where it forms the mature organism. Filariæ have been found very frequently in the blood of healthy individuals in China.

The cause of the non-parasitic form of chyluria which sometimes develops in temperate climates is not known.

The PROGNOSIS is generally favorable. So long as the parent worms remain alive in the human body complete recovery from chyluria is not to be expected. The embryos are probably not endowed with long vitality. It may be expected that sooner or later the parent worms will die, and then recovery takes place. As already mentioned, these worms have been discharged from abscesses. They seem to suffer in their vigor by febrile diseases; at least, it has been repeatedly noticed that the embryos disappear from the blood during attacks of fever.

The rational indication for TREATMENT is of course the destruction of the parasite in filarial chyluria, but no drugs are known which will accomplish this. Gallic acid, from one to two drachms given daily, is the remedy which thus far has proved most useful. Iodide of potassium in large doses has been beneficial in a few cases. Special virtue has been claimed for the decoction of mangrove-bark (*Rhizophora racemosa*) and the picro-nitrate of potash, but without satisfactory evidence. Rest, tonic treatment, and change of climate meet manifest indications in many cases.

Diabetes Insipidus.

The term "diabetes" signifies simply increased flow of urine, or diuresis. The term is applied to two affections which differ essentially each from the other. In one of these affections notable diuresis exists, a given quantity of the urine containing a small proportion of solid constituents; that is, the spefic gravity is low, being sometimes but little above that of spring-water. The morbid abundance of urine in this affection is due to an excessive elimination of water by the kidneys. Considered as a urinary affection, it is called diabetes insipidus. The other affection to which the term diabetes is applied is characterized by the presence of sugar in the urine. This affection will be considered in the next article.

A morbid excess of water in the urine, expressed by the terms hydruria and polyuria, is a symptom occurring in various pathological connections. It is a frequent symptom in cases of the small granular kidney, and is not infrequent in cases of the waxy kidney. Polyuria has been observed also as a symptom of hydronephrosis with wasting of the kidneys and in cases of enlarged prostate and some other surgical affections of the urinary organs. Polyuria occurs also, in some cases, as a symptom of cerebral diseases and of lesions caused by injuries of the skull. It is a symptom in cases of hysteria, asthma, and other nervous affections. In all these instances the polyuria is not properly to be considered as constituting an individual disease. Polyuria and hydruria, therefore, are terms not synonymous with diabetes insipidus. The latter term, if properly applied, denotes a disease characterized by an excess of water in the urine when not symptomatic of the foregoing diseases.

In well-marked cases of diabetes insipidus the quantity of urine greatly exceeds that of health. It may amount to fifteen and even twenty quarts per diem. The specific gravity varies between 1003 and 1007. There is no essential change in the composition of the urine except the excess of water. There is thirst, which leads the patient to ingest fluids in an amount proportionate to their loss by the kidneys. When deprived of other liquids patients have been known to drink their own urine. The need of drinking often and the frequent acts of micturition occasion much annoyance and interfere with sleep. The disease may not otherwise interfere with health. Patients of either sex, affected with the disease, have been able to perform active labor, have had children, and their lives have apparently not been shortened by it. It is not incompatible with good appetite, digestion, and nutrition. It has been tolerated for fifty years.

The PROGNOSIS is favorable as regards the duration of life and the preservation of general health if the disease be uncomplicated; but it is unfavorable as regards recovery from the disease. As remarked by Senator, "Recovery is rare, but death is still more rare."

The ETIOLOGY is obscure. The disease has followed injuries of the head, and an hereditary influence is involved in some cases.[1] In the majority of instances the disease cannot be traced to any adequate cause. It occurs not infrequently in infancy. In a proportion of about one-half, when the disease is developed, patients are less than twenty years of age, and in about the same proportion the age is between thirty and fifty. It is extremely rare after the age of fifty. The number of males affected with the disease is considerably larger than of females. There have been a few instances recorded in which diabetes mellitus has passed into diabetes insipidus, the sugar disappearing from the urine.

The PATHOLOGY is unsettled. The discovery by Bernard that a puncture in the floor of the fourth ventricle of the brain will produce polyuria without sugar in the urine naturally directed attention to this situation as the seat of the disease. Cases have been observed in which after death lesions were found to involve this part. There is not, however, sufficient ground for assuming that diabetes insipidus depends on anatomical changes here or elsewhere in the brain. The absence of all symptoms of cerebral disease, exclusive of the polyuria, in cases the duration of which extends over many years, militates against this assumption. That the disease is neuropathic is rendered probable by the occurrence of polyuria as a symptom in various nervous affections. The production of polyuria in animals by division of the splanchnic nerve favors this view. A reasonable hypothesis attributes the polyuria to the dilatation of the renal capillaries from influences exerted through the vaso-motor system. This result may be produced either by a paralytic effect through the splanchnic nerves, or by an active dilatation of the capillaries by an excitation of vaso-dilator nerves. (Vide p. 863.) The presence of inosite in the urine has been shown to be an effect, not a cause, of the polyuria. This principle appears in the urine if a large quantity of fluids be ingested in healthy persons.

The polyuria in cases of diabetes insipidus may be suspended during the continuance of an inflammatory or febrile disease. Excluding cases of polyuria as symptomatic of cerebral or of renal lesions, diabetes insipidus is an extremely rare disease. The basis of the DIAGNOSIS is a persistent polyuria which is independent of these lesions. The disease is to be distinguished from polydipsia. In the latter disease the excess of water in the urine is an effect of the excess of liquid ingested. If the quantity of urine exceed the quantity of drink, and if polyuria continue notwithstanding a restriction of the latter, the differential diagnosis is established. From diabetes mellitus the discrimination is easy. The low specific gravity of the urine is distinctive, but the exclusion of that disease is rendered positive by the negative result of testing the urine for the presence of sugar.

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[1] Weil has reported the history of a family of 91 members, extending through four generations. Of these, 23 had polyuria, and in 13 it could not be determined whether this existed or not. This affection existed throughout life without impairment of the general health (*Virchow's Archiv*, Bd. 95, p. 70).

It is injudicious to attempt to limit the excess of the water in the urine by diminishing the amount of ingested liquid. The disease is not thereby cured, and not only is there suffering from thirst, but, inasmuch as the polyuria continues, serious evils may arise from a morbid reduction of water in the blood. Theoretically, it is an object of TREATMENT to diminish the size of the renal capillaries. In a case reported by DaCosta recovery within a short period was apparently effected by the fluid extract of ergot in drachm doses, increased to two drachms and given three times daily. This remedy was recommended, on the basis of experience, many years ago by Prof. Gross. Ergot or ergotin has been found useful by others. Other remedies which are recommended are gallic acid, dilute nitric acid, and valerian.

The patient's strength is to be maintained as far as possible by tonic remedies, good alimentation, and favorable hygienic influences.

The disease is not in itself fatal, but it may occasion more or less exhaustion, and thus increase the liability to death from associated or intercurrent affections.

Diabetes Mellitus.

An increased flow of urine or diuresis is generally a notable feature of the affection now to be considered. The distinctive feature, however, is the presence of grape-sugar or glucose in the urine; and hence the significance of the term mellitus. *Glycosuria* and *melituria* are other names applied to the affection characterized by saccharine urine. The affection is far from being of frequent occurrence, yet it is not so rare but that cases come under the observation of every physician. From the number of cases which have come under my observation in recent years I have been led to think that the disease is less rare than formerly in this part of the world. One reason of its apparent infrequency is that it is liable to be overlooked in cases which end fatally with some intercurrent disease.

Sugar in the urine, like albuminuria, may occur as a symptom in various pathological connections. It follows anæsthesia produced by the inhalation of chloroform or ether, an epileptic or an hysteric paroxysm, some violent emotion, etc. The sugar under these circumstances is usually not abundant, and the urine contains it for a brief period only. As the distinctive feature of an individual affection the sugar which the urine contains is in greater or less abundance, and it continues more or less persistently. Even when the affection exists the saccharine urine is in fact merely a symptom. It does not constitute the affection. It is incidental to or an effect of the real disease. The sugar exists in the urine because it pre-exists in the blood and is excreted by the kidneys; but with our present knowledge the true seat and nature of the disease are not established, and therefore, provisionally, it is named as if it were a disease of the urinary system, and is included for the sake of convenience among the diseases affecting this system.

An abnormal accumulation of sugar in the blood constitutes the morbid condition called glycohæmia. Glycohæmia stands in an immediate causative relation to the presence of glucose in the urine, and glycosuria is an effect of the glycohæmia. The latter stands in a nearer relation than the former to the pathology of diabetes mellitus. The pathology of glycohæmia is the pathology of the diabetic disease. Glycohæmia has been considered in Part I among the morbid conditions of the blood. (Vide p. 69.)

ANATOMICAL APPEARANCES.—Although the pathological anatomy of diabetes mellitus is not known, certain anatomical changes have been found with sufficient frequency to deserve mention in this connection. That any of these changes are concerned in the production of diabetes mellitus has not been proven.

On page 71 the existence of changes in the central nervous system, and especially the observations of Dickinson on this point, have been considered. Frerichs also found dilatation of the vessels, capillary hemorrhages, and minute myelitic foci frequently present in the medulla oblongata after death from saccharine diabetes.

The liver may be large, small, or of normal size, hyperæmic, anæmic, or of normal vascularity, and the hepatic cells fatty, atrophic, or normal. In other words, there is no anatomical basis for the current theory which refers diabetes mellitus to abnormalities in the circulation or in the functions of the liver. The amount of glycogen in the liver of diabetics seems to be diminished during life.

Attention has been directed in recent years especially to alterations in the pancreas in cases of diabetes. In many cases atrophy of the pancreas has been observed. The atrophy of the gland-structure may be combined with increase of the interstitial connective and adipose tissue. Whether, as has been surmised, the cœliac plexus be involved in the pancreatic disease is not known. Glycosuria is occasionally present in cases of cancer and of calculous disease of the pancreas. It is claimed that diabetes mellitus associated with pancreatic disease runs a particularly rapid and severe course, and is accompanied often with fatty diarrhœa.

The kidneys of diabetic patients are often large and hyperæmic, probably in consequence of their increased functional activity. Glycogenic degeneration of the epithelial cells lining the looped tubules of Henle is a constant change in diabetes mellitus (Ehrlich). These tubes occupy the boundary-zone of the pyramids. The affected cells are swollen and hyaline in appearance. The presence of glycogen is proven by the reaction with iodine. This change is probably secondary to diabetes. Chronic diffuse nephritis is not a rare complication of diabetes.

A hyaline degeneration of the walls of small arteries in various parts of the body has been often seen in diabetic cases.

An excessive amount of fatty matter in the blood, constituting lipæmia, is common in diabetes.

The complications of diabetes will be mentioned in connection with the Clinical History.

CLINICAL HISTORY.—Notable increase of the urine, or polyuria, is usually the symptom which first awakens suspicion of the existence of the disease. Not only are the acts of micturition more frequent, but the quantity of urine passed in a given time is much greater than in health. The urine is deficient in color in proportion to the amount of sugar which it contains. The appearance is generally clear as well as pale. It has frequently a sweetish odor like that of whey. The presence of sugar is perceptible to the taste. Flies and bees are attracted to it if emitted on the ground. The density exceeds that of healthy urine in proportion to the amount of sugar. The specific gravity varies between 1025 and 1074, the latter being an extreme of increased density. A specific gravity below 1020 is exceptional. The reaction is in most cases feebly acid. The water of the urine is more or less increased; and hence the increased quantity of urine. The increase in the quantity of urine is generally very great, and sometimes enormous. It may amount to fifty pints or even more in the twenty-four hours. The frequent calls to micturate are a source of great discomfort, and especially during the night they constitute a serious evil by interfering with sleep. The action of the sugar on the urethra may occasion a sense of heat and stinging pains. Inflammation and excoriation of the prepuce and glans are sometimes produced, and may lead to phimosis. I was led to the discovery of diabetes by an examination of the urine in a case in which the patient complained only of an itching sensation in the middle of the penis, there being no increase of urine, no thirst, and no symptom of any disease except that just mentioned. The urine was found to be loaded with sugar. In women eczema of the vulva is a frequent effect, giving rise to distressing pruritus. It has been stated that more liquid is contained in the urine in some cases than is ingested. This may be true for a limited period, but examinations extending over several days show that, large as is the quantity of urine, it falls short of the amount of liquid contained in food and drink.

Cases differ in respect of the quantity of urine and the amount of sugar. Generally, at the beginning of the disease the quantity of urine and the amount of sugar are not large, but the increase of both is progressive as the disease advances. Exceptionally, the quantity of the urine is not increased, although abounding in sugar. This has been true of several cases which have come under my observation, the patients not being under treatment, and for this reason the disease is liable to be overlooked. The quantity of both urine and sugar also varies at different periods of the day, being generally largest within a certain period after meals. The proportion of sugar in the urine varies between ½ and 10 per cent., in extreme cases being 15 per cent. Usually from 2 to 4 per cent. is found. The quantity of sugar contained in the urine during twenty-four hours has been known to amount to fifty ounces. The quantity of sugar and of urine may be diminished by intercurrent febrile diseases and toward a fatal termination of the disease. Inosite has been found in the urine in some cases with grape-sugar, or after this had disappeared.

The daily excretion of urea in diabetes is generally markedly increased. Uric acid, although a frequent sediment in diabetic urine, is not excreted in abnormal amount. In many but not in all cases of diabetes there is a notable excess in the excretion of ammonia by the urine. This is associated with the presence of oxybutyric acid, a substance which by oxidation gives rise to diacetic acid. Acetone has been frequently found in diabetic urine, but whether it be preformed or originate from some other substance in the urine is uncertain. Many refer the formation of acetone to the presence of diacetic acid, which is probably the substance which imparts to the urine a burgundy-red color upon the addition of ferric chloride (Gerhardt's reaction. See p. 72). Albuminuria, although not rare, is to be regarded as a complication.

The urine when preserved for several days undergoes the alcoholic fermentation. It remains acid and is devoid of the ammoniacal odor which is emitted by decomposed healthy urine. It is rendered turbid by the development of the *Torula cerevisiæ*, or yeast-fungus. This alcoholic fermentation imparts to the urine irritating properties which cause inflammation and irritation of the skin with which it comes in contact.

Emaciation is usually marked after the disease has existed for some time. Cases differ as regards the rapidity of the emaciation. In general, it is in proportion to the abundance of sugar in the urine and the duration of the disease, but it is affected in a greater measure by the condition of the digestive organs. It is evident that certain alimentary principles which in health contribute to the growth and repair of the body are lost to nutrition in this disease. The development of other affections, especially pulmonary tuberculosis, increases the emaciation. The occurrence of diabetes is not infrequently preceded by *embonpoint* more or less excessive.

Increase of the appetite is usually a prominent symptom. The patient ingests much more food than in health. The abnormal appetite may amount to bulimia or polyphagia. This, conjoined with progressive decrease of weight, should excite suspicion of the disease. Exceptionally, the appetite is not increased, but, on the contrary, it may be more or less impaired. Not infrequently, in the course of the disease the appetite fluctuates, being sometimes greater and sometimes less than in health. It fails as the disease approaches a fatal termination.

Thirst is one of the first symptoms, occurring as soon as the quantity of urine is increased. It is urgent in proportion to the degree of diuresis. The quantity of liquid ingested is sometimes enormous. The necessity of drink during the night contributes, with the calls to urinate, to prevent sleep. Dryness of the mouth is a source of considerable inconvenience. The tongue is sometimes moist and coated, and sometimes dry and reddened. The gums are generally affected. They are softened, and either pale or reddened, bleeding readily on pressure. The teeth may become loosened and carious. The saliva is usually acid.

Digestion, until the disease is far advanced or until serious complications occur, generally appears to be well performed. Dyspeptic ailments, however, are not uncommon. If vomiting occur, the vomited matter is found to contain sugar. Sugar is also contained in the feces. Constipation is the rule, but in some cases diarrhœa alternates with constipation. The dejections show a deficiency of bile.

The respiratory function is not disordered, save by the pulmonary affections with which the disease is liable to become complicated, especially pulmonary tuberculosis. The respirations are somewhat fewer than normal if there be no pulmonary complication. It is stated that the amount of carbonic acid in the expired air is less than in health. The breath has a mawkish, sweetish odor which is quite characteristic. It has been compared to the odor of hay and of apples. I have recognized a patient whom I had examined at a clinic months before, and whose features I had entirely forgotten, as soon as he came sufficiently near for the odor of his breath to be perceptible. In repeated instances I have been led to suspect the disease by this peculiar odor. The condition of the gums and teeth renders the breath in some cases fetid.

The circulation often offers no symptoms of importance. The pulse, if at all affected, is oftener retarded than accelerated, exclusive, of course, of the effect of complications. I have observed capillary congestion of the surface in a notable degree. Symptoms denoting cardiac weakness are sometimes present, causing shortness of breath and tendency to syncope. Failure of heart-power may occur suddenly and terminate fatally. The temperature of the body is lowered, the thermometer in the axilla showing a temperature of 96° or 95° F. in some cases in which the disease is uncomplicated. The skin usually is dry and rough. It is rare for perspiration to occur except when pulmonary tuberculosis becomes developed. Furfuraceous desquamation of the epidermis is not uncommon. Itching of the skin is a symptom which sometimes occasions much annoyance. Diabetics are subject to carbuncles, boils, and various eruptions—namely, psoriasis, lichen, eczema, and impetigo. Spontaneous gangrene of the lower limbs is an occasional event. I have known of several instances in which this occurred. The gangrene resembles gangræna senilis. Ulcerations of the lower limbs without gangrene have been observed. Wounds and surgical operations often take an unfavorable course in diabetic patients. In protracted cases œdema of the legs frequently occurs. Bright's disease is an occasional complication of diabetes.

Muscular feebleness is more or less marked and progressive. Aside from the effect of complications, the patient is enfeebled in proportion to the excessive excretion of the urine, the amount of sugar which it contains, and the duration of the disease. Troubles of vision are not very infrequent. Amblyopia, or feebleness of vision, sometimes occurs early, and may eventuate as the disease advances in confirmed amaurosis, either complete or incomplete. Troubles of accommodation are not rare. In the majority of cases in which the vision is affected the defect is slight or moderate. Complete amaurosis is extremely rare. In the cases in which amaurosis, complete or incomplete, exists, the ophthalmoscope shows atrophy of the optic discs. Retinitis is a ground for suspecting that albuminuria coexists. The diabetic condition also favors the development of soft cataract. In some cases recovery from defect of vision occurring in diabetes takes place after the sugar has disappeared from the urine. Diminution, and at length extinction, of venereal desire belong to the clinical history of the disease. This effect frequently occurs early in the history of the disease, before the general powers of the system are much reduced. It may be the first symptom noticed by the patient. It is not, however, a constant symptom. In women the menstrual discharge gradually diminishes and is at length suppressed.

Certain symptoms referable to the nervous system are of great importance in the history of diabetes. The mental condition frequently undergoes a marked change; the patients become irritable, sad, and hypochondriacal; and the intellectual faculties are enfeebled. Neuralgia, affecting most frequently the sciatic nerve and often occurring on both sides of the body, is an obstinate symptom in some cases of diabetes. Occipital and trigeminal neuralgias have also been repeatedly observed. Absence of the knee-phenomenon is not an infrequent symptom in diabetes.

By far the most important of the nervous symptoms of diabetes are those which have been embraced under the name diabetic coma. The coma may follow physical over-exertion, mental excitement, or some slight bodily ailment, such as pharyngitis, gastritis, or bronchitis, or it may appear without any apparent exciting cause. The advent of coma may be sudden, but more frequently it is preceded by premonitory symptoms, of which the most important are headache, nausea, and vomiting, a sensation of anxiety and of oppression, restlessness, and noisy delirium, sometimes attended with fits of screaming. With or without this premonitory period of excitement and delirium the patient sinks into a state of somnolence deepening into coma. The coma is usually accompanied with a peculiar dyspnœa, without evidence of disease of the lungs or air-passages, the respirations being strong, deep, and often noisy. The number of respirations is either normal or more frequently moderately increased. The pulse usually is small and frequent. The temperature usually is subnormal. A peculiarity of diabetic coma is the marked ethereal odor of the breath, compared to the odor of fruit or of chloroform. The urine in the great majority of cases presents Gerhardt's reaction. The duration of the coma is generally between one and three days, but it may be shorter or longer. The termination is generally, but not necessarily, fatal. Coma may occur without the characteristic dyspnœa.

The most plausible explanation of diabetic coma refers this symptom to the presence of some toxic substance in the blood, but we are ignorant as to the nature of this substance. The fruity odor of the breath and the changes in the urine have led to the supposition that acetone or some kindred substance is the toxic agent; but it has not been possible to confirm this supposition. (See p. 72.) It is not probable that the coma is due to fatty embolism, as has been suggested.

Coma may occur in diabetes from uræmia, sudden failure of heart-power, and cerebral hemorrhage, but these cases are not identical with the so-called diabetic coma.

The COURSE of diabetes is generally slow. Its development is imperceptible, and when discovered it had probably, in most cases, existed for a considerable period. It is therefore difficult to fix the date of its origin; but, dating from the time when its existence is known, in most cases it continues for many months, and not infrequently for many years, before ending fatally. Its duration is indefinite and variable. Exceptionally, it runs rapidly to a fatal issue. Generally in these cases the patient is cut off by some intercurrent affection. The existence of diabetes notably impairs the tolerance of other diseases. Griesinger found the mean duration in 225 cases to be between two and three years. Cases have been described under the name diabetes acutus in which the disease has appeared only a few days or weeks before death.

CAUSATION.—In the vast majority of cases of diabetes the age is between thirty and fifty years. The disease occurs in infancy, youth, and advanced life, but instances are rare. Facts go to show that predisposition is an important factor in the causation, and that this predisposition may be inherited. Of 36 cases recorded, with reference to this point, by A. Flint, Jr., in 10 two or more cases occurred in members of the same family. In one instance a father, an uncle, two sisters, two brothers, and a son of one of the sisters were affected with the disease.[1] As regards pathogenetic influences pertaining to climate, season, diet, temperament, habits of life, etc., we have only conjectures without any precise information. Men are oftener affected than women. Of the 225 cases analyzed by Griesinger, in 172 the patients were men, and in 53 women.

In a considerable—not a large—number of instances the disease follows some injury of the head or spine or a concussion of the whole body. In some of these instances the glycosuria may have been only a transient symptom, and in other instances there may be room for doubt as to any causative connection, considering that in a large majority of cases no such causation is evident. A constant connection has not been established between the disease and cerebral lesions affecting the floor of the fourth ventricle, which the experiments of Bernard on animals showed to stand in a causative relation to glycosuria. Autopsical examinations in cases of diabetes mellitus have failed to discover lesions in this situation, and lesions have been found here when diabetes had not existed. No pathological connection of the disease with any anatomical changes in the liver has been ascertained. Syphilis and gout have been assigned a share in the causation, but with what propriety is uncertain.

The disease in some cases has been attributed to violent mental emotions.

DIAGNOSIS.—It is to be borne in mind that the fact of sugar existing in the urine is not alone sufficient evidence of the existence of the disease under consideration. Sugar, generally in small quantity and for a transient period, is not infrequently found in the urine in the course of various maladies. In aged persons the urine sometimes temporarily contains a considerable amount of sugar. A number of instances have fallen under my observation. I have known much needless uneasiness to be occasioned by announcing to a patient the discovery of a trace of sugar after an analysis of the urine.

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[1] *Journal of the American Medical Association*, April 17, 1886. The "son of one of the sisters" developed diabetes since the note appeared in the *Journal*.

If diabetes exist, saccharine urine is more or less persistent, the amount of sugar is unusually large, and generally the quantity of urine is notably increased. These points relating to the urine render the diagnosis sufficiently easy and positive. The diagnosis is confirmed by other symptoms belonging to the clinical history of the disease. If the quantity of urine be considerably increased, and on testing with the urinometer the specific gravity be found high, sugar is probably present. Sugar, however, is sometimes present in urine of low specific gravity. In cases of polyuria in which only the water of the urine is increased, as in diabetes insipidus, the specific gravity is extremely low, sometimes but little above that of spring-water. The fragrant odor of diabetic urine is characteristic, and it froths readily on agitation, the air-bubbles remaining for some time, but the practitioner should not be satisfied without demonstrative proof of the presence of sugar. Of the several methods of testing urine for sugar, one readily available is the fermentation test. A common test-tube or phial containing a little ordinary yeast is to be filled with urine, inverted and placed in a saucer containing the urine, care being taken to prevent the entrance of air. Kept at a temperature of 70° F., fermentation ensues, and the gas formed rises in the tube and displaces the urine. The gas is shown to be carbonic acid from its failure to support combustion. Christison states that a cubic inch of the carbonic acid formed in the fermentation test represents a grain of sugar. Of the various other methods of testing for sugar, the employment of Fehling's solution is the most convenient, and it is sufficiently reliable. I have found this test, as prepared by Squibb in two separate solutions, to remain unchanged for a year kept in glass-stoppered bottles, although used almost daily. Böttger's test with the subnitrate of bismuth is readily applied. The polarization test is probably the most accurate, but it is not necessary to resort to this for clinical purposes. If it be desired to ascertain the actual amount of sugar excreted in a given period, the volumetric method of analysis by Fehling's test or Roberts's differential density method may be employed.[1] Examinations of urine are to be repeated, in the first place, to settle the persistence of the saccharine condition, and in the second place with reference to improvement or otherwise.

PROGNOSIS.—As regards prognosis, a distinction is to be made between cases in which the sugar disappears from the urine when the diet is free from carbohydrates, and cases in which sugar persists during a strictly albuminous diet. The former class of cases is in general more amenable to treatment and offers a better prognosis than the latter. The milder cases may, however, become severe, and they may terminate fatally by some of the many complications to which diabetic patients are subject.

An early diagnosis diminishes the gravity of the prognosis. This fact enforces the importance of examining the urine for sugar in all cases in which there are any grounds for suspecting its presence; and it is to be recollected that sugar is sometimes persistently present when the quantity of urine is not much, nor even at all, increased, and when the specific gravity is not notably above the normal standard. Examination of the urine for sugar should not be omitted whenever failure of strength or emaciation occurs without apparent cause, and especially if the appetite be increased rather than diminished. Unusual thirst, dryness of the mouth or fauces, acidity of the saliva, impairment of vision, obstinate neuralgia, dimininution or extinction of venereal desire, dryness and itching of the skin, irritation or inflammation of the prepuce or glans penis, and pruritus vulvæ, should suggest the possibility of the existence of this disease. A certain amount of increase in the quantity of urine may not attract the attention of the patient, or the patient may not deem it a symptom of importance, and it is therefore necessary for the physician to direct inquiries to this point.

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[1] Vide Roberts, *Urinary and Renal Diseases*, Philadelphia, 1879.

The age and the weight of the body have a bearing on the prognosis. The prognosis is in general unfavorable in proportion to the youth of the patient, and as regards this disease corpulence is a distinct advantage.

The existence or development of complications is of great influence in the prognosis. The majority of fatal cases of diabetes terminate either in phthisis or in diabetic coma. According to some statistics, the most frequent cause of death is coma. The phthisis is a genuine pulmonary tuberculosis. Other not infrequent complications which may prove fatal are—pneumonia, nephritis, carbuncles, gangrene, and cerebral hemorrhage. The existence of diabetes lessens the ability to cope with affections which under other circumstances might not prove serious. Not a few cases terminate in debility and marasmus.[1]

In respect of prognosis much depends on the ability and willingness of the patient to efficiently carry out the dietetic treatment.

Undoubtedly, the majority of cases of diabetes mellitus terminate fatally sooner or later. My experience, which has been considerable, has led me, however, to modify somewhat the views respecting prognosis which were expressed in the early editions of this work. The disease seems to me less formidable than heretofore, provided proper treatment be adopted and persisted in. Moreover, I have met with a number of cases which illustrate the tolerance of the disease by those who have not been aware of its existence and by those who refuse to submit to rigid dietetic restrictions. In the great majority of cases the diabetic condition may be notably ameliorated, but there is always more or less danger of a relapse even in cases in which the sugar has been made to disappear and the patient has regained his usual health.

TREATMENT.—The essential treatment of diabetes mellitus is dietetic. The object is the exclusion, as far as possible, of sugar and starch from the diet. In proportion as this object is effected the glycohæmia and glycosuria diminish. Not infrequently all evidence of the disease disappears under strict dietetic treatment, and the patient is apparently in perfect health. Difficulties in the way of success under this treatment arise from the inability to carry it out rigidly. Patients sometimes have not sufficient strength of will to adopt and persevere in the dietetic restrictions. In some cases this difficulty does not exist, but the system will not tolerate the restrictions, and the vital functions give way under the continuance of the treatment. Complications or associated diseases sometimes render its adoption impracticable.

In not a few instances the physician is responsible for want of success with dietetic treatment, his instructions not being given with sufficient precision. It will not be sufficient to say, in general terms, that saccharine and starchy articles of diet are to be avoided. Most patients have not sufficient knowledge of alimentary principles to discriminate correctly the food which is allowable.

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[1] Of 250 fatal cases observed and analyzed by Frerichs, the following were the causes of death: Exhaustion, 18; phthisis, 34; pneumonia, 7; nephritis, 8; carbuncles, 6; cerebral hemorrhage, 10; cerebral softening, 2; cerebro-spinal meningitis, 3; other complications (cancer, etc.), 9; and in all the remaining cases (153) coma diabeticum (Frerichs, *Ueber den Diabetes*, Berlin, 1884). Most statistics have shown a larger proportion of phthisical cases; for instance, 43 per cent. in Griesinger's analysis.

I have known instances, for example, of diabetic patients eating freely of rye and corn-meal bread under the belief that only wheat bread was interdicted. It is essential that a written or printed list be furnished to patients of all the articles which may enter into the diet and of all those which are to be excluded. Such a list is essential in order to meet another requisite for success. The dietetic treatment must be made to satisfy not only the needs of nutrition, but the desires of the patient. This can be done only by varying the articles of diet from day by day, by making combinations to suit the appetite, and by the preparation of food in ways to be acceptable to the taste. Hence the diet-list should be as comprehensive as possible.

Bread occasions more difficulty than any other article of food. The force of the phrase "staff of life," as applied to bread, is appreciated when the attempt is made to exclude it persistently from the diet. Diabetic patients of good resolution will begin the dietetic treatment with the conviction that they can do very well without bread, but after a time the desire for it becomes too imperative to be resisted without great discomfort. The fact, then, is to be accepted that some form of bread must enter into the diet. Different substitutes for bread which have been devised do not satisfy the want of it. Flour consisting only of bran, prepared after Camplin's method, is generally tolerated but for a short time. Patients soon have a repugnance for it, and, as it is innutritious, there is then no advantage in persisting in its use. This is measurably true, also, of flour consisting of pure gluten, and of the almond-flour recommended by Pavy. Either a certain quantity of ordinary bread must be allowed, or a bread must be used containing a diminished proportion of starch without its entire exclusion. A bread made by Messrs. H. V. Hecker & Co. of New York contains a very small proportion of starch. This bread is light and agreeable, but it requires great care in its preparation. Analyses made by Prof. C. A. Doremus gave between 1 and 2½ per cent. of starch, whereas in ordinary bread the proportion is about 55 per cent. In a number of cases in which I have advised its use patients have found it to be a satisfactory form of bread.

The bill of fare for diabetics is very extensive. All varieties of shellfish are allowable. Soups of various kinds may be prepared without flour, rice, vermicelli, or any other article which contains starch. Fish of every description may be eaten, either fresh, salted, or preserved in oil. Of meats, poultry, and game, there is but a single exception—namely, the livers may contain enough sugar to warrant their exclusion. Salads may be made of lettuce, celery, cucumber, romaine, watercress, Brussels sprouts, chicory, dandelion, young onions, coldslaw, and olives. Of vegetables, other than those just named, the list embraces spinach, cabbage, cauliflower, string beans, pickles, beet-tops, sorrel, radishes, mushrooms, asparagus, truffles, artichokes, oyster-plant or salsify, and tomatoes. Eggs, caviar, cheese of all kinds, butter, cream in moderation, unsweetened jellies, filberts, pecan-nuts, almonds, butternuts and walnuts, Brazil-nuts, and cocoanuts are included. Of drinks, coffee, tea, and cocoa without sugar, whiskey, brandy, gin, claret, Burgundy, and dry sherry are permissible. Glycerin may be used in lieu of sugar in coffee and tea. A patient under my care ate with relish custards made with glycerin instead of sugar. It is stated that mannite, inosite, levulose, and inulin are forms of sugar which may be taken without rendering the urine saccharine. This statement needs further confirmation based on experimental observation. The interdicted kinds of food are—sugar, syrups of all kinds, and honey, wheat and rye flour and corn-meal, arrowroot, sago, tapioca, oatmeal, barley, potatoes, beets, parsnips, carrots, rhubarb, peas and beans, chestnuts, sweet fruits of any kind, chocolate, cider, most malt liquors, champagne and other sparkling wines, all the sweet wines, sweetened spirits, and liqueurs. The admissible articles of diet have been conveniently arranged by Austin Flint, Jr., in the form of a bill of fare.[1]

The very bitter ales may be taken moderately. Turnips are considered by some as allowable. Celery is interdicted by some, but probably without reason. There have been doubts as to tomatoes, but they are probably correctly placed in the list of allowable articles. The quantity of ingested fluids should be moderately restricted.

For the sake of emphasis it may be repeated that success will depend on the pains taken to vary, combine, and prepare the allowable kinds of food, so as to render the diet satisfactory as regards the patient's taste, appetite, and digestion. To do this will require much and constant attention on the part of those who have charge of the dietary of diabetic patients. It is difficult to carry out the dietetic treatment properly in hospitals. Patients can rarely have it properly carried out in hotels and boarding-houses. Poverty is sometimes a serious obstacle in the treatment of this disease.

Under the dietetic treatment, faithfully carried out, the sugar in the urine is speedily diminished. In a considerable proportion of cases it disappears; the quantity of urine decreases and often becomes normal in this regard; the thirst disappears, and there may be no evidence of ill health. Patients have repeatedly declared, even when the urine still contained some sugar, that were it not for the knowledge of this fact they would not suspect the existence of any malady. Not infrequently there is a consciousness of better health than for a long time before the diabetic disease was discovered.

The return to the articles which are not in the list of those allowable is to be made after a time gradually and tentatively, watching the effect upon the urine. Probably in most instances an unrestricted diet will cause a return of the saccharine urine if the cases of transient diabetes, to which reference has been made, be excepted. Under these circumstances patients cannot be considered as cured; but, on the other hand, the restricted diet, with proper pains, may be well tolerated indefinitely, the general health remaining excellent. Although, therefore, the disease be not cured (quoting the language of Senator), it may be made entirely harmless.

Reference may be here made to the skimmed-milk treatment proposed by Donkin. His method consists in confining the patient strictly to skimmed milk, from six to eight pints being taken daily, the treatment to be continued for ten or twelve weeks, after this period other articles of diet being gradually added and at length substituted. That this plan of treatment has proved temporarily useful in some instances must be admitted; in most instances, however, it has been not only nugatory, but hurtful and even destructive.

As regards the employment of drugs, if there be no special symptomatic indications to be met by remedies, before any medication is employed it should be ascertained how much may be effected by diet alone.

The alkalies, first employed for theoretical reasons, have been found to be useful in some cases. Trousseau's method was to give a drachm daily of the bicarbonate of soda, and to increase the doses gradually to three or even four drachms. In order to avoid the evils incident to super-alkalinity of the blood he advised that the remedy be given only during ten consecutive days in each month. I have seen the good effect, apparently, of this treatment when there was not much restriction in diet. The benefit which patients derive from treatment at Carlsbad and Vichy cannot depend greatly on the alkaline springs, for the same benefit is not derived from the water when taken at home. The credit belongs, in a great measure, to accessory circumstances in these watering-places and to the treatment in other respects. A cure is not to be expected from the use of alkalies, and it is seldom advisable when patients can carry out and tolerate strict dietetic treatment.

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[1] A. Flint, Jr., "On the Treatment of Diabetes Mellitus," *Journal of the Am. Med. Association*, July 12, 1884.

Opium causes diminution of the sugar in the urine. Pavy, who advocates the opium treatment, advises, in preference to other preparations, codeia, giving it in doses gradually increased from a quarter of a grain to two grains three times daily. The treatment relieves, but it does not effect a cure, and it is open to objection on the score of a liability to the formation of the opium habit. Opium, however, is indicated in some cases for the relief of muscular pains, from which diabetics not infrequently suffer, and which prevent sleep.

Other remedies among the great number which have been recommended are—the carbonate of ammonia, lactic acid, cod-liver oil, the iodide and the bromide of potassium, iodoform, ergot, arsenic, arsenite of bromine, carbolic acid, salicylic acid, salicin, strychnia, jambol, and sulphide of calcium. Each of these in certain cases is more or less useful, but no one of them is entitled to be considered as a curative remedy, The late Dr. Frick of Baltimore studied the relative effects of different remedies in a case under his observation for nine months, and the diminution of the sugar in the urine was most marked under the use of strychnia. Dickinson remarks that "strychnia is, of all remedies, the most constantly useful." Some cases are notably benefited by salicylic acid or salicylate of sodium.

There may be indications for remedies without reference to any curative effect. Tonics are indicated if the appetite or the digestion be impaired. Anæmia, as in other pathological connections, calls for iron. The nervous system may be tranquillized and sleep induced by the bromides or other hypnotic remedies. Various symptoms may require palliative treatment.

An important part of the treatment of diabetes pertains to hygienic observances. The body should be protected against atmospherical changes and the functions of the skin should be well maintained. The warm bath or sponging the body, if the immediate effect be agreeable, is to be recommended. The hydropathic pack has been found serviceable. A change from a climate which is cold and variable to one uniform and warm is often highly advantageous. A long sea-voyage in a warm latitude has proved signally beneficial. Exercise in the open air is to be advised, not exceeding so far the limits of comfort as to induce over-fatigue or exhaustion. Gymnastic exercises may sometimes be resorted to with profit. Last, but not least in importance, mental relaxation and recreation are to be enumerated in the list of regiminal measures.

An efficient treatment in cases of diabetic coma has not been found. Camphor, ether, and other stimulating restoratives may be tried.

Involuntary Seminal Emissions—Spermatorrhœa—Impotence.

Involuntary seminal emissions, spermatorrhœa, and impotence frequently depend on abnormal conditions which belong to surgery, but not infrequently they fall properly within the sphere of the practice of medicine. By involuntary seminal emissions is meant the ejaculation of semen and the venereal orgasm without any voluntary effort, either natural or unnatural. Involuntary emissions occurring during sleep are common, taking place in connection with erotic dreams. Occurring after intervals of several days in persons of continent habits, they are physiological rather than pathological. They denote simply a certain amount of functional activity of the generative organs. They do not imply a morbid condition nor do they lead to any morbid effects. They occur especially when habitual sexual intercourse is interrupted from any cause. Under these circumstances they are manifestations of health rather than of disease. The physician is warranted in giving such assurances to patients, who are often rendered needlessly apprehensive and unhappy by popular works or lectures the object of which is to excite fears for a mercenary purpose.

Occurring more frequently, the emissions denote a morbid erethism and weakness of the organs of generation. They occur sometimes nightly, and sometimes even repeatedly during the same night. They then call for remedial measures, although even under these circumstances their morbid effects have been much exaggerated. Occurring in persons debilitated from any cause, they may take place without erection and with little or no venereal excitement, the patient sometimes becoming aware of their occurrence only by the seminal stains. They occur sometimes during the day when the venereal desire is excited. These are called diurnal in distinction from nocturnal emissions. A patient stated to me that they had occurred in his case from excitement caused by shampooing the head in a barber-shop. Persons who have been addicted to venereal excesses or to unnatural abuse of the sexual function are especially subject to this morbid frequency of involuntary emissions. The mind in such cases, particularly if the affection be attributable to unnatural abuse, may become greatly depressed; the patient is apprehensive of impotence or fancies that he is already impotent; his attention is absorbed with the affection; he suffers from a sense of pollution and degradation; he believes that his constitution is ruined and that there is danger of insanity. Patients in the condition just sketched form an unfortunate class with which every practitioner is more or less familiar. They go from one physician to another, and upon patients of this class quacks prey largely. The affection sometimes exists in a still greater degree. The emissions are diurnal as well as nocturnal. They are produced by anything which provokes the sexual passion, and may take place with little or no excitement of the external organs.

Spermatorrhœa means, strictly, the discharge of semen without the occurrence of the orgasm. It is an affection superadded to morbid seminal emissions, the seminal secretion passing into the urethra, and sometimes into the bladder, without the consciousness of the patient. In cases of spermatorrhœa the semen is expelled in the acts of micturition and defecation. Cases of true spermatorrhœa are far less frequent than has been supposed. Persons have been regarded as having this affection whenever a mucilaginous fluid bearing some resemblance to semen is discharged from the urethra. The microscope affords the only trustworthy mode of determining that the liquid is seminal. Were this mode of examination generally employed, cases of spermatorrhœa would be found to be extremely rare. In examining the sediment of the morning urine it is to be borne in mind that spermatozoa will be likely to be found if sexual intercourse or a seminal emission have taken place during the night. The presence of the spermatozoa under these circumstances is not evidence of spermatorrhœa. Veritable spermatorrhœa doubtless occurs, but probably only in persons who are affected with a morbid frequency of seminal emissions. Muco-purulent matter escaping from the urethra or observed in the urine in cases of cystitis may give rise in the mind of the patient to the apprehension of spermatorrhœa. A transparent viscid liquid escaping in small quantity from the urethra, especially after prolonged erection, not infrequently occasions much uneasiness in the minds of persons whose attention is directed to the sexual organs with reference to spermatorrhœa, and of those who have gonorrhœa. This is treated as a gleety discharge by empirics. The discharge is a secretion from the prostate gland and is of no practical importance. It is sometimes of importance in a medico-legal view to determine whether stains on linen be caused by dried semen. The physical characters of suspected stains are not to be relied upon, nor is chemical analysis reliable. If, however, water be slowly added to the stains and portions be examined by the microscope, spermatozoa will be recognizable when the stains are seminal.

Involuntary seminal emissions and spermatorrhœa are frequently associated with symptoms denoting various kinds of functional disorder, especially disturbance of the nervous system. These symptoms have been considered as direct morbid effects of seminal losses. More or less general debility is undoubtedly a result of an abnormal discharge of semen, but the latter is often incidental to disorder of the system otherwise produced; and the disturbance of the nervous system so frequently associated with seminal losses is measurably due to the mental depression and apprehensions which they frequently occasion.

In the TREATMENT of seminal emissions the distinction which has been made between their physiological and pathological character is to be kept in view. Occurring infrequently in a person of full health, in addition to assurances of their innocuousness it will generally suffice to advise moderation in the use of wine or spirits and stimulating articles of food, saline laxatives occasionally, cold ablution of the genital organs at night, avoidance of undue warmth from the bed or bedclothes and of distension of the bladder, and that the mind be diverted as much as possible from provocatives of sexual desire. If these measures do not suffice, certain remedies which appear to exert an anaphrodisiac influence may be prescribed. Remedies which may be given for this purpose are camphor, lupulin or the tincture of hops, conium, belladonna, and the bromide of potassium. The remedy last named has seemed to me to be distinctly efficacious. Marriage is always remedial in these cases.

Cases in which the emissions depend upon a morbid erethism and weakness of the generative organs, and cases of spermatorrhœa, are of more importance and the treatment is more difficult. The anaphrodisiac remedies are indicated, but in these cases much benefit is derived from tonic remedies and an invigorating regimen. Quinia, small doses of strychnia or nux vomica, and the preparations of iron are useful. Sea-bathing or the sponge-bath and out-of-door life or gymnastic exercises are useful regiminal measures. The diet should be nutritious, but not stimulating, and alcoholic stimulants are to be taken very moderately or interdicted. Everything calculated to provoke sexual desire should, as far as practicable, be avoided. Healthful mental occupation and chaste associations do very much toward effecting a cure. It is obvious that to carry out the moral part of the treatment requires on the part of the patient a determination and perseverance which are often wanting. Taking into view all the circumstances in individual cases, it is sometimes judicious to encourage or recommend marriage.

Attention is to be directed to the condition of the urethra in cases of seminal emissions and spermatorrhœa. They often follow gonorrhœa, and are sometimes dependent more or less on urethral stricture. According to Lallemand, seminal losses often depend on an abnormal sensibility at or near the prostatic portion of the urethra, and the treatment in these cases requires the use of the bougie and cauterizing applications. The opinion of Lallemand is held by distinguished surgeons at the present time. For information on this point the reader is referred to surgical works.

Impotence means inability to perform the act of coition. The term does not mean want of the power of procreation, and the latter may be wanting when there is no incapacity for sexual intercourse. Exclusive of malformations and other abnormal conditions of a surgical nature, impotence, in the first place, may involve absence of sexual desire. Different persons in health differ widely as regards the venereal propensity; in some persons the sexual desire is intense and importunate, in others it is feeble, and sometimes it is altogether wanting. The propensity may, on the one hand, be increased by indulgence and by fostering in various ways ideas connected with it; and on the other hand it may be diminished and extinguished by an opposite course. In the marital relation frigidity, as regards this propensity, may depend on personal antipathy, and hence is a not infrequent source of domestic unhappiness. Absence of sexual desire may arise from general debility, and is incident to various diseases. If the disease with which it is connected be not serious nor permanent, a return of sexual desire may be expected on recovery of the accustomed health. Intense mental preoccupation, the depressing emotions, and exaltation of the higher sentiments tend to repress sexual desire.

As the loss of virility is generally dreaded, physicians are often consulted when a notable change as regards the venereal propensity is experienced. The circumstances which have led to the change in individual cases are to be investigated, and the conclusion with respect to the probability of impotence, together with the treatment, is to be based on these circumstances. In a large proportion of cases the deficiency or absence of sexual desire is due to temporary causes. Premature decline or extinction may result, on the one hand, from early and excessive indulgence or abuse of the generative function, and on the other hand from prolonged continence. Under certain circumstances aphrodisiac remedies are desired. The remedies so called are of doubtful efficacy. Phosphorus, cantharides, and nux vomica have been considered as special stimulants acting on the organs of generation. They should be given circumspectly. It is sometimes useful to secure the moral influence of the administration of remedies supposed to have this effect.

Impotence may exist without loss of sexual desire. Temporary incapacity proceeds from timidity or deficient self-confidence. The physician is not infrequently consulted by persons about to enter upon marriage who are apprehensive of being incompetent to accomplish coition. Assurances of capability, if it be evident that the apprehensions are imaginary, may be all that these cases require. Persons who have practised masturbation in boyhood, and they who are subject to involuntary emissions during sleep, are not infrequently led to believe that they are impotent by pernicious publications or lectures designed to alarm sensitive minds; and apprehension on the score of ability to accomplish the act of coition creates incapacity. A failure to accomplish the act is likely to be followed by the conviction that impotence exists, and, practically, it does exist so long as this conviction continues. In such cases all that is requisite is that the act of coition should be undertaken without any anxiety as to success. It is useful in such cases to interdict sexual intercourse for a certain time, in order to prevent tentative efforts, which, if unsuccessful, fasten the conviction of impotence more and more upon the mind. If a certain period of abstinence be enforced, it will be likely to happen that the patient is impelled by his desires to demonstrate his capacity before the period expires. It is sometimes the case, however, that coition is permanently prevented by fancied incapacity. I have known an instance in which this was the probable explanation of the absence of sexual intercourse during the whole of a married life lasting over fifteen years, and in another instance five years had elapsed without any accomplishment of the act of coition. In both instances seminal emissions occurred not infrequently. A remedy prescribed with an assurance of a cure doubtless sometimes proves curative by a moral effect. In certain cases impotence, with sexual desire, is a result of the erethism and weakness of the generative organs associated with seminal emissions and spermatorrhœa. The ejaculation and orgasm take place at the beginning of, or prior to, the act of coition. This kind of impotence is sometimes, temporarily, a result of prolonged continence. The measures of treatment in such cases are those indicated by seminal emissions and spermatorrhœa. Persistence in the employment of these measures, together with a proper degree of reserve as regards the frequency of the attempts to consummate sexual intercourse, will generally prove successful.

Excessive indulgence in venery, when it does not lead to involuntary seminal emissions, spermatorrhœa, or impotence, is a not infrequent cause of ill health. Dyspeptic ailments, attacks of vertigo, functional disorder of the heart, melancholia, and hypochondriasis are among the morbid effects of this cause. It is the duty of the physician to inquire into the habits of patients in this regard. The evils of excessive indulgence, aside from moral consequences, are greater when it is illicit than when it is marital; and moreover, the former is far more likely to be excessive. The limits of a healthful indulgence doubtless vary according to constitutional vigor, but it may be considered as excessive if the acts of coition are repeated several times a week.

The unnatural abuse of the sexual function, or masturbation, as a cause of involuntary seminal emissions, spermatorrhœa, and sometimes of impotence, has been already referred to. The effects on mind as well as body are pernicious. It is plainly the duty of the physician not only to endeavor to discover and arrest this practice in individual cases, but to enjoin upon parents, teachers, and associates the importance of watchfulness in this respect over those for whose welfare they are to a greater or less extent responsible. There is also another duty of the physician connected with this important topic. Many sensitive persons who have been addicted to self-abuse in early life suffer greatly from the belief that their mental and physical powers have been irreparably injured. The evils, great as they sometimes are, are much exaggerated by popular works and lectures designed to excite fears for a mercenary end. The physician may often do much good by removing undue apprehensions connected with the errors of youth.

SECTION SEVENTH.

FEVERS AND OTHER GENERAL DISEASES.

CHAPTER I.

CLASSIFICATION AND PATHOLOGY OF FEVER.—FEBRICULA. —CONTINUED FEVERS.

Classification of Fevers.—The General Pathology of Fever.—Febricula.—Typhoid Fever: Anatomical Characters and Clinical History.

Classification of Fevers.

THIS concluding section will be devoted to fevers and other general diseases.

Fevers are distinguished as *essential* and *symptomatic*. A symptomatic fever is one which is secondary to some local affection, such as an acute inflammation. The term essential distinguishes a fever which is not secondary or symptomatic, but is primary or idiopathic. Local affections may exist in essential fevers, but they cannot be considered the cause of the fever. The essential fevers are febrile infectious diseases. The essential fevers are those which are to be considered in this section.

According to the usual symptomatic classification of fevers we distinguish—

1. *Febricula*, a form of fever characterized by its short duration and mildness.
2. *Continued fevers*, which are distinguished by the unbroken continuity of the febrile phenomena, such as typhoid and typhus fevers. Relapsing fevers and erysipelatous fever may be included in this class.
3. *Periodical fevers*, which are distinguished by the occurrence of distinct paroxysms or notable exacerbations as regards the febrile phenomena, such as intermittent fever, remittent fever, typho-malarial fever, yellow fever, and dengue.
4. *Eruptive* or *exanthematous* fevers, in which an eruption on the surface of the body is a prominent feature, as in smallpox, scarlet fever, measles, roseola, and typhus.

Fevers are also often classified on an etiological basis into miasmatic, contagious, and miasmatic-contagious fevers, according to distinctions which have already been defined on page 84. The malarial fevers are the type of the *miasmatic* forms. Probably pneumonia, and perhaps acute articular rheumatism, are also to be included in this category. *Contagious* fevers are smallpox, chicken-pox, scarlet fever, measles, rötheln, typhus fever, and relapsing fever. As *miasmatic-contagious* fevers are reckoned typhoid fever, yellow fever, and dengue. Epidemic dysentery and Asiatic cholera are also to be regarded as miasmatic contagious diseases.

The foregoing list of fevers does not comprehend all that exist. It comprehends, however, those which from their prevalence and established identity it is desirable to consider in this work. Some of the diseases which for convenience have been considered in preceding sections of this work, such as epidemic dysentery, Asiatic cholera, cerebro-spinal meningitis, trichinosis, and pneumonia, might with propriety have been included in the present section.

There are certain terms in common use which in their application to fevers require a few words of explanation. Fevers characterized by great prostration have been called *adynamic*, *asthenic*, or *low*. Fevers have been said to be *ataxic* when symptoms referable to the nervous system, such as delirium, etc., are prominent. Fevers of unusually long duration have been called *slow;* and when the febrile phenomena—especially the temperature—are intense, the fever is said to be *high*. The *initial stage* of a fever extends from the beginning of the disease until the full development of the fever. The period of full development, or acme, of the fever is sometimes called the *fastigium*. The fastigium is followed by the *stage of defervescence*, or decline of the fever. When the decline is rapid, occupying only a few hours or a day or two, the fever is said to terminate by *crisis*. When the decline is protracted the termination is by *lysis*. These different stages of fevers, to which may be added the *period of convalescence*, are of very unequal duration in the different forms of fevers.

General Pathology of Fever.

It is deemed desirable to introduce this subject in this connection, although properly it belongs to the department of General Pathology considered in Part I. of this work. The general causes and the theory of fever are among the most difficult and least understood subjects in general pathology.[1]

It has been proven experimentally that various substances when injected into the blood or the tissues of an animal are capable of exciting fever. It has not been found possible to determine any general characters which distinguish these pyrogenic substances. It is of especial interest that various ferments, particularly fibrin ferment, and many of the so-called ptomaïnes or cadaveric alkaloids, possess pyrogenic properties. This has given rise to the supposition that the fever which is secondary to local inflammations, to necrotic foci, and to injuries is often due to the absorption of fermentative substances which are produced by the local disease. Micro-organisms are among the most important of fever-exciting agents, and are probably involved in the causation of all the essential fevers. It has been proven by experiment that injuries of various parts of the central nervous system, especially puncture of certain circumscribed regions of the brain, are followed within a few hours by marked elevation of temperature. There is reason, therefore, to believe that fever may be in some cases a purely nervous affection produced without the introduction of any pyrogenic substance into the blood.

In what way is the abnormal elevation of temperature produced in fever? It seems to be established, on the one hand, that an excessive amount of heat is produced in fever within the body, and, on the other hand, that in proportion to this excessive production less heat is given off from the body than in health. It is well known that animal heat is the result of chemical processes, especially processes of oxidation, which are constantly going on within the body. Now, it is found that in fever, in proportion to the amount of nourishment taken, there is an abnormally large quantity of urea excreted by the urine, and particularly that more oxygen is absorbed and more carbonic acid given off by the respiration than in health. These changes have been observed to be present before there is any elevation of temperature, so that they cannot be considered, at least not entirely, as secondary to the fever. They also indicate an increase in the oxidizing processes within the body, and therefore an increase in the amount of heat produced.

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[1] An important monograph on this subject is that of H. C. Wood—*Fever: A Study in Morbid and Normal Physiology*, Washington, Smithsonian Institution, 1880.

It has furthermore been rendered probable that the amount of heat given off from the body in fever is diminished relatively to the amount produced. When a fever is ushered in by a chill or by chilly sensations, the arterioles of the skin are contracted, the surface is cold, and there is an absolute diminution in the radiation of heat from the body. There is at this time the most rapid development of pyrexia. When the surface of the body is abnormally hot, as it is during the greater part of most fevers, it is evident that more heat must be given off by radiation than at the normal temperature of the skin; but there seems to be no doubt that, in proportion to the amount of heat produced in the body, there is less heat given off in fever than would be the case during a corresponding increased production of heat in health, as by severe muscular exercise. In other words, the equilibrium which normally exists between the amount of heat produced and that given off is disturbed in fever. Perspiration, which is an important factor in the regulation of the bodily temperature, is usually, although not always, diminished during the height of a fever.

We have no positive knowledge as to how fever-producing agents act in causing pyrexia. Since experiments have shown that injury of certain parts of the brain is followed by rapid elevation of temperature, which cannot be identified with ordinary traumatic fever, the theory has gained wide acceptance that the pyrogenic agents act primarily upon certain nervous centres. Whether these centres be special heat-centres, as has been surmised, or vasomotor or other centres, is uncertain.

Our knowledge of the effects of the elevation of temperature upon the various organs and functions of the body is imperfect. Fever is accompanied with increased frequency of pulse and of respiration, and it has been shown experimentally that these effects follow elevation of the temperature of the blood. It has not, however, been proven, nor is it indeed probable, that all of the circulatory and respiratory disturbances of fever are directly referable to the elevation of temperature. The injurious effects of the special agents which cause the fever must also be taken into consideration. There has doubtless been a tendency to exaggerate the immediate effects of the pyrexia as such, and to disregard the action of the special agents of infection. Parenchymatous degeneration of muscular tissue, particularly of the heart, and degeneration of glandular cells, are common, but by no means constant, in fevers, even in severe forms, and are referable probably more to the infection than to the elevation of temperature. Nor is it justifiable to refer disturbances of the nervous, digestive, and renal functions in fever exclusively to the elevation of temperature. We possess no satisfactory explanation of the diminution of the chlorides in the urine in fever. The blood-pressure is generally diminished in fever. It has been shown that many fevers are accompanied by a destruction of red blood-corpuscles. The increased quantity of coloring matter in the urine also points to this. That fever of long duration should cause a loss of weight of the body is not strange when one considers the increased consumption of tissue and the small amount of nutriment ingested.

Facts warrant the conclusion that each of the essential fevers has its own special cause; in other words, that each requires a particular causative agent capable of producing that fever and none other. It is most consistent with our present knowledge, if it be not logically certain, that this causative agent is a living organism. The unlimited reproduction of the special cause within the body in the infectious fevers is a fact of which the causation by living organisms alone affords a rational explanation. In relapsing fever and in typhoid fever it is probable that the causative organisms have been discovered.

Febricula—Ephemeral Fever—Simple Fever.

The term *febricula*, a diminutive of *febris*, is perhaps the best name to denote a mild, short fever which is everywhere of not infrequent occurrence. If very short, lasting only a single day, it is appropriately called an *ephemeral fever*. When not ephemeral the fever may continue for a period varying between three and eight, even ten, days. It has been known by a variety of names. It is frequently called *simple continued* or *simple fever*.

This fever has no known anatomical characters. The opportunity of post-mortem examination is seldom offered, for the disease rarely if ever of itself ends fatally, at least in cold or temperate climates. No lesions belonging to the fever have been found in cases in which, owing to complications, death has taken place.

The invasion in cases of febricula is generally abrupt, but in a certain proportion of cases lassitude, loss of appetite, and general malaise exist for two or three days before the fever is developed. Irregular chilly sensations may accompany the attack, but there is seldom a well-pronounced chill. The febrile movement is more or less intense; and not infrequently the acceleration of the pulse and heat of the surface are greater than at the beginning of typhus or typhoid fever. Pain in the head is a prominent symptom, the pain being generally frontal. There is more or less pain in the loins and limbs. Anorexia is usually complete. Constipation is the rule, and the abdominal symptoms which belong to typhoid fever are wanting. The urine is scanty and high-colored; the intelligence remains unaffected; and the hebetude which characterizes typhus and typhoid fever is not observed. There is no characteristic eruption; herpetic vesicles about the mouth sometimes occur; and bluish spots (*tâches bleuâtres*), from three to eight lines in diameter, not elevated and not disappearing on pressure, have been observed. These occur occasionally in typhoid fever.

The febrile career ends suddenly after one, two, three, or more days, the limit of the duration being ten days. Frequently with the cessation of fever there is a copious perspiration. Epistaxis and, occasionally, hemorrhage from the uterus and rectum have been observed at the end of the fever. The convalescence is usually rapid, and there are no sequels. The thermometer indicates a sudden rise of temperature of four or five degrees, and as rapid a decline, the heat passing in one or two days to the normal standard.

This kind of fever is supposed to occur irrespective of any special cause; that is, ordinary causes are supposed to produce it. It is observed to follow over-exertion, dietetic excesses, and exposure to the sun's rays. This view of the causation is, however, conjectural. It is probable that certain cases which from their short duration are considered as cases of febricula are in fact cases of typhoid or typhus fever, the disease ending without going through its usual career; in other words, cases of ephemeral fever may be cases of abortive typhoid or typhus fever.

The DIAGNOSIS of febricula is of importance with reference to the success of measures employed to arrest or abridge the duration of typhoid and typhus fever. In cases in which measures employed for these ends appear to prove successful, the question is, Was the disease typhoid or typhus fever, or was it a febricula? The following are considered as diagnostic points: 1, the abrupt development of the fever; 2, the attack succeeding obvious and ordinary causes. To these is to be added absence of diagnostic traits belonging to the clinical history of either typhoid or typhus fever. Cases, however, occur in which it is difficult or impossible to decide during the progress of the fever whether it be typhoid or typhus or a febricula, and after the fever ends whether it have been a febricula or an abortive or arrested typhoid or typhus fever.

Febricula in cold and temperate climates is rarely, if ever, fatal, except from an accidental complication of a serious nature. It is said to prove fatal not infrequently in tropical countries.

As regards the TREATMENT of febricula in cold and temperate climates, hygienic and palliative measures only are called for. A saline laxative, refrigerants, and anodynes are the remedies indicated.

Typhoid Fever.

The name *typhoid* signifies *typhus-like*. The pertinency of this name, as applied to the fever to be now considered, consists in the resemblance of the disease to typhus fever. It might, therefore, seem most appropriate to consider first typhus fever; but typhoid fever prevails very extensively in this country, and has been known with us as *common continued fever*, whereas typhus is comparatively rare, and the latter is perhaps never an indigenous disease. For this reason, reversing the order which the name suggests, I shall consider first typhoid fever with reference to its anatomical characters, clinical history, causation, diagnosis, and prognosis; afterward, typhus under the several aspects just named; and last, the treatment of typhoid and typhus fever.

The name *typhoid* is open to objection. It is customary to apply this name to a condition or state incident to many diseases, and hence has arisen confusion. Of other names which have been proposed, no one has been generally adopted, and probably the disease will continue to be called typhoid fever. This name was introduced by Louis, whose clinical researches were of great value, not merely with reference to our knowledge of this disease, but as inaugurating the true method of the study of the clinical history of all diseases—namely, by means of the analysis of carefully recorded cases. The correspondence of the results of the researches of Louis as regards typhoid fever with the results of similar researches by others in different countries furnishes the best possible proof of the value of the method of study, and at the same time shows the remarkable uniformity of the disease at different times and places. By German writers the disease is called *abdominal typhus* or *ileo-typhus*. Prof. Geo. B. Wood suggested as an appropriate name *enteric fever*. Both these names relate to the intestinal lesions which are characteristic of the disease, and they are objectionable because they convey the impression that the fever is the result of these lesions. Murchison proposed as the name *pythogenic fever*—a name which implies that the disease is produced by putrescent matter. It is an objection to this name that the source of the special cause, implied in the name, has not been established.

ANATOMICAL CHARACTERS.—The characteristic lesions of typhoid fever are seated in the lymphatic structures of the intestine—namely, Peyer's patches and the solitary follicles. Corresponding to the intestinal changes are alterations in the mesenteric glands. There are also important changes in other organs of the body, but most of the latter changes are common to many infectious diseases.

Four stages of the morbid process in the *intestine* may be distinguished: 1, the stage of congestion and simple inflammation of the mucous membrane; 2, the stage of medullary infiltration of the lymphatic structures; 3, the stage of retrograde metamorphosis and ulceration; 4, the stage of repair. These stages correspond approximately to certain periods of the clinical history. In the *first* week of the disease are developed congestion and inflammation of the mucous membrane of the small intestine, together with swelling of a certain number of solitary follicles and agminated glands. In the *second* week the general congestion of the mucous membrane diminishes, and the swelling and infiltration of the solitary follicles and Peyer's patches increase. During this week necrotic changes in the swollen follicles and patches take place. The casting off of the necrotic tissue results in the formation of the typhoid ulcers. In the *third* week the process of ulceration is completed. After this follows the period of repair of the ulcers, which occupies a variable time. It is to be noted that different stages of the lesions are usually found in the same case. The changes are most advanced in the small intestine near the ileo-cæcal valve.

At the onset of the disease the mucous membrane of the lower part of the small intestine is hyperæmic, swollen, softened, and coated with mucus. At the same time Peyer's patches and the solitary follicles of the lower half of the ileum begin to enlarge. As their enlargement increases the hyperæmia diminishes, except in the immediate neighborhood of the swollen follicles and patches; these remain surrounded by a zone of congestion. The morbid process is most intense in the ileum near the ileo-cæcal valve. In some cases the solitary follicles of the large intestine are swollen, and they may ulcerate. This involvement of the large intestine is more common in some epidemics than in others. The follicles of the jejunum may likewise become swollen, but ulceration in this situation is rare. According to Hoffmann,[1] the disease rarely extends higher than nine and a half feet above the valve. Usually, both Peyer's patches and the solitary follicles are involved. Sometimes the changes are more marked in one of these structures than in the other. In the stage of *medullary infiltration* the solitary follicles and Peyer's patches are swollen, and project, one, two, three, or even four, lines above the plane of the adjacent mucous membrane. The enlarged solitary follicles preserve their round shape and may attain the size of a pea. The swollen Peyerian plaques are elliptical or long-oval in shape. Swellings many inches in length and in breadth may be produced, especially near the valve, by the confluence of several enlarged patches. Usually the patches are indurated to the touch. Their surface is sometimes smooth, and sometimes it presents an irregular, granular aspect. The edges are generally abrupt, and sometimes they are constricted near the base, so that the patch has somewhat of a mushroom shape. Upon section the infiltrated follicles and plaques have a grayish-red or a whitish color. The appearance on section frequently resembles that of the white matter of the brain or spinal cord, and hence the name medullary infiltration which is applied to this condition. The peritoneum corresponding to the affected patches is usually congested. The swelling of Peyer's patches and of the solitary follicles is due to an increase in the number of lymphoid cells normally present in these structures. In addition to the ordinary lymphoid cells there are also found larger cells with a greater amount of granular protoplasm. Some of these large cells contain several nuclei. The new cells are generally considered to be the offspring of the pre-existing lymph-cells of the follicles, and the whole process is regarded as a hyperplasia of the lymphatic structures. The cellular infiltration is not confined to the limits of the solitary follicles or of Peyer's patches, but extends for a certain distance into the surrounding mucous membrane. It also extends in depth for a variable distance. Sometimes chains of lymphoid cells can be traced from the follicles through the muscular coat to the subserous tissue.

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[1] *Untersuchungen über die Path. anatom. Veränderungen der Organe beim Abdominaltyphus*, Leipzig, 1869.

The medullary infiltration may terminate in *resolution* or in *ulceration*. In the process of resolution the swelling of the follicles gradually diminishes, and many of the new cells undergo fatty degeneration and are absorbed. As a rule, this retrograde metamorphosis by degeneration and absorption occurs in some of the swollen follicles and plaques, while in others ulceration takes place. Sometimes the individual follicles of a Peyerian patch soften and discharge their contents. In these patches the intervening tissue forms a projecting network around the sunken follicles. This appearance characterizes the so-called reticulated patches. A reticulated aspect of the patches may be due also to simple degeneration and absorption of the infiltrated material in the follicles. The most characteristic termination of the process, however, is in the formation of a slough, and by the separation of the slough the production of the typical typhoid ulcer. The slough may occupy a whole Peyer's patch or only portions of a patch. It has usually a yellowish color from imbibition with bile. It may present a hemorrhagic appearance. The rapidity with which the sloughing process takes place varies in different cases. The process usually reaches its acme in the second week, but cases have been described in which the ulcers were fully formed on the seventh or eighth day of the disease. It must be remembered that it is by no means easy to determine in all cases the day on which the disease begins. It may be stated here that there is no constant relation between the extent of the intestinal lesions and the severity of the disease as denoted by the abdominal and other symptoms. The sloughing and ulceration take place earliest in the glands nearest the ileo-cæcal valve, and usually different degrees of progress are presented in the series above this point. The necrosis or sloughing of the infiltrated glands is sometimes ascribed to arrest of the circulation from the pressure upon the small blood-vessels exerted by the excessive production of new cells; but it is equally probable that it is due to the presence of the typhoid bacilli. The necrosis does not usually extend beyond the boundaries of the affected Peyerian patches. Sometimes the slough separates *en masse*, but more frequently it is thrown off in fragments, leaving at first small, irregular losses of substance. After the sloughing is completed ulcers take the place of the affected solitary follicles and Peyer's patches. These ulcers have certain distinctive features. Those resulting from necrosis of the solitary follicles are round, while those which take their origin from Peyer's patches preserve the oval or elliptical shape of these patches, the long axis of the ulcer being parallel to the direction of the intestine. The bottom of the ulcer is usually smooth, and often presents to view the transverse muscular fibres of the intestine. The edges are undermined and more or less swollen and congested. The ulcer may extend in depth and reach the deeper layers of the muscular coat, or its floor may consist only of the serous membrane. *Cicatrization* is the last step in the series of processes. Granulation-tissue springs from the floor of the ulcers. The undermined edges become united with the bottom of the ulcer. The resulting cicatrix is whitish, or more frequently pigmented, slightly depressed, and covered by epithelium. The villi and glands of the mucous membrane, according to most writers, are not regenerated. It is a remarkable fact that the cicatrices of typhoid ulcers have very little tendency to cause stenosis of the intestinal lumen. The process of cicatrization usually begins in the third week of the disease and continues during convalescence. The time occupied in the completion of this process varies in different cases. It is sometimes unusually slow—a fact which accounts for the persistence of the abdominal symptoms in certain cases for some time after the career of the fever is ended.

Perforation of the intestine is liable to occur in one or more of the ulcers. According to Hoffmann, perforation occurs in about 1 per cent. of the cases of typhoid fever, and in about 8 per cent. of the deaths from this disease. It is most frequent between the third and the fifth week of the disease. It is rarely caused by the primary sloughing, but is due to secondary ulceration after the separation of the slough. The perforation may be due to rupture of the thin floor of an ulcer by distension of the intestine with gas, by the contact of feces, or by the presence of round worms. The opening in the peritoneal coat may be of considerable size or it may be no larger than a pin's point. With intestinal perforation are associated appearances denoting acute diffuse peritonitis, the latter being occasioned by the escape of the gaseous and other contents of the intestine into the peritoneal sac. In rare instances the peritonitis is circumscribed around the seat of perforation by means of adhesions. Death may occur before sufficient time has elapsed for the production of inflammatory exudation. Profuse hemorrhage may occur from the primary sloughing or from the secondary ulceration.

More or less enlargement of the *mesenteric glands* is always associated with the intestinal lesions. The mesenteric glands which are in immediate relation to the affected Peyerian and solitary glands are especially enlarged. Their volume is often much increased, and not infrequently they attain to the size of a pigeon's egg. The enlargement is due to congestion and to the production of new cells resembling those normally present in the glands. The same large, granular, sometimes multinuclear, cells found in the infiltrated follicles of the intestine are met with in the swollen mesenteric glands. The morbid process, in fact, in these glands is of the same nature as that in the lymphatic structures of the intestine. Only in exceptional cases does the morbid growth which gives rise to the enlargement produce sloughing. This process of necrosis is, however, an occasional source of peritonitis developed in the course of the disease. Usually the glands diminish in size as the process of ulceration is going on in the intestine. The new cells undergo fatty degeneration. The resulting molecular mass in part is absorbed, and in part remains as a dry detritus which may become infiltrated with the salts of lime.

The *spleen* is enlarged, dark red in color, and softened. It is often two or three times the normal size, and may be larger. In very exceptional cases no swelling of the spleen is found. The enlargement is due to congestion and to hyperplasia of the cellular elements. Large cells containing one or more red blood-corpuscles or fragments of corpuscles are found in the splenic pulp and veins. Hemorrhagic infarctions are occasionally met with in the spleen. Instances of rupture of the capsule of the spleen with escape of blood into the peritoneal cavity have occurred. The result is a fatal peritonitis or sudden death.

Typhoid fever, like other infectious diseases, is attended by *parenchymatous degenerations*. The parts most affected are the cardiac muscle, the liver, the kidneys, and the glands of the stomach. The voluntary muscles also undergo the change. For the description of this metamorphosis the reader is referred to Part I. p. 53, and to the accounts of this degeneration in connection with diseases of the organs named. The degeneration is probably due to infectious blood-changes. Hyaline or pseudo-waxy degeneration occurs in voluntary muscles. This change affects only certain muscular fibres, not the whole muscle uniformly. It is observed most frequently in the recti abdominis, in the adductors of the thigh, and in the diaphragm. It may also occur in the cardiac muscle. The affected portions of muscle are pale and have a grayish waxy lustre. The muscular fibres are transformed into a homogeneous, friable mass without trace of the normal transverse striation. This change may lead to rupture of the muscular fibres with hemorrhage. This metamorphosis is not confined to typhoid fever, but it may occur in other diseases, and may be produced in animals by injuries to the muscles. Regeneration of muscular fibres destroyed by parenchymatous or by hyaline metamorphosis takes place during convalescence. (Vide p. 39.)

Changes in the *lungs* are present in nearly all cases of typhoid fever. Congestion and inflammation of the bronchial mucous membrane form an almost constant element in the disease. Hypostatic congestion and the condition called splenization of the lungs are found in a large proportion of cases. Pulmonary œdema is not uncommon. Pneumonia, usually in the lobular, but sometimes in the lobar, form, is a frequent complication. The lobular pneumonia is due often to aspiration of sputum and of foreign particles. Hemorrhagic infarctions may be caused by emboli derived from thrombi formed in the right cavities in the heart.

Ulcerations in the *larynx* are found in many cases. These occur most frequently on the edges of the epiglottis and on the posterior wall of the larynx just above the vocal cords. Writers are not agreed as to whether some of these ulcers be specifically typhoid or not. These ulcers may extend in depth and cause perichondritis. Œdema of the glottis is a rare complication of typhoid fever. Various forms of angina, catarrhal, follicular, croupous, and ulcerative, occur. The follicles at the base of the tongue are often swollen. Aphthous inflammation of the mouth and pharynx is not very infrequent in typhoid fever.

The *liver* is usually somewhat swollen, pale, and softened. The hepatic cells are in a condition of parenchymatous degeneration. Accumulations of lymphoid cells in the interlobular tissue about the branches of the portal vein are described by Wagner under the name of typhoid lymphomata. The bile is usually pale yellow and thin. The *kidneys* may present lymphomata similar to those in the liver. Parenchymatous degeneration of the renal epithelium is the rule. A genuine acute nephritis may occur, but it is rare. Hoffmann describes as a frequent occurrence proliferation and parenchymatous degeneration of the cells in the acini of the *salivary glands* and *pancreas.* Abscess of the parotid gland may occur in typhoid as well as in typhus fever.

No changes have been discovered in the *central nervous system* to explain the mental disturbance which characterizes this disease. There may be some increase of the cerebro-spinal fluid. Lymphoid cells are sometimes found around the ganglion-cells of the brain, but these wandering cells may be present in various other conditions.

The *blood* in typhoid fever, as in other infectious diseases, is poor in fibrin, and contains a relatively or absolutely larger number of white blood-corpuscles than normal.

No disease is accompanied by so great a number and variety of *complications* as typhoid fever. Some have already been mentioned. Other possible complications or sequelæ are—peritonitis without perforation, pleuritis, pericarditis, endocarditis, meningitis, cerebral hemorrhage, otitis media or externa, cystitis, epididymitis, orchitis, embolism of arteries of the lower extremities with gangrene, embolism of the pulmonary artery, thrombosis of the femoral vein with phlegmasia alba dolens, thrombosis of other veins, thrombosis of the heart, arteritis, necrosis of bone, periostitis, noma, gangrene of various parts of the body, bed-sores, septicæmia from bed-sores and other sources, pyæmia with abscesses in the lungs, spleen, and other parts, erysipelas, furuncles, retro-pharyngeal abscess, dysentery (during convalescence), abscess of the liver, suppurative nephritis, croupous or diphtheritic inflammation of the larynx and pharynx, hemorrhagic diathesis, icterus gravis, ulcer of the cornea, peripheral degenerative neuritis, neuro-retinitis, inflammation of the mucous membrane of the gall-bladder, and pyæmic or rheumatic polyarthritis.

A micro-organism peculiar to typhoid fever was discovered by Eberth in 1880, and was further investigated, and for the first time isolated in pure culture, by Gaffky, whose researches were published in 1884. Its pathogenic properties were demonstrated experimentally by Fraenkel and Simmonds in 1885.[1]

The organism is a short, thick bacillus with rounded ends. The average length is about one-third of the diameter of a red blood-corpuscle and three times the breadth of the bacillus. The individual bacilli vary considerably in size. According to Gaffky the bacillus forms endogenous spores, even within the body.[2] Each spore occupies one end of a bacillus. The bacillus can be cultivated upon nutrient gelatin, agar-agar, potato, blood-serum, and various other media. It grows readily at ordinary temperatures, but even more rapidly at the temperature of the human body. It does not form spores at a temperature much below 70° F. In nutrient gelatin it forms at first bluish-white, later whitish, opaque colonies upon the surface. It does not liquefy the gelatin. It is aërobic, but grows to some extent in the depths of the solid culture media. The most characteristic growth is upon the surface of sterilized, steamed potato, on which it grows rapidly, but in such a way as to leave the surface of the potato almost unchanged, so that to the naked eye no growth is apparent, or at the most a delicate pellicle may be discovered. If, however, scrapings from the surface of the potato be examined microscopically, there is found a luxuriant growth of the bacilli. Examined in a drop of liquid, the living bacilli are found to be endowed with the property of active movement.

The morphological characters of the typhoid bacillus are not sufficiently marked to distinguish it from other similar organisms. For this distinction it is necessary, as with other forms of bacteria, to take into consideration all of the properties of the organism, particularly its peculiar growth on potato. When all of these properties are considered, it is found that the typhoid bacillus is a species distinct from any other known species of bacteria.

The typhoid bacillus is constantly present in the early stages of typhoid fever in the affected solitary follicles and Peyer's patches, in the swollen mesenteric glands, and in the spleen. It has also been found in the liver, the kidneys, and the lungs. It has been found only exceptionally in the blood, although it is often found microscopically in the interior of small blood-vessels in the liver. The bacillus is usually absent after the fourth week of the disease, and is often not found during the fourth week. It may, however, be present at a much later period. Upon microscopical examination the bacilli are found in irregular clumps which stain deeply with various aniline dyes. The examination is usually made in sections of the spleen or of the mesenteric glands, as other organisms are usually mixed with the typical bacillus upon the surface of the intestinal lesions. Often prolonged search is required to discover the colonies, and sometimes the microscopical examination is negative. Gaffky found the colonies in 27 out of 28 cases, and in the exceptional case death occurred at the end of the fourth week. Other observers have been less fortunate in their microscopical examinations; but even in the cases with negative microscopical result the bacillus can be obtained by pure culture from the spleen unless the examination be made at too late a stage of the disease.[3] The culture method is a much more delicate method of demonstrating the presence of the typhoid bacilli than the microscopical examination. It is needless to say that the typhoid bacillus has not been found in any disease other than typhoid fever, although many diseases have been examined with that object in view. We may conclude, therefore, that a specific micro-organism is constantly present in the typical lesions of typhoid fever.

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[1] Eberth, *Virchow's Archiv*, 1880, Bd. 81; Gaffky, *Mittheil. aus d. Kaiserl. Gesundheitsamte*, Berlin, 1884; Fraenkel u. Simmonds, *Die aetiologische Bedeutung d. Typhusbacillus*, Hamburg u. Leipzig, 1886.

[2] Other observers have failed to detect spores. Vacuoles in the ends and sides of the bacillus are common.

[3] The greater certainty with which the presence of typhoid bacilli can be demonstrated in the spleen and other organs by means of cultures than by the microscope is prob-

Gaffky failed to obtain positive results by the inoculation of cultures of the typhoid bacillus in inferior animals, but the experiments of Fraenkel and Simmonds have shown that this bacillus possesses pathogenic properties. The majority of the mice inoculated died, and the majority of the rabbits inoculated within the peritoneal cavity or by injection into the blood died. Many of those which did not die were manifestly ill. In the organs of the dead animals were found the characteristic bacilli. The important lesions consisted in swelling of various lymphatic glands, including the solitary follicles and Peyer's patches, and of the spleen. It is not claimed that the disease produced is identical with typhoid fever as it occurs in man. It is not known that the inferior animals are susceptible to typhoid fever.

The constant presence in the characteristic lesions of typhoid fever of a pathogenic organism which does not occur in any other disease or in health hardly admits of any other interpretation than that this organism is the special cause of the disease. The idea that this organism is secondary to typhoid fever, being present simply because the disease produces favorable conditions for its growth, is met with insuperable difficulties.

The typhoid bacillus has been repeatedly demonstrated by means of cultures in the stools of patients affected with typhoid fever. This demonstration, however, does not always succeed.

The discovery of the typhoid bacillus makes it possible to determine in many cases whether the various and frequent complications of typhoid fever be the direct effect of the typhoid poison or not. The question here raised has been answered in only a few instances. It has been found that the abscesses and the severe inflammations of the throat have been caused by the secondary invasions of other organisms. A secondary invasion of micrococci is not uncommon in typhoid fever.

We are ignorant as to the manner in which the typhoid bacilli produce the manifestations of the disease. The fact that the bacilli are found in small number or not at all in the blood, taken in connection with the severe constitutional and nervous symptoms, suggests the possibility that they produce some toxic substance which is absorbed by the blood. (The etiological relations of the typhoid bacillus will be further considered under Causation.)

CLINICAL HISTORY.—Typhoid fever, as a rule, is developed gradually. After the development of the disease the most convenient division of stages is into septenary periods. The career of the disease will embrace the first, second, third, and sometimes a fourth, fifth, sixth, or even a seventh, week.

In a large proportion of cases patients are unable to fix the precise date of their ailments, so imperceptible is the beginning of the disease. It is therefore often not easy to determine accurately the duration of the prodromic period. This period ends when the disease is developed. My rule

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ably owing to the circumstance that isolated typhoid bacilli or their spores cannot be discovered microscopically, or at least are readily overlooked. Reher and Fraenkel and Simmonds find that from the isolated organisms the characteristic colonies may develop in the spleen after death, so that they advise, for microscopical purposes, preserving the spleen for twenty-four hours after death, wrapped in a cloth wet with an antiseptic solution, before hardening in alcohol. For one familiar with the modern methods of cultivation on solid transparent media it is very easy to demonstrate the presence of the typhoid bacillus by means of cultivation has been to consider the fever as established when the patient takes to the bed. This is an arbitrary criterion, and not always accurate, but the instances in which patients take to the bed before and after the full development of the disease will about compensate for each other. The first chill or chilly sensations and an increase of the temperature are more correct criteria; but in a large majority of cases patients do not come under observation until the fever is developed, and they often cannot recall the first chill or chilly sensations. Judged by the rule just stated, the prodromic period varies between one day and ten days, and the mean duration is about five days. Exceptionally, the attack is abrupt, the patient taking at once to the bed.

The following are the prodromic symptoms: Chills, more or less pronounced, or chilly sensations, recurring irregularly, sometimes followed by perspiration and sometimes not; cephalalgia, the pain generally referred to the frontal region; mental irritability, with difficulty of concentrating the attention or exerting the faculties of the mind; loss of appetite, occasional nausea, and sometimes slight vomiting; epistaxis in a pretty large proportion of cases; pain in the loins and limbs; looseness of the bowels or hypercatharsis if a mild purgative be given (the bowels may at first be constipated); lassitude and progressive debility until at length the patient, who up to this time has kept up with more or less effort, feels compelled to take to the bed. The duration of this period is of importance in diagnosis.

The symptoms which after this period make up the clinical history may be distributed into those referable to the countenance or general aspect, the nervous system, the digestive system, the skin, the respiratory system, the circulation, the temperature, and the urinary system.

Countenance and General Aspect.—There is no marked alteration of the countenance for the first few days. The face in the early period of the disease is usually more or less flushed. Afterward the countenance presents a dull or listless expression, and in severe cases there is lack of expression or an appearance of stupidity. The latter corresponds with the gravity of the disease, and is more and more marked as the disease continues. These alterations of the physiognomy are dependent on the mental condition.

The surface presents slight or moderate capillary congestion, like that produced by the action of cold. This is most marked on the face, especially on the cheeks. Distinctly circumscribed redness of the cheeks is rare save in the cases in which the disease is complicated with pneumonitis. The redness disappears on pressure, and returns more or less quickly. In some cases the hue of the surface is slightly dusky. The capillary congestion observable on the face is diffused over the body, but, next to the face, it is most marked on the hands and arms. It is rational to consider this appearance as denoting paresis of the peripheral arteries. Slight or moderate congestion of the conjunctiva is not infrequently observed.

Nervous System.—Pain in the head is more or less complained of during the first week. It is not always a prominent symptom, and it never has the intensity which belongs to the pain in the first stage of acute meningitis. It gradually becomes less and less a subject of complaint, and usually ceases during the second week. The cephalalgia is not accompanied with notable intolerance of light and sounds. Pain in the back is also complained of in the early part of the disease, but this is rarely a very prominent symptom.

Delirium is manifested in the majority of cases. As a rule, it is not manifested until the second week, and sometimes not until the third or fourth week. Exceptionally, it is manifested in the first week, and even when the patient first takes to the bed. Cases differ much as regards the degree of delirium. It may be slight, or, on the other hand, a very prominent symptom. Generally, the first evidence of mental aberration is temporary confusion on awakening from sleep, the patient being unable to recall where he is and asking incoherent questions. Increasing, the patient talks incoherently and mutters like a person talking in sleep. Efforts to get out of bed are common, and when asked where they would go patients often say they wish to go home. In most cases they are easily persuaded to lie down, but after a few moments the effort to get up is repeated. If not watched, they sometimes get up and succeed in getting on their clothes. The delirium is always greater during the night, and it may be manifested only at that time. In the great majority of cases the delirium is of a quiet, passive kind. It apparently proceeds from weakness of mind, there being inability to carry on connected trains of thought. The incoherent talking or muttering usually relates to habitual pursuits. The patient is in a dreamy state, a succession of disjointed ideas passing through the mind.

There are occasional exceptions to the rule regarding the kind of delirium. It is sometimes noisy and active. The patient shouts and makes strong and persistent efforts to get up. Constant forcible restraint may be necessary. Fixed delusions, either of the senses or ideas, during the progress of the disease are certainly rare; but they may occur at the time of convalescence. In one of my cases the patient fancied that he had become immensely rich, and this delusion continued for several days after convalescence. In another case, the patient being a young woman, the delirium in convalescence had the character of delirium tremens. The delirium during the course of the fever is in general marked in proportion to the intensity of the disease. So far as my observations go, persisting, active, or violent delirium, requiring restraint, is an extremely unfavorable symptom.

Patients are sometimes perfectly coherent, but not rational. They reply to questions at haphazard, and no reliance is to be placed on their statements with respect to the events of their illness. In some cases there are no manifestations of delirium during the whole course of the disease, but after recovery there is very little recollection of anything that occurred. The delirium sometimes has an hysterical character, consisting in emotional manifestations—weeping and sobbing—the emotions being very rarely mirthful.

Delirium, however prominent as a symptom, is no evidence of encephalic inflammation. The appearances of meningitis are not found after death in cases characterized by very active delirium, nor is there an abnormal amount of congestion.

Irrespective of delirium, the mental condition in the second, third, or fourth week, or later, is characterized by hebetude, indifference, and inanimation. Blunted perception is shown by various circumstances. The patient asks for nothing—not for drink, although the mouth may be dry and desiccated, or for change of the position of the body. Flies creeping over the face may occasion little or no annoyance. In grave cases the urine and feces may be passed in bed, not from paralysis of the sphincters, but through indifference and a reluctance to make any effort. The special senses are impaired, especially the sense of hearing, and deafness in one or both ears is frequently marked. General and special sensibility are diminished, as a rule, in proportion to the intensity of the disease. Frequently there is dilatation of the pupils.

Wakefulness is complained of during the first few days, the patient experiencing discomfort from the want of refreshing sleep. Afterward the want of sleep may not be appreciated, owing to the mental condition. The patient, indeed, may seem to doze most of the time, but without obtaining true sleep, being easily aroused, but soon relapsing into a pseudo-somnolent state. This state, in which the patient may be said to be both sleeping and wakeful, is called *coma-vigil*, the significance of the term depending on the incongruity of the words which it combines. This name is also applied to a state occasionally observed in which the patient is unconscious with the eyes open. In coma-vigil or the pseudo-somnolent state, from which the patient is readily aroused, the mind is frequently occupied with a series of disconnected ideas or dreamy delusions which give rise to the muttering delirium. This combination of stupor and delirium is expressed by the term *typho-mania.* The want of true sleep doubtless contributes not only to the exhaustion, but to delirium and other disordered nervous phenomena, as in delirium tremens.

Coma-vigil and typho-mania do not involve a tendency to true coma; but in a certain proportion of cases coma is gradually developed in the latter part of the career of the fever, and under these circumstances a fatal result is to be expected. In a certain proportion of cases coma is sudden. It occurs unexpectedly, and in mild as well as in severe cases. These cases end fatally, the mode of dying being by apnœa. This apoplectic coma is often preceded by disturbance of the rhythm of respiration, the inspiratory act being shortened and quickened.

Other symptoms referable to the nervous system, or ataxic symptoms denoting gravity of disease, are—grasping at invisible objects or carphologia, pulling up of the bedclothes or fumbling with the body-linen, visible twitchings of the muscles of the face and of the extremities, and movements of the tendons of the wrist perceptible to the touch (the latter called *subsultus tendinum*), rigidity of the muscles of the neck or extremities, and convulsions. Muscular rigidity and convulsions are very rare, and betoken a fatal termination. There is reason to believe that the pathological condition giving rise to convulsions with coma is frequently, if not always, uræmia. In these cases the urine will be found to be albuminous.

Digestive System.—Anorexia is the rule, but to this rule there are exceptions; the appetite being sometimes preserved throughout the disease. Food is sometimes acceptable when, owing to the mental condition, it is not asked for. Thirst is usually a prominent symptom until perception becomes blunted. Drink, although not asked for, is often taken with avidity when given. The condition of the mouth would involve a desire for drink were the ability to perceive morbid sensations not impaired.

The tongue generally presents morbid appearances. It may be simply furred or frosted, but it is oftener covered with a coating more or less thick, which in different cases is whitish, yellowish, brownish, or even black. Not infrequently the coatings are thrown off once or repeatedly, and the surface is then usually reddened. Exfoliation of the coating or its gradual thinning, the surface being moist and of a natural color, betokens convalescence. The varied appearances of the coatings have no special significance. The surface of the tongue is sometimes reddened and smooth or glazed. It sometimes becomes dry and hard. This occurs in coma-vigil. The patient breathing with the mouth open, the surface is desiccated. It sometimes becomes cracked and deeply fissured. Tremulousness of the tongue, as in cases of delirium tremens, is sometimes observed, usually preceding or accompanying grave ataxic symptoms. The tongue in some cases at an advanced period is protruded with apparent hesitation and difficulty, and when protruded may not be withdrawn, apparently from forgetfulness. The delay in protruding and withdrawing the tongue represents the weakness and slowness of the mental acts.

Dark or black matter, called *sordes*, after the first week often accumulates upon the teeth and lips, especially in grave cases. Hemorrhage from the gums is an occasional symptom, occurring in mild as well as in severe cases. Redness of the gums and bleeding on slight pressure are common.

A rare complication is inflammation of one or both of the parotid glands.

It leads to notable enlargement, and the appearance is like that of ordinary *parotiditis* or *mumps*; but, unlike the affection just named, in the great majority of cases suppuration takes place, and not infrequently there is more or less sloughing of the areolar tissue. This complication adds to the danger and retards convalescence. It may occur at any period of the febrile career or during convalescence. It is not to be regarded as a critical event. The discharge of pus is sometimes into the meatus auditorius. This complication occurs in typhus as well as in typhoid fever. It occurred in 5 of 30 cases of typhus and typhoid fever which I recorded in the winter of 1849–50. Prior to that year I had never met with an instance, and since that year I have met with a very few examples, but in a much larger field of observation.

Vomiting is an occasional symptom. Generally it is a result of over-ingestion. As regards the occurrence of this symptom, typhoid fever is in striking contrast with remittent fever.

Diarrhœa or looseness of the bowels exists in a large majority of cases, and this belongs among the diagnostic symptoms of the disease. The dejections often have a yellow, ochre color. They give an alkaline reaction, but this is not distinctive of the disease. Exceptionally, constipation exists, and the dejections may have a natural appearance throughout the disease. The bowels are often evacuated in bed, either from indifference or from paralysis of the sphincter ani. Involuntary evacuations denote gravity of disease.

Hemorrhage from the bowels is an important event in the clinical history of this disease. It occurs in a proportion of between 5 and 8 per cent. The hemorrhage may be slight, moderate, or profuse. It may cease after a single occurrence, or there may be a greater or less number of recurrences. The loss of blood is sometimes the immediate cause of death. Statistics, however, show that in the larger number of cases patients recover. Even very profuse hemorrhages do not always prove fatal. I have known the loss of blood to occasion complete collapse for several hours in cases which ended in recovery. The hemorrhage occurs late in the disease, and in most instances is from the intestinal ulcers. Its occurrence may be foreshadowed by a sudden and notable decline of the temperature of the body. I have met with a case in which the decline of the axillary temperature to 98° was for a time considered as evidence that the fever had ended, but within a few hours profuse hemorrhage from the bowels occurred and the patient died from the loss of blood.

Other abdominal symptoms are—meteorism or tympanites, tenderness or pain on pressure, and gurgling. The abdomen is always resonant from the presence of intestinal gas. Frequently there is more or less distension, and sometimes the tympanitic enlargement is great. Tenderness on pressure in the iliac region, especially on the right side, is very rarely absent. Sudden and forcible pressure may be required for the manifestation of pain if perception be much blunted. The tenderness is often marked, and sometimes it extends over the greater part of the abdomen. Gurgling on pressure in the iliac region as a sound and as a tactile sensation is a frequent symptom. These three symptoms—namely, meteorism or tympanites, iliac tenderness and gurgling, especially the two former conjoined with diarrhœa or looseness and ochre-colored discharges—form a group of abdominal symptoms highly diagnostic of this form of fever. They may, however, all be absent. The tympanites, tenderness, and gurgling, as regards their prominence, are not proportionate to the amount of intestinal lesions. By percussion and by palpation over the region of the spleen this organ is found to be enlarged.

Perforation of the small—or in rare instances the large—intestine takes place in a certain proportion of cases; namely, in between 8 and 10 per cent. of fatal cases, and between 1 and 2 per cent. of all cases. It takes place late in the disease or during convalescence, and sometimes even after apparent recovery from the fever. It occurs as often in mild as in severe cases. It occurs oftener in men than in women, and is very rare in children.

Perforation gives rise to peritonitis, which is generally developed abruptly. The sudden occurrence of notable tenderness diffused over the abdomen, abdominal pain, increased tympanitic distension, rigidity of the abdominal walls, with marked frequency of pulse, prostration, haggard expression of countenance, etc., point to peritonitis from perforation. Replacement of hepatic flatness by tympanitic resonance usually occurs in perforative peritonitis, but it may be absent, and this symptom may be present without pneumo-peritoneum. In some cases, the perforation being quite small, the development of the peritonitis is gradual, and it may be circumscribed under these circumstances. Peritonitis does not invariably denote perforation. It may be caused by the evacuation into the peritoneal sac of liquefied matter within a mesenteric gland. It may occur irrespective of either this cause or perforation. Probably in most of the reputed cases of recovery after the occurrence of peritonitis perforation was not involved. Peritonitis from perforation is almost certainly fatal.

Skin.—Of the symptoms referable to the skin, the most important is a characteristic eruption. The eruption consists of isolated papules, generally limited to the trunk, of a rose or pink color, called by Louis lenticular rose-colored spots. They are slightly elevated, and therefore are *papulæ*, not *maculæ*. The elevation is distinctly felt if the finger be carried lightly over them. They are slightly oval in form, and are from one to two lines in diameter. The redness momentarily disappears on light pressure. The number of papules is generally small, in the majority of cases not exceeding fifteen or twenty, and sometimes only four or five can be discovered. Exceptionally, they are abundant over the trunk, and the eruption sometimes extends to the extremities. Fresh papules appear from time to time during the course of the fever, taking the place of those of anterior date. They are to be sought for over the chest and abdomen in front and over the back, and they are sometimes found exclusively in the latter situation.

Dating from the time of taking to the bed, the average time of the appearance of the eruption is about the seventh day. Papules continue to disappear and to appear during the career of the disease, and sometimes the eruption persists into the stage of convalescence. The papules are not apparent after death.

The eruption is not invariably present. The proportion of cases in which it occurs varies in different years, as my statistics show. It appears to be less frequent in the country than in the city. It is less frequent in children than in adults. The copiousness of the eruption is no evidence of the severity of the fever, and on the other hand it is as likely to be absent in mild as in severe cases. A scarlet rash occasionally precedes the characteristic roseolar eruption.

Miliary vesicles or sudamina are not infrequently observed in the middle and latter part of the disease on the neck, chest, and abdomen. This eruption is observed in various affections in which perspiration occurs. Petechiæ or minute ecchymoses are occasionally observed. These do not denote unusual gravity of the disease. They occur in mild as well as in severe cases. Herpes labialis is so rare an occurrence in typhoid fever that its presence is evidence against the diagnosis of this disease.

Bluish patches (peliomata or *tâches bleuâtres*) of irregular form, from three to eight lines in diameter, are occasionally observed on the abdomen, back, and thighs. They are not peculiar to this disease, and are more likely to occur in mild than in severe cases.

Perspiration, more or less abundant and recurring more or less frequently, is observed in about one-half of the cases, exclusively of its occurrence at the time of convalescence or just before death. It occurs in about an equal proportion of fatal cases and of those ending in recovery. Observation does not show a connection between this symptom and any particular antecedent events; but, taking the frequency of the pulse as a criterion of the gravity of the disease, and comparing the pulse before and after the occurrence of perspiration, in the majority of cases improvement follows. While this symptom, therefore, has no bearing on the prognosis, it often betokens some amelioration. As a rule, during the greater part of the career of the fever the skin is dry.

Gangrene is liable to occur in situations exposed to pressure. Spontaneous gangrene of the extremities has been observed, the probable explanation being arterial thrombosis. Gangrene and troublesome ulcers are liable to follow vesication. The liability to bed-sores on the nates, hips, and shoulders is to be borne in mind. These may be prevented by changing from time to time the position of the body, by the use of air-pillows, etc. Erysipelas is an occasional complication of typhoid fever.

Respiratory System.—Cough is not a prominent symptom unless an important pulmonary complication exist, but slight or moderate cough is almost invariably present, proceeding from subacute bronchitis. A sibilant râle is frequently heard over the chest on auscultation.

Pneumonia is a not infrequent complication. It is rendered probable by prominence of cough and accelerated breathing, but the proof of its existence is afforded by the physical signs. It is sometimes latent as regards diagnostic symptoms, and is determinable only by physical exploration. Its occurrence adds to the danger, but it by no means precludes recovery. Hypostatic congestion and œdema of the posterior portions of both lungs are incident to the feeble circulation in the latter part of the disease. These physical conditions are represented by dulness on percussion, weak respiratory murmur, and subcrepitant râles. Irrespective of any pulmonary complication except subacute bronchitis, the frequency of the respirations is increased, the average being about 21 per minute.

Frequent sighing in the early part of the disease is a forerunner of ataxic symptoms of grave omen. A spasmodic or jerking inspiration when pneumonia does not exist is a precursor of coma. This statement holds good in other forms of fever. The importance of this symptom is enhanced by the fact that it may sometimes be observed when no other symptoms denote impending coma. This was true of several cases which I have observed.

Epistaxis has been mentioned as a symptom of diagnostic value early in the disease. It occurs not infrequently afterward. Generally the hemorrhage is small, but occasionally it is profuse, and it may require to be arrested by mechanical means. When slight or moderate it does not appear to have any influence on the progress of the disease. Uterine hemorrhage is not uncommon. Its occurrence is not limited to the period when menstruation should take place.

Pharyngitis, laryngitis, and œdema of the glottis are occasional complications of typhoid fever.

Circulation.—More or less acceleration of the pulse belongs to the history of the disease. The acceleration varies considerably in different cases, and at different periods of the disease in the same case. There is a greater average frequency in fatal cases than in those ending in recovery. The danger is usually considerable if the pulse for many days exceed 120 per minute, and the danger is augmented in a geometrical ratio as the frequency rises higher than this. The mean frequency in cases not proving fatal of those which I have analyzed was 93; and in the fatal cases, 110. In some mild cases the average frequency during the whole of the disease is less than the average frequency of health. In one of my cases the average frequency was 69. In these cases, however, the pulse at times exceeds the healthy standard, hence the pulse may at times fall below the standard of health. This is not infrequently observed at or near the time of convalescence. I have noted it as low as 64, 60, and 40. Others have observed it to fall still lower. Oftener the pulse continues more or less accelerated after convalescence is declared. A sudden and considerable increase of the frequency of the pulse often denotes the occurrence of some important event, such as pneumonia or peritonitis.

As regards characters of the pulse other than frequency, it is quick and vibratory, not hard or resisting. It denotes increased action, but not increased power, of the ventricular systole. It becomes feeble or compressible in proportion as it is frequent. The force is always notably diminished if the frequency much exceed 120. An unfavorable progress of the disease toward asthenia is shown by progressive increase in frequency and diminution of force. Under these circumstances the first sound of the heart, on auscultation over the apex, is notably weakened and may be inappreciable. The form of the pulse, as traced by the sphygmograph, presents the following characters: a short and nearly vertical line of ascent, indicative of weakness and quickness of the ventricular systole; a line of descent, denoting the free passage of blood from the arteries in the capillaries, and notable dicrotism. The dicrotism of the radial artery is sometimes appreciable by the touch. These characters belong to the graphic pulse also in cases of typhus and in all affections with which is associated the typhoid condition.

Temperature.—More or less increase of the heat of the body is invariable. In general, the thermometer in the axilla shows a daily increase of temperature for the first five or six days. This gradual rise of temperature from day to day during the first week is diagnostic of typhoid fever. At the end of this period the temperature rises to at least 103° F. If, in the progress of the disease the thermometer show much increase above this, the prognosis is unfavorable, whereas a decline of the temperature in the morning to near the normal standard is evidence of convalescence. During the progress of the disease there is, as a rule, an oscillation of temperature between morning and evening, the difference varying between half a degree and a degree and a half up to the time of convalescence, when the oscillations become much greater, the difference being four to five degrees, the temperature in the morning falling to the normal standard, 98°, or lower. The difference between the morning and evening temperature at the time of convalescence has been observed to be more than nine degrees. The temperature in typhoid fever does not always pursue the typical course which has been described. The temperature may reach its acme by the end of the third day. Not very infrequently the course of the temperature is of a remittent type, and even distinctly intermittent types of temperature have been observed during the first and second weeks. A high elevation of temperature in the morning—namely, 106° to 108°—indicates the approach of death. The laws of the disease as regards temperature are sufficiently characteristic to render the thermometer useful in diagnosis as well as in prognosis. A sudden and considerable rise of temperature during the progress of the disease points to the occurrence of an inflammatory complication. A sudden and considerable fall of temperature, other symptoms not denoting convalescence or improvement, is unfavorable. This often indicates hemorrhage from the bowels. It is important, however, to note that, irrespective of complications, the temperature in some cases becomes increased, and the rise may continue for several days; and on the other hand a decline of from one to two and a half degrees sometimes takes place, lasting for several hours, without any obvious cause and having no special significance.

Urine.—The urine is usually scanty and of high specific gravity until the approach of convalescence, when it becomes abundant and of a low specific gravity. During the progress of the disease, as a rule, the urea and uric acid are absolutely increased and the chlorides are diminished. The coloring matter is also more abundant than in health.[1] The presence of albumen during a part of the febrile career is common. An abundance of albumen denotes gravity. This symptom occurs in the middle or latter part of the disease, and it may continue for a period varying between twenty-four hours and twelve days. In the fatal cases in which it occurs it continues up to the time of death. In these cases the kidneys after death may be found to be simply congested, no deposit or structural change existing.

A genuine acute diffuse nephritis may develop during the course of typhoid fever, even in the early stages. Cases in which the renal symptoms predominate have been designated as renal typhoid. Such cases are rare, and may offer considerable difficulty in diagnosis. It is probable that coma and convulsions occurring in the progress of typhoid fever are due to uræmic poisoning. I have met with a case in which, following a large hemorrhage from the bowels, profuse hæmaturia took place, the blood passed from the bladder forming clots after its emission. The patient became much blanched from the loss of blood, but he recovered.

The urine is sometimes retained in cases of typhoid fever, and owing to the blunted perception the bladder may become greatly distended. The practitioner should not omit to ascertain the condition of the bladder by examination of the abdomen during the progress of the disease, and resort seasonably to the use of the catheter when required. On the other hand, the urine is often passed in bed, sometimes from indifference and sometimes from incontinence.

In order to determine the duration of the disease it is necessary to fix upon certain points which shall mark the beginning and the end of the febrile career. The time of taking to the bed is a convenient, and in a collection of cases a tolerably correct, criterion of the development of the fever. It is difficult to find any one circumstance which will answer equally well to denote the date of convalescence. The career of the fever rarely ends abruptly; the termination in convalescence, like the development, being gradual. The decision that a patient is convalescent is a matter of judgment, and is to be based on the *ensemble* of symptoms. Of 42 cases ending in recovery, analyzed with reference to the duration of the fever, dating from the time of taking to the bed to the time when the improvement in all the symptoms was sufficient for the patients to be considered convalescent, the average duration was sixteen days. The maximum duration was twenty-eight days, and the minimum five days. The longest duration in any case under my observation was in a female hospital patient, the disease continuing in this case without any important complication fifty-eight days. It is perhaps of importance to state that these cases were observed long before the recent introduction in medical practice of antipyretic measures of treatment. The mean duration in 75 cases observed by Murchison was a fraction more than twenty-four days. This greater duration, as compared with my cases, is probably owing to the beginning being fixed at an earlier period than the date of taking to the bed. Of 45 fatal cases in my collection, the mean duration was a fraction more than fourteen days, the maximum being twenty and the minimum nine days. The mean duration in 12 fatal cases observed by Murchison was a fraction more than twenty-two days. The average duration of convalescence is between one and two weeks. The return of the axillary temperature to the normal standard—that is, complete defervescence—shows of course that the febrile career is ended. Sometimes the defervescence is rapid, and the temperature quickly falls to that of health; but generally it is gradual, and paroxysms or exacerbations of fever, as indicated by the thermometer, are liable to occur for some days after the appetite, mental condition, and other symptoms denote convalescence. The temperature may be kept up by complications which persist after the career of the fever is ended. A temporary fever often accompanies the change of diet from liquid to solid animal food.

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[1] The so-called diazo-reaction, first described by Ehrlich, can usually be produced in the urine of typhoid-fever patients. The test consists in mixing the urine with a solution of sulfo-diazo-benzol, and then adding ammonia, when, if the reaction is present, the urine acquires a red color which is followed by the precipitation of a greenish sediment. The reaction is found chiefly, although not exclusively, in febrile diseases. Its diagnostic and prognostic importance is not yet established, the results of different observers being contradictory (Ehrlich, *Charité Annalen*, Jahrg. viii., and *Deutsche med. Wochenschrift*, 1883, No. 38).

Relapses of typhoid fever sometimes occur. Many examples have fallen under my observation. A return of the fever may take place after ten days or a fortnight from the date of convalescence, and the patient pass through a second career, the eruption and other characteristic symptoms being reproduced. The duration of the second career is usually shorter and the severity greater than the first, but a fatal termination is rare.

Complications which are likely to arise in the course of typhoid fever have been noticed in connection with the anatomical appearances and with the symptoms referable to different anatomical systems. The occasional occurrence of thrombosis of the iliac or femoral vein on one side or on both sides is not rare, and is attended with swelling and pain of the corresponding limb. This complication rarely leads to evil consequences, but sudden death has been known to occur from an embolus detached from the thrombus and occluding the pulmonary artery. Typhoid fever may be associated with intermittent fever (typho-malarial fever), scarlatina, rubeola, diphtheria, and perhaps with typhus. Occasional sequels are acute miliary tuberculosis, pulmonary phthisis, and subcutaneous abscesses. Progressive emaciation and death from inanition have been known to follow; but in many instances nutrition becomes extremely active after recovery and the patient attains a greater weight than ever before. The mental powers in some cases are enfeebled for a considerable period.

CHAPTER II.

CONTINUED FEVERS (CONTINUED).

Causation of Typhoid Fever; Diagnosis; Prognosis.—Typhus Fever: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis.

THE anatomical characters and clinical history of typhoid fever have been described in the preceding chapter; and it remains to consider the causation, diagnosis, and prognosis of this disease before entering on the consideration of typhus fever.

CAUSATION.—Typhoid fever is not restricted within any geographical limits. It is endemic in every quarter of the globe. It prevails, however, often within circumscribed districts, and not infrequently does not extend beyond a few houses or even a single house. Although it now prevails within many malarial regions, it is certain that in some situations in this country well-marked cases of typhoid fever were hardly known so long as malarial fevers were rife, but the former became the common form of fever after intermittent and remittent fever ceased to prevail. As regards the season of the year, it shows in this latitude a decided predilection for the autumn.[1] It is stated that a warm, dry summer favors the occurrence of the disease in the following autumn. According to Buhl and Pettenkofer, the disease prevails in Munich when the level of the ground-water is low, and the prevalence has been observed to end when the level of the water has been raised by abundant rain. A similar relation has not been observed in all places.

The susceptibility to typhoid fever is greatest between the ages of fifteen and twenty-five, next between ten and fifteen, and next between twenty-five and thirty. It is rare in infancy, but not infrequent in childhood. A considerable proportion of cases of so-called infantile remittent fever are cases of typhoid fever. The susceptibility is much diminished after sixty, but the disease may develop in persons more than seventy years of age.

Both sexes appear to be about equally liable to the disease. Generally, persons are in good health when attacked. Other diseases do not seem to predispose to typhoid fever. No causative influences relating to social position, occupation, or habits of life have been ascertained. A certain degree of protective influence against typhoid fever seems to be exerted by phthisis and other diseases attended by cachexia and severe anæmia, but this protection is not absolute. A protective influence exerted by pregnancy and lactation, if it exist at all, is much less than was formerly supposed. Typhoid fever developed soon after delivery is liable to be mistaken for puerperal fever. Pregnant women when attacked with typhoid fever generally abort, but this does not seem to influence the prognosis unfavorably. The fœtus is generally dead or dies soon after birth. The typhoid bacillus has been found in the fœtus, in which have also, but very rarely, been found the lesions of the disease.

New-comers in an infected district are more likely to be attacked than those who have long resided there. As of those who are exposed to the typhoid-fever poison only a certain number are attacked, it is evident that a certain degree of individual immunity exists.

The reasons have already been given for believing that the so-called bacillus of typhoid fever is the specific cause of the disease. Of the properties of this organism bearing upon the causation, may be mentioned its growth at ordinary temperatures, although best at a temperature between 85° and 100° F.; its growth upon a great variety of substances, such as vegetables, milk, meat-infusions, etc.; its formation of spores; and its presence in the typhoid stools. We may therefore infer that the typhoid bacillus may grow readily under ordinary conditions outside of the body, as in the soil or in liquids containing sufficient nutriment, and that by the formation of spores it may preserve its vitality for a long time, even under unfavorable conditions. There is no evidence that the typhoid bacillus exists in the exhalations from the patient.

Typhoid fever is communicable, but rarely if ever in the same way as the strictly contagious diseases, such as typhus fever, smallpox, scarlet fever, and relapsing fever. Emanations from the bodies of those affected with the disease do not infect the surrounding atmosphere, and hence in hospital wards the disease is not diffused by typhoid cases among patients affected with other diseases if care be taken to thoroughly disinfect the dejecta. Physicians, nurses, and members of families brought into close proximity to cases seldom contract the disease in consequence. Nevertheless, the causative germs multiply within the bodies of typhoid patients, and are expelled from the body with the intestinal dejections. It is not necessary to suppose that these germs, the typhoid bacilli, must undergo any further modification or development outside of the body in order to communicate the disease. It is possible to explain the infrequency with which those in attendance upon typhoid patients contract the disease by the fact that with ordinary care portions of typhoid stools are not likely to be taken into the alimentary canal, and there is no reason to suppose that any exhalations from the patient contain the bacillus. Washerwomen not infrequently contract the disease from washing linen soiled with typhoid excreta, and it is easy to understand how infection might take place in this way.

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[1] Of 5988 cases admitted during twenty-three years in the London Fever Hospital and analyzed by Murchison, 41 per cent. occurred in autumn, 21.3 per cent. in winter, 12.7 per cent. in spring, and 24.8 per cent. in summer.

Of 45 cases which I have analyzed with reference to this point, 16 were in October, 9 in November, and 12 in December.

It is probable, however, that the typhoid stools are the chief source of infection of a locality in which the disease becomes endemic. Instances might be multiplied to show that persons who have contracted typhoid fever in one locality, then go to another locality and become a source of infection for a region in which the disease has not hitherto prevailed. In the preceding editions of this work I have reported a remarkable instance of this nature. In this instance a stranger ill with typhoid fever came to a little settlement called North Boston, consisting of nine families. Up to this time typhoid fever had never been known in that neighborhood. In a few days the stranger died, and in a month more than one-half of the population, numbering 43, had been affected, and 10 died. Both the symptoms and post-mortem examination showed the disease to be typhoid fever. Of the nine families, one family escaped which obtained its water from a source different from the others, which used a common well.[1]

That infection with typhoid fever often takes place from the drinking-water cannot be doubted. The typhoid bacilli have been found in the drinking-water of regions where typhoid fever was prevailing as an epidemic (Mörs, Michael). Such water may be perfectly clear and apparently pure. Meade Bolton has shown that typhoid bacilli may exist in drinking-water for at least a month, although they do not multiply unless the water contain an amount of organic matter not likely to be present in any water which would be used for drinking purposes. In a number of instances of infection through the water it has been possible to show some communication or leakage between a well used for drinking purposes and a privy-vault, cesspool, sewer, or drain. That such contamination, as well as the contamination of the soil in a similar way, is an important factor in the causation of the disease seems certain; but, in accordance with our present views, we must suppose that no amount of contamination by the ordinary products of decomposition is capable of causing typhoid fever, and that the typhoid bacillus must always be present when this occurs.

Allied to epidemics traceable to the drinking-water are epidemics referable to the milk-supply. A number of such epidemics have been reported, especially in England.[2] Here it is probable that the vehicle of infection is the water used in cleansing the cans or in diluting the milk. As already mentioned, milk is a favorable medium for the growth of the typhoid bacillus. Boiling the milk is the only sure preventive.

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[1] For fuller details concerning these cases, vide A. Flint, *Clinical Reports on Continued Fever, etc.*, 1852, and *American Journal of the Medical Sciences*, July, 1845.

[2] Dr. F. C. Curtis, in the *Fourth Annual Report of the State Board of Health of New York*, gives an account of an outbreak of typhoid fever at Port Jervis in the autumn of 1883, traceable to milk-supply. (See, also, *The Lancet*, Feb. 7, 1885.)

Some believe that the chief source of infection is through the atmosphere in regions in which the ground contains the specific germ. It is probable that infection may take place from uncooked food, especially from fruits and vegetables which are contaminated by the presence of the specific organism.[1]

A question of importance is whether an outbreak of typhoid fever be always referable to the introduction of the typhoid germ derived directly or indirectly from a person affected with the disease, or whether the special poison may be generated outside of the human body. That the former method of infection is a common one cannot be doubted, but the properties of the typhoid bacillus are such that it is capable of living independently of the animal body, and there is therefore no *a-priori* objection to the assumption that typhoid fever may develop in regions which have never been visited by any one affected with the disease. It is, however, very difficult to prove the latter view, for the typhoid germ may be transported by an individual who does not contract the disease, and it is certain that this germ may preserve its vitality for a long time, at least for several years.[2]

The early and predominant involvement of the intestine in typhoid fever seems to indicate that the infection takes place primarily from the alimentary tract. Even if the typhoid bacilli should be inspired, it is not necessary to suppose that the infection is through the respiratory organs, for after reaching the mouth or throat the organisms could be swallowed.

The agency of bad cesspools, sewers, and privies in the causation of typhoid fever seems to be that here the bacilli, derived usually from typhoid dejecta, find suitable conditions for their growth, and that with imperfect drainage from these sources the surrounding soil and neighboring waters may become infected with the specific organism. This important agency enforces the necessity of measures of prophylaxis consisting of effective sewerage, provision against obstructions of house-drains, prevention of leakage from these and cesspools, care that the trapping of hand-basins, sinks, and water-closets is complete, and the disinfection and speedy removal of the excreta of patients affected with the disease.[3]

The interval between the introduction of the germs and the first manifestation of the disease, or the period of incubation, is usually about two weeks. The duration is longer than this in some instances, but probably never several months, as has been supposed. On the other hand, the duration sometimes does not exceed one or two days.[4] This disease is one of those which are very rarely experienced twice; and a person who has had the disease, as a rule, is thereafter unaffected by the special cause. As already stated, however, a relapse sometimes occurs shortly after the date of convalescence.

DIAGNOSIS.—Typhoid fever is to be discriminated from other essential fevers, especially typhus, remittent, and typho-malarial fever. The differential diagnosis cannot be fully considered without anticipating the diagnostic events which belong to the clinical history of these fevers. The discrimination is to be made by ascertaining the presence of more or less of the events which are diagnostic of typhoid fever and the absence of events diagnostic of other fevers. The points in the clinical history which are distinctive of typhoid fever are as follows: the gradual development; the absence of marked remissions; the abdominal symptoms—namely, diarrhœa with ochre-colored dejections, tympanites, tenderness and gurgling in the right iliac region; the occurrence of epistaxis; and the characteristic eruption. As regards the eruption, a fact important to be borne in mind is that rose-colored papules, like those belonging to the history of typhoid fever, are sometimes found in other affections. Other points to be taken into account are the autumnal season and the youth of the patient. The foregoing points, taken in connection with the absence of features distinctive of other fevers, generally render the diagnosis easy after a certain duration of the disease. In typho-malarial fever, as will be seen, the symptomatic phenomena of typhoid and malarial fever are commingled in variable relative proportions in different cases.

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[1] In Switzerland epidemics of typhoid fever have been reported in which the infection has been referred to eating the meat of diseased calves; but this interpretation of the cases is not free from doubt.

[2] According to bacteriological nomenclatures, the typhoid bacillus may be regarded as a saprophyte and a potential parasite; that is, all of the conditions for its development are present outside of the animal body, and it may therefore exist as a saprophyte. It occasionally makes incursions into the human body, where it becomes a parasite.

[3] A full account of the different agencies involved or supposed to be involved in the etiology of typhoid fever will be found in J. H. Hutchinson's article on this disease, in *Pepper's System of Practical Medicine, by American Authors*, Philadelphia, 1885, vol. i.

[4] Vide Murchison, in *St. Thomas's Hospital Reports*, vol. ii.

Other affections with which typhoid fever may be confounded are—cerebral and cerebro-spinal meningitis, bronchitis, pneumonitis, acute tuberculosis, and enteritis.

Acute meningitis, as distinguished from typhoid fever, is characterized by more intense cephalalgia, by intolerance of light and sounds, early and active delirium, frequently by vomiting as a prominent symptom, by rigidity of the muscles at the back of the neck, sometimes by paralyses, and by somnolency and coma succeeding the delirium. There generally is constipation. The abdomen is not tympanitic, but frequently depressed, and iliac tenderness is wanting. This differential diagnosis is required chiefly in children.

Subacute bronchitis is an element of typhoid fever. If the bronchitis be unusually prominent and the fever unusually mild, the latter may be overlooked and the disease considered a primary bronchitis. This error can only happen in the early part of the fever; and the duration and progressive increase of the disease, together with the characteristic events of the fever, will lead to a correction of the diagnosis. If the bronchitis be capillary, owing to the predominance of the pulmonary symptoms, this may be supposed to be the primary and sole disease. An instance has fallen under my observation, but capillary bronchitis as a complication is extremely rare.

Typhoid fever may be confounded with pneumonitis when the latter exists as a complication. The existence of pneumonitis is shown by its physical signs. The differential diagnosis is to be based on facts which show the existence of fever prior to the occurrence of the pneumonitis, and on the events characteristic of typhoid fever—namely, the abdominal symptoms and eruption. Cases of primary pneumonitis in which the existence of typhoid fever may be suspected are cases of so-called typhoid pneumonitis; that is, pneumonitis with phenomena denoting the typhoid state—namely, low delirium, prostration, subsultus tendinum, etc.

Acute miliary tuberculosis has not infrequently been mistaken for typhoid fever. The former is to be discriminated by the notable frequency of the respirations, the prominence of the cough, the occurrence of hæmoptysis in some cases, the abundance of subcrepitant râles; by marked lividity in some cases; by the presence of choroid tubercles; and by the absence of the abdominal and other characteristic events of typhoid fever.

Typhoid fever may be considered as primary enteritis when the abdominal symptoms are unusually prominent from the beginning. The liability to this error is chiefly in cases occurring in children. Events other than the abdominal symptoms belonging to the history of typhoid fever are to be sought after. Some of these are not so readily appreciated in children as in adults, especially those relating to the mind, and the eruption is oftener wanting. Hence typhoid fever in children is not infrequently called enteritis. With due care in tracing the development and progress of the disease this error of diagnosis should generally be avoided.

It is evident that familiarity with the phenomena and laws of typhoid fever and of the diseases with which it is liable to be confounded—namely, other fevers and the local affections just referred to—is an essential qualification for accuracy in diagnosis. This remark is of general application to diagnosis.

The thermometer may be brought to bear upon the diagnosis of this disease at an early period, before the eruption or other distinctive features are fully declared. A moderate increase of temperature daily for several successive days, with an evening exacerbation, points to typhoid fever as contrasted with typhus, periodical, relapsing, or an eruptive fever, assuming the disease to be an essential fever and not an inflammatory disease. Various circumstances, however, may occasion deviation from this gradual increment of fever in typical, uncomplicated cases.

In some cases the disease may be overlooked in consequence of the phenomena being imperfectly developed. These cases were called by Louis cases of "latent typhoid fever." They are called by German writers cases of "*typhus ambulatorius;*" and the term "walking cases" is in use in this country. The patients do not take to the bed, and perhaps they continue their usual avocations. They complain only of debility, loss of appetite, and mild diarrhœa. Under these circumstances death sometimes takes place from intestinal perforation, profuse hemorrhage, or some grave complication, and on post-mortem examination the characteristic typhoid lesions of the intestine are found. The only explanation of these cases to be given is that the effects of the special cause are concentrated on the glandular elements of the small intestine.

Errors of diagnosis are likely to be made in cases of abortive typhoid fever, in very mild cases (typhus levissimus), and in cases in which the temperature-curve has a remittent or possibly intermittent character. In children the existence of typhoid fever is doubtless often overlooked.[1] Here abdominal symptoms may be slight, particularly intestinal hemorrhages and peritonitis, while severe cerebral symptoms are usually present. In old persons the course of the disease is often irregular, pulmonary and cerebral symptoms frequently being most marked. The occurrence of cases of typhoid fever in which the symptoms of an acute Bright's disease appear early is rare, but this should be borne in mind.

PROGNOSIS.—Of 18,612 cases aggregated by Murchison, the mortality was 18.62 per cent., or 1 in 5.4. Of 303 cases received in the Massachusetts General Hospital and analyzed by James Jackson, 42 were fatal, being 1 in about 7 cases, or a fraction less than 13 per cent. Of the 73 cases which I have analyzed, 18 were fatal, nearly 1 in 4, or about 24 per cent. These facts show much variation in the death-rate in different collections of cases occurring at different places and periods. That the variation is often considerable at different seasons in the same place, and under similar circumstances as regards surroundings and treatment, is shown by the ratio of deaths in successive years in the Massachusetts General Hospital.[2] In 1828, of 22 cases, 2 died; in 1829, of 25 cases, 1 died; in 1830, of 14 cases, 4 died; in 1831, of 29 cases, 2 died; in 1832, of 23 cases, 4 died; in 1833, of 37 cases, 6 died; in 1834, of 34 cases, 6 died; and in 1835, of 35 cases, 6 died. Statistics show that the average mortality from typhoid fever in recent years is less than formerly, being about 10 per cent. This diminution in mortality may be fairly attributed to improved methods of treatment. It is, however, evident that there are wide differences in the intrinsic tendency of the disease to a fatal issue at different periods and places—a fact to be considered in estimating the influence of therapeutical measures.

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[1] Henry D. Chapin, *Am. Journ. of Obstetrics and of Diseases of Women and Children*, July, 1883.

[2] *Report on Typhoid Fever*, by James Jackson, M. D.

As regards age, Murchison's statistics show the lowest rate of mortality to be in cases between ten and fifteen years, and the next lowest between five and ten years, the number of cases analyzed at less than five years being insignificant. The highest rate at less than fifty-five years is between thirty and thirty-five years. Jackson's statistics show the death-rate to be greater above than below thirty years of age, and they show a greater mortality in the cold than in the warm months. It is a matter of common observation that vigorous persons are more likely to succumb than those of feeble constitution.

A fatal result in a considerable proportion of cases is attributable to complications or accidents, such as pneumonitis, peritonitis, and hemorrhage. Pre-existing disease may lead to a fatal result, as, for example, chronic disease of the kidneys. Unusual severity of the abdominal lesions is sometimes the cause of death. In the great majority of fatal cases the mode of dying is by asthenia. Apnœa is combined with asthenia in the cases in which sudden coma precedes death.

Coma and convulsions, probably uræmic in most cases, render the prognosis extremely unfavorable. Other marked ataxic symptoms, such as carphologia, subsultus, etc., are ominous. Cases characterized by active persistent delirium usually end fatally. The prognosis is unfavorable whenever the pulse becomes extremely frequent and feeble and the first sound of the heart is notably weakened. The immediate cause of death in these, as in all cases in which the mode of dying is by slow asthenia, is the giving way of the power of the heart's action. Great prostration is evidence of much danger. The chances of recovery may be good, notwithstanding the occurrence of pneumonitis limited to one lobe. Intestinal hemorrhage is less serious than has been supposed by some; recovery taking place in the majority of the cases in which this event occurs. Death may be caused by gangrene or bed-sores in parts exposed to continued pressure from the weight of the body. An increase of the axillary temperature above 105° denotes great danger, and a temperature of 106° or 107° or upward is a fatal prognostic. On the other hand, a considerable decrease of temperature below the minimum of health is ominous. In general, after the first few days of the disease, the temperature having daily increased up to this time, the heat as shown by the thermometer varies in the morning between 102° and 103°, and in the evening between 103° and 105°.

A favorable prognosis may be entertained so long as ataxic symptoms are not marked, the adynamia is not great, the pulse not very frequent or feeble, and there are no serious complications; but under these circumstances the prognosis should always be guarded, in view of the liability at any period of the disease to serious complications or accidents. Let it be borne in mind that sudden coma and perforation of intestine are not less liable to occur in mild than in severe cases. The liability to perforation during convalescence is to be recollected. On the other hand, recovery may be hoped for in cases in which the symptoms denote the utmost gravity.

Typhus Fever.

The fever called typhus, known from the earliest antiquity, has received a great variety of names. The name typhus, introduced by Sauvages in 1759, and now generally adopted by writers of all countries, has the negative merit of not involving any hypothesis concerning the nature and seat of the disease. Derived from τυφος, denoting stupor, it relates to a feature which is usually more or less prominent in this disease. In this country, from the fact that the disease is imported in vessels bringing emigrants from Ireland, it has been commonly known as *ship fever.*

This fever has many features in common with typhoid fever. The identity or non-identity of the two affections was once a mooted question. They present points of contrast amply sufficient to show that they are distinct diseases. In considering typhus it will suffice to present the traits by which it is distinguished from typhoid fever.

ANATOMICAL CHARACTERS.—In typhus the abdominal lesions which are characteristic of typhoid fever are wanting. The Peyerian and solitary glands of the small intestine are either unaffected or they are simply more conspicuous than usual, sometimes presenting a number of black points, giving rise to what has been called the *shaven-beard appearance.* The mesenteric glands are not infiltrated as in typhoid fever, and are generally healthy.

The disease has no known special anatomical characters; that is, there are no lesions peculiar to the disease and constantly present. Morbid appearances in different parts are frequently found after death, but they are due to complications or are such as belong to infectious diseases in general.

The spleen is usually large, soft, and of a dark, bluish-red color. In very rare instances there is no enlargement of the spleen. The heart is often flabby and softened. The blood in the heart and large vessels is unnaturally dark and fluid, and coagula, if they exist at all, are dark and soft. Hypostatic congestion of the lungs, bronchitis, lobular pneumonia, and pulmonary œdema are very frequent. Slight extravasation of blood into the arachnoid cavity occurs in a small proportion of cases. Cerebral congestion and effusion of serum into the ventricles of the brain, the subarachnoid space, and the arachnoid cavity are not uncommon, but they are usually unattended by inflammatory exudation. Meningitis is a rare complication. The same parenchymatous degenerations which are present in typhoid fever may occur in typhus. The kidneys are often congested. They are sometimes enlarged, and may present the appearances of an acute nephritis. Nephritis is more common in typhus than in typhoid fever. Subserous ecchymoses are common. The granular and hyaline changes in fibres of voluntary muscles, noticed as occurring in typhoid fever, have also been observed in typhus.

CLINICAL HISTORY.—The duration of the access is shorter than in typhoid fever, and cases of an abrupt invasion are not as rare. Patients in the great majority of cases take to the bed on the second or third day after the first manifestation of illness. The symptoms during the development of the disease are essentially the same as in typhoid fever, with this important difference—namely, the abdominal symptoms of typhoid fever, diarrhœa, meteorism, iliac tenderness, and gurgling, are wanting. Pain in the back and limbs and muscular weakness are more prominent as prodromata of typhus than of typhoid fever.

Countenance and General Aspect.—Capillary congestion of the face, extremities, and trunk is more marked in typhus than in typhoid fever. It gives to the surface, especially of the face, a dusky or dingy hue, so distinctive of typhus as compared with typhoid fever that even nurses and attendants, after becoming conversant with the two diseases, learn to discriminate them by the physiognomy. The conjunctiva is more frequently and more deeply congested. A besotted or expressionless countenance is earlier and more strongly marked.

Nervous System.—The symptoms referable to the nervous system which belong to the clinical history of typhoid fever occur in typhus. Coma-vigil and typho-mania are observed in a larger proportion of cases of typhus, they are oftener present in a marked degree, and they occur earlier in the career of the disease. Other ataxic symptoms, such as subsultus and carphologia, are of more frequent occurrence. Prostration is generally more marked and is earlier manifested. Persistent active delirium, in my experience, has occurred oftener in typhoid than in typhus fever. Contraction of the pupil often accompanies delirium or stupor, and is sometimes extremely marked, called by Graves the *pinhole* pupil. I have observed oscillations of the eyeballs occurring in a marked degree, the movements being lateral and notably rapid. The oscillations occurred whenever the patient was raised. The cases ended fatally. Coma and convulsions occurring in typhus as in typhoid fever are generally attributable to uræmia. Hyperæsthesia of the surface, even when the mental perception is notably blunted, is sometimes manifested.

Digestive System.—The tongue, more frequently than in typhoid fever, becomes covered with a thick brown or black coating. It is less frequently reddened, glazed, and fissured. It is oftener with difficulty protruded. Sordes occur more frequently, earlier, and in greater abundance. As regards the abdominal symptoms which are highly diagnostic of typhoid fever, the two diseases present a striking contrast. Diarrhœa very rarely occurs in typhus, and if present it is almost invariably slight. The stools have not the ochre color of those in typhoid fever. Tympanites is much oftener wanting, and generally when present it is slight in typhus. This statement applies also to iliac tenderness. Intestinal perforation does not occur in typhus. Hemorrhage from the bowels is one of the rarest of events, exclusive of cases in which dysentery is a complication.

Parotiditis is liable to occur, and the remarks made with reference to this complication in typhoid fever are equally applicable to its occurrence in typhus.

Skin.—A characteristic eruption occurs in a larger proportion of cases of typhus than of typhoid fever. Murchison states that of 3506 cases received at the London Fever Hospital, it was not observed in 403, or a fraction more than 11 per cent. Of 65 cases which I have analyzed, it existed in 57. It is oftener wanting in children than in adults. The eruption appears earlier in typhus. The average period from the time of taking to the bed to the first appearance of the eruption is a little less than three days. The eruption in typhus is much oftener abundant, frequently being copious over the extremities as well as on the trunk, and sometimes appearing on the face.

Other distinctive points relating to the eruption are as follows: It is a maculated, not a papular, eruption. The spots become of a dull, dingy or dark-red color, and after the first two or three days the redness cannot readily be made to disappear by pressure. In some cases in the latter part of the disease the spots become truly petechial. They are smaller than the papules of typhoid fever. They do not come and go like the rose papules of typhoid; they all appear in the course of three or four days, and remain during the greater part or the whole course of the disease, sometimes even continuing into convalescence. They are often apparent after death.

The copiousness of the typhus eruption represents generally a corresponding gravity of disease. This statement does not hold good with respect to the eruption in typhoid fever.

In a certain proportion of cases of typhus intermingled with the characteristic maculæ are more or less of the rose papules which are characteristic of typhoid fever.

Petechiæ are observed in some cases of typhus, as in cases of typhoid fever, as well as in various other diseases. As already stated, the characteristic typhus-spots sometimes become petechial. The eruption of typhus is sometimes called a petechial eruption. This is incorrect. *Petechiæ* are spots caused by minute extravasations of blood; that is, they are ecchymoses, and these are not distinctive of typhus. Vibices are occasionally observed in cases of typhus. Sudamina or miliary vesicles occur in cases of typhus as well as of typhoid fever.

The statements with respect to sweating and moisture of the skin in typhoid are also applicable to typhus fever.

The odor from typhus patients is stated to be characteristic. I have met with persons who declared that they were able to discriminate cases of typhus from typhoid fever by the sense of smell. I can bear testimony to an offensive odor emanating from patients affected with either disease, but I have not been able to appreciate its value as a diagnostic symptom.

Erysipelas sometimes occurs in typhus as in typhoid fever. Bed-sores are less liable to occur, in consequence of the shorter duration of typhus. Spontaneous gangrene, not produced by pressure, is an occasional event. I have seen a case in which gangrene of both feet took place. The nose, penis, and scrotum have been known to slough away. Ulceration or sloughing of the cornea has been observed. The affection known as *noma* is another serious complication occurring sometimes, not only in children, but in adults. These complications occur in hospitals among patients who prior to the disease were suffering from innutrition.

Respiratory System.—The account of symptoms referable to the respiratory system in typhoid fever is mainly applicable to typhus. The significance of a spasmodic inspiration occurring without any pulmonary complication is the same in the latter as in the former disease. Cough from bronchitis is generally more prominent than in typhoid fever. Pseudo-pneumonitis—that is, hypostatic congestion and œdema—is more frequent, whereas true pneumonitis appears to be less frequent than in typhoid fever. Epistaxis occurs less frequently. It appears to be established that the expired breath contains more ammonia than in health.

Circulation and Temperature.—The average frequency of the pulse in cases of typhus is greater than in typhoid fever. The significance of frequency and other characters of the pulse, and of weakness or absence of the first sound of the heart over the apex, as representing the state of the vital forces, is not less in typhus than in typhoid fever.

The temperature of the body, as determined by the thermometer, is more or less increased, varying between 102° and 107° F. The increase of temperature corresponds to the intensity of the disease. A decline of temperature is coincident with the occurrence of convalescence. The defervescence in some cases is notably rapid, but in other cases it is gradual. At the time of convalescence the temperature frequently falls below the standard of health. As in typhoid fever and in other essential fevers, a sudden and considerable rise of temperature renders probable some inflammatory complication; and on the other hand a sudden fall of temperature below the average range in health denotes a serious change for the worse. As a rule, the increase of temperature daily is progressive from the beginning of the disease to about the fifth day, and the daily oscillations of the morning and evening temperature are not observed prior to this period. An analysis by Dr. Maclagan of 58 cases ending in recovery gave, as the average temperature in the morning, a fraction over 102°, and in the evening a fraction over 103° F. The highest point reached in any of these cases was a fraction over 105°. In typhus, as in other fevers, a fall of temperature not infrequently denotes approaching convalescence, prior to any appreciable improvement in the pulse and other symptoms.

Urine.—The urea and uric acid are generally increased, as in cases of typhoid fever. According to Murchison, the chlorides are greatly diminished or disappear entirely.

Albuminuria is of more frequent occurrence in typhus than in typhoid fever. Murchison found it in 20 out of 28 cases, or a fraction more than 71 per cent. In about one-half of the cases the amount of albumen was considerable. In most of the latter cases there was no evidence of pre-existing renal disease. In typhus as in typhoid fever the early appearance of albumen in the urine, its abundance, and its duration, denote gravity of disease. Epithelial and blood-casts are sometimes observed.

The duration of typhus is less than that of typhoid fever. Of 53 uncomplicated cases ending in recovery analyzed by Murchison, the duration varied between eight and twenty days, the mean duration being a fraction more than fourteen days. The mean duration in 10 fatal cases was fifteen days. Of 45 cases ending in recovery which I have analyzed, the maximum duration was twenty-six and the minimum nine days, the mean being fourteen days. The mean duration in 9 fatal cases was ten days. In my cases the beginning of the disease was reckoned from the time of taking to the bed.

Relapses of typhus are extremely rare. Examples of this fever having been experienced more than once are also very infrequent. There are no special sequels, and in the great majority of cases the health is excellent after recovery. Temporary loss of the hair is a common consequence of both typhoid and typhus fever. Mental imbecility sometimes remains for a certain period after the general condition of the body denotes recovery.

Typhus fever is sometimes associated with dysentery. Typhus and variola have been known to be combined. Probably typhus and typhoid fever may coexist.

CAUSATION.—The contagiousness of typhus is shown by the occurrence of successive cases in particular houses and neighborhoods, by the number who are attacked of persons brought into contact with typhus patients, especially in hospitals, either as physicians, attendants, or fellow-patients, and by the importation of the disease in localities where it did not previously exist. Referring the reader to works on fever for the further evidence of the contagiousness of typhus, I shall subjoin some illustrative facts falling within my own knowledge:

In 1850, 1851, and 1852, I was engaged in the clinical study of typhus and typhoid fever in the Buffalo Hospital of the Sisters of Charity. I recorded during these years 65 cases of typhus. Twelve Sisters of Charity were assigned to this hospital during the period just named. Of these twelve sisters, five contracted typhus fever. These five sisters alone had charge of the fever patients; and the remaining seven sisters, performing other duties, were not brought into contact with the fever patients. Thus, every sister who nursed fever cases had an attack of the disease. During the last of these years eleven patients, admitted for other affections, contracted the disease, the number of cases of typhus received during this year being considerably larger than during the preceding two years.

Typhus fever began to prevail in certain parts of the city of New York as an epidemic in 1861, the disease having been imported from Ireland. From Jan. 1, 1861, to Nov. 1, 1864, 1428 cases were admitted into Bellevue Hospital, more than 500 cases having been received during the six months preceding Nov. 1, 1864. Of 22 members of the house staff during the time above stated, 15 contracted the fever. Of persons employed in the hospitals as orderlies, watchers, etc., 16 were attacked with fever. From June, 1863, to June, 1864, 26 patients, admitted into the hospital for various affections, contracted the fever. In May, 1864, the fever patients were transferred to tents, and no new cases were received. There was subsequently no development of the disease in the hospital.

The infectious poison emanating from typhus patients rarely communicates the disease except to those who are in immediate proximity to patients. Concentration of the poison is generally necessary. Hence, the disease is likely to be contracted by those who render personal attentions to patients, and especially by those who are exposed to the atmosphere of ill-ventilated fever-wards containing a large number of cases. A single patient in a spacious, well-ventilated apartment seldom communicates the disease. All persons at all times, moreover, are not equally susceptible, and hence some contract the disease after comparatively slight exposure, while others who are much exposed escape. There is no proof that the disease is diffused from one house to another or from hospitals to adjoining houses, except by intercommunication. Facts show that the disease may be communicated by fomites, but a considerable impregnation of articles of clothing, etc. is necessary. Exposure of infected clothing to a dry heat of 200° F. probably destroys the miasm. Some facts appear to show that the disease may be contracted by dissecting bodies dead with typhus, but this is not certain. The period of the disease in which it is most likely to be communicated is after the first week. Facts appear to show that it may be communicated during convalescence.

The fact that in the emanations from the bodies of patients a contagium exists capable of producing the disease is distinctive of typhus as contrasted with typhoid fever. There is no reason for supposing that the infecting capability of the typhus germs depends on any changes taking place after they are thrown off from the body.

There is considerable variation in the estimation by different observers of the period of incubation. Probably this period varies considerably in different cases. Murchison has determined the average period to be about twelve days. Exceptionally it is longer than this, but it rarely if ever exceeds three weeks. On the other hand, in many cases it is less than twelve days, and occasionally the symptoms begin almost at the instant of exposure.[1] Persons have sometimes had an impression that they received the poison at a particular moment when they were conscious of a peculiar sickening odor inhaled from the body of a typhus patient. Little or no reliance is to be placed on this impression as evidence of the reception of the infection. Probably physicians have very many times experienced this sickening odor without having had typhus. Such has been repeatedly my experience.

As a rule, in this country typhus fever is an imported disease and is diffused exclusively by contagion. It is, however, difficult to trace all outbreaks of the disease to contagion, and hence the opinion is held by some that the special cause is sometimes produced outside of the body. The disease sometimes appears to be developed as a consequence of overcrowding and deficient ventilation; in other words, the concentrated emanations from the bodies of healthy persons apparently suffice for the generation of typhus germs.[2] Outbreaks in jails, hospitals, workhouses, ships, and unventilated tenement-houses crammed with occupants are thus accounted for in some instances irrespective of contagion.

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[1] Vide *St. Thomas's Hospital Reports*, vol. ii.

[2] For facts bearing on this point, vide Murchison, *op. cit.*

In 1841, I reported four cases of typhus developed in the Erie county almshouse. They occurred during the winter months. The wards in this institution were quite small and crowded, and for the sake of warmth fireplaces, which had previously been used, had been bricked up and close stoves substituted. There was an abundant eruption in all these cases, and an examination after death in the only case which proved fatal showed the absence of the typhoid lesions. In these cases overcrowding and want of ventilation seemed to be important elements in causation.[1] No cases of typhus occurred out of the almshouse, and the disease was not imported.

Overcrowding, deficient ventilation, especially if conjoined with innutrition, are powerful auxiliary causes acting in conjunction with contagion. The causative influence of destitution and starvation is strikingly shown by the prevalence of typhus in Ireland in periods of famine, and by the large proportion of cases among those received in hospitals in which the deprivations and hardships of poverty had been experienced prior to the attack of fever.

Typhus attacks the two sexes in about an equal proportion. Persons are more liable to be attacked during adult age, but children are not exempt, and this fever does not, like typhoid, spare those who are beyond the middle period of life. It is more likely to prevail in the winter and spring months than in autumn, in this respect differing from typhoid fever.

DIAGNOSIS.—The remarks on the discrimination of typhoid fever from remittent fever and several local affections will apply to typhus fever. (Vide p. 966.) The discrimination of typhoid and typhus fever is to be here noticed. The following are the more important points in this differential diagnosis: The relatively long duration of the forming stage in typhoid, and its short duration in typhus; the prominence of pain in the loins and limbs and muscular prostration at an early stage of typhus; absence, in typhus, of the characteristic abdominal symptoms of typhoid—namely, diarrhœa, meteorism, iliac tenderness, and gurgling—or their presence in comparatively a slight degree; the dusky or dingy hue of the surface in typhus; the appearance of the eruption earlier in typhus, and the different characters of the eruption—namely, in typhoid, papular, rose-colored, the redness disappearing on pressure; in typhus, after two or three days, if not at first, maculated, the color dark red, and the redness not disappearing on pressure. Other distinctive points relate to the eruption—namely, the sparseness in typhoid and the copiousness in typhus; the frequent extension in the latter of the eruption over the extremities; the persistence of the spots in typhus, and the disappearance of papules with the production of fresh papules in typhoid. It is to be borne in mind that in some cases of typhus rose papules are intermingled with the characteristic maculæ. In typhoid fever the temperature reaches its acme at a later period than in typhus. The rise of temperature is more gradual and regular in typhoid than in typhus. In typhus the average duration of the fever is shorter than in typhoid, and the defervescence is more rapid.

Corroborative diagnostic points are—the occurrence of peritonitis and hemorrhage from the bowels in some cases of typhoid, and the earlier occurrence of delirium, coma-vigil, and other ataxic symptoms in typhus.

If the eruption be present, typhus is generally recognized without difficulty. The eruption is not always present, and the diagnosis may then involve delay and difficulty. The prevalence of typhus and exposure to contagion are to be taken into account in doubtful cases.

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[1] Vide *Boston Med. and Surg. Journal*, June, 1841.

Prognosis.—Of 18,592 cases aggregated by Murchison, occurring at the London Fever Hospital in fourteen years, at King's College Hospital, the Edinburgh Infirmary, and at Glasgow, Scotland, the mortality was 18.78 per cent., or 1 in 5.27. This rate of mortality differs only by a small fraction from the rate in about the same number of cases of typhoid fever analyzed by Murchison. The death-rate is found to vary at different times and places between 9 and 25 per cent., the variations being due to differences as regards an intrinsic tendency of the disease to a fatal result. In epidemics of typhus, as of other diseases, it has been often observed that the fatality is greatest at first and decreases as the number of cases diminish. The ratio of fatality is greater among males than females. The fatality is less in early life than subsequently. It is greater at less than ten years of age than between ten and twenty years. After thirty years of age the fatality progressively increases, and after fifty years the proportion of deaths is nearly one-half.

Of the 63 cases which I have analyzed, 12 were fatal; that is, 1 in a fraction more than 5, or a fraction more than 18 per cent.

A fatal result is oftener due to the intensity of the fever—that is, occurring irrespective of complications—in typhus than in typhoid fever. Peritonitis, which destroys a certain proportion of patients with typhoid fever, does not occur in typhus. This is true also with respect to intestinal hemorrhage. Pneumonitis is less frequent in typhus, and there are no complications peculiar to the latter. Acute diffuse nephritis attended by uræmic symptoms is more liable to occur in typhus than in typhoid fever. A fatal result appears to be oftener attributable to feebleness of constitution, or to causes which impair the power of resisting the disease, in typhus than in typhoid fever. Of 38 cases which occurred among the members of the resident medical staff and persons employed in Bellevue Hospital, 17 ended fatally, whereas of 1428 patients admitted with fever during the same period, the number of deaths was 243, or 1 in 5.87. This difference in the mortality in the two classes of cases is the more remarkable because the resident physicians and persons employed in the hospital were treated for the disease under more favorable circumstances than the hospital patients. It is difficult to account for the greater fatality among the former class, except by supposing that living in the hospital involved exposure to nosocomial depressing influences which rendered the system less able to resist the disease.

In general, the symptoms pointing on the one hand to a favorable, and on the other hand to an unfavorable, prognosis are the same in both typhoid and typhus fever.

CHAPTER III.

CONTINUED FEVERS (Continued).

Treatment of Typhus and Typhoid Fevers.—Relapsing Fever; Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Erysipelatous Fever.—Epidemic Fever characterised by Mild Erythematic Pharyngitis.

THE general principles of treatment in typhoid and typhus fever are essentially similar, and indeed are applicable to all the fevers. It will suffice to notice incidentally indications pertaining exclusively either to typhus or typhoid fever, without considering the treatment of each fever separately.

It must be admitted that the known resources of therapeutics do not afford reliable means for the arrest of these fevers, nor even for shortening the duration of the febrile career. Quinia, so potential in arresting malarial fevers, has no abortive potency in the continued fevers. Calomel in doses of from ten to fifteen grains repeated daily for several days has been recommended by Wunderlich, Liebermeister, and other German writers as shortening the course of typhoid fever, diminishing the mortality, and sometimes causing the disease to abort. Iodine or the iodide of potassium and carbolic acid have been thought to have a special influence over this fever. Liebermeister states that the iodide of potassium in doses of a scruple to a drachm daily, given in more than two hundred cases, while it produced no appreciable effect upon any of the febrile phenomena, apparently diminished the rate of mortality. That the mineral acids—namely, the dilute phosphoric, hydrochloric, nitric, and nitro-hydrochloric—have a favorable modifying influence upon the disease there is abundant testimony. In 1863 and 1864, I instituted a comparison in the male fever-wards at Bellevue Hospital of the rate of mortality during six months without, and the same period with, the use of dilute sulphuric acid, eliminating all cases in which death took place within forty-eight hours after admission. Of 70 cases treated from January 1 to July 1, 1863, without acid, the death-rate was 20 per cent.; of 78 cases treated with acid from July 1, 1863, to January 1, 1864, the death-rate was 10.25 per cent. The treatment in the two collections of cases was the same, exclusive of the acid.[1] My observations do not enable me to state which one of the mineral acids is to be preferred. They are generally relished by fever patients if sufficiently diluted, with the addition of syrup of orange-peel or simple syrup, and they may be given, not as a medicine, but as a drink. The death-rate, however, in the cases treated with the acid was not below that in 307 cases treated by Dr. Cotting of Boston without drugs of any kind. The rate in these cases was 10 per cent.

Of all therapeutic measures, those directed toward the reduction of the fever take the first rank. The *modus operandi* of antipyretic measures is not thoroughly understood, but there is no doubt that their efficacy consists in something more than the mere reduction of the temperature, and that indications for their employment are to be sought not only in the height of the temperature, but especially in symptoms denoting severity of infection, such as stupor and other ataxic disturbances.

Reduction of the intensity of the fever may be accomplished either by the application of cold or by the administration of antipyretic drugs. Currie a century ago pointed out the efficacy in fever of water employed as a cold affusion. The modern revival of the use of cold water for an antipyretic effect in typhoid fever is attributable to a publication by E. Brand in 1861. Bartels, Jürgensen, and Liebermeister are to be named as especially prominent in bringing to the test of clinical experience antipyretic measures of treatment. There is no doubt that a remarkable reduction in the death-rate of typhoid fever has been brought about by systematic antipyretic treatment.

The methods of refrigeration by cold water now in vogue are the bath, sponging the body, and the wet sheet with sprinkling. The bath is the method chiefly employed by German physicians. The following method of administering the bath may be recommended: The bath-tub should be drawn up beside the bed. The patient should be lifted and placed in the bath by attendants without any exertion on his part and with as little disturbance as possible. The temperature of the bath should be from 75° to 80° F., and the patient should be removed after ten minutes from the bath and dried. It is not necessary to use baths at a temperature much if any below 75°, but it may be advantageous to immerse the patient in water of a temperature of about 90°, and then gradually cool the water down to about 75°. It is important to know that the rectal temperature sinks for half an hour after removal from the bath. If the patient feels very cold after the bath and there are any symptoms of collapse, heat should be applied and stimulants administered. Generally in from two to three hours the temperature has risen, sometimes to its former height. It is generally sufficient to administer three to five baths during the day, and it is rarely desirable to give them at night. It is usually stated that a temperature of 103° or upward is an indication for the bath, but, as has already been mentioned, other symptoms than the temperature are also to be considered. When it is apparent that notwithstanding the temperature named the patient's general condition is good and is not suffering from the pyrexia, there is no necessity of the bath; on the other hand, when there are stupor and other symptoms to indicate that the system is being overpowered by the severity of the infection, then it may be desirable to employ the bath even at a temperature lower than 103°. Contraindications to the use of the bath are intestinal hemorrhage, perforation of the bowel, and great weakness of the heart's action.

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[1] Vide report by Dr. Irving W. Lyon, then one of the house-physicians of Bellevue Hospital, in *American Medical Times*, Feb., 1864.

Although the bath is the most effectual mode of reducing the temperature, the method is open to objections on the score of inconvenience, especially in private practice, and of the excitement of the patient which it is apt to occasion. Considerable judgment is required in its employment to avoid unpleasant depressing influences. For these reasons other measures are more commonly employed in this country. Sponging is the simplest method, involving the least inconvenience to the patient and others. It should be first employed, and in a large proportion of cases it will suffice. Sponging either the whole body at once or different parts in succession with water at a temperature not uncomfortable to the patient for as long a period as it is unattended by discomfort, repeating the measure as often as indicated by a rise of temperature, will generally prove successful. If not conducted by a skilled nurse, it is difficult often to have this method properly carried out—in part, because persons cannot appreciate that so simple a measure can have much potency, and partly from the popular fallacy that there is danger of "taking cold." The sponging should be resorted to when the axillary temperature is 103° and that of the rectum 102½°. It should be continued until the temperature in the rectum falls to 100° or 101°, and repeated whenever it again rises. A little wine or spirits should be given if the patient manifest chilliness.

If this method prove ineffective, the wet sheet and sprinkling may be employed. The patient is placed on a cot, wrapped in a wet sheet without any other covering, and the body sprinkled with cold water at short intervals. For sprinkling a common watering-pot is suitable. This may be continued for half an hour or even longer, according to the effect on the temperature and other symptoms. The thermometer must be introduced into the rectum. This method is to be repeated as often as the temperature rises. The temperature will be found to fall after the patient has been transferred from the cot to the bed. A cot with arrangements for the drainage of the water is convenient. A sense of chilliness, feebleness of the pulse, and cyanosis contra-indicate the continuance of the sprinkling and are indications for wine or spirit.

We possess a considerable list of drugs which have antipyretic effects. The most important of these are quinia, salicylic acid, kairin, resorcin, hydrochinon, antipyrine, and thallin. Of antipyretic drugs, quinia, administered in doses of 20 to 40 grains before the evening exacerbation, until recently held the first rank. Antipyrine, however, is at present the favorite drug for reducing temperature. This remedy has many advantages, and is attended with but few secondary disagreeable effects. The taste is slightly bitter, but not particularly unpleasant. It is readily soluble in water, and may be administered either in powder or in solution. The method of administration recommended by Filehne, who introduced the drug into medicine, is to give at first 30 grains, followed in an hour by another 30 grains, and at the end of another hour by 15 grains. Striking reduction of temperature is obtained by the administration of smaller doses, which are less likely to be attended with any unpleasant effects. The fall in temperature is considerable and rapid. The heart's action is usually at the same time increased in force and decreased in frequency, and the mental state of the patient is improved. In a few instances symptoms of collapse or of notable prostration have followed the administration of antipyrine. The unpleasant secondary effects which have been noted in some cases are vomiting, profuse perspiration, and an erythematous eruption. Should the drug excite vomiting, it may be given hypodermically or preferably by the rectum. The eruption often resembles somewhat that of measles. It disappears, as a rule, soon after the cessation of the drug. The other antipyretic drugs which have been mentioned are less commonly used, and are accompanied by more or less disagreeable secondary effects.

Good results have been obtained by the use of veratrum viride, digitalis, salicin, and salicylate of sodium, but these drugs are to be given circumspectly and are not very commonly employed.

Valuable as are the antipyretic remedies in typhoid fever, there is no proof that they exert any specific effect upon the special poison of the disease or that they shorten the duration of the disease. The injurious effects of pyrexia *per se* have undoubtedly been much exaggerated. Some late writers have even revived the old notion that a certain amount of increase of heat in the infectious diseases has a useful purpose. Probably too much weight has been assigned to the mere abstraction of excessive heat in the treatment of fevers. The beneficial effects of antipyretic treatment are manifested even more in the removal of other symptoms, such as somnolence, delirium, dryness of the tongue, weakened heart's action, and adynamia; and when such symptoms are not present it is not necessary to resort to vigorous measures to reduce temperature.

Indications for treatment relate to particular symptoms or events and complications embraced in the clinical history of these fevers.

In the early part of the disease cephalalgia claims palliative measures. This symptom is relieved by ice-water, spirit and water, distilled vinegar, or cologne-water freely applied to the head, and if more efficient measures be required the cold douche or ice-cap may be employed. As soon as the existence of either typhus or typhoid fever is declared the hair should be closely cut. This renders the patient more comfortable and facilitates local applications.

Insomnia is a symptom calling for treatment. To procure sleep is desirable, not only for immediate comfort, but as a means of averting ataxic symptoms. An opiate often proves beneficial. It may procure refreshing sleep in place of pseudo-somnolence or coma-vigil. The latter condition, not denoting a tendency to true coma, does not contraindicate the trial of opium. Other remedies which may be prescribed are hyoscyamus and the bromide of potassium, either separately or in combination.

Delirum, if slight or moderate, does not call for remedies; but if prominent as a symptom, as shown by constant talking and attempts to get out of bed, it leads to other ataxic symptoms and to exhaustion. Opium in some form, and other anodynes, are indicated. If these fail, the combination of antimony in small doses with opium, as recommended by Graves, will often prove efficacious. An eighth or a sixteenth of a grain of the tartrate of antimony and potassa may be given hourly or half-hourly until the patient becomes quiet, unless nausea or vomiting occur, when it is to be at once suspended. The remedies given for insomnia in addition to opium are appropriate. The tincture of digitalis in half-drachm doses sometimes has a happy sedative effect upon the nervous system. The wet pack—that is, the body wrapped in a wet sheet and covered with a blanket—is a soothing measure which may often be resorted to with advantage. It is also an antipyretic measure, although less effective than continued sponging or the wet sheet with sprinkling. Insomnia, delirium, and other symptoms denoting disorder of the nervous system are less marked in proportion as the antipyretic treatment is effective. This statement is probably applicable in general to the complications and serious events liable to occur during the course of the disease, including perhaps perforation of the intestine and intestinal hemorrhage.

Nausea and vomiting seldom occur spontaneously in either typhoid or typhus fever to an extent to call for anything more than regulation of the ingesta. Diarrhœa in typhoid fever may require to be restrained, and for this end opiates and astringents, given by the mouth or rectum, may be employed. Mere looseness of the evacuations without undue frequency does not call for treatment. On the other hand, in typhus and sometimes in typhoid fever constipation may furnish an indication for treatment. As a rule, cathartics, or even laxative remedies, are not required for constipation. Simple enemata suffice and are to be preferred. Evacuations may be delayed for two or three days, or even longer, without injury if there be no evidence of discomfort and if the abdomen be not distended. Tympanites, if great or considerable, occasions discomfort and embarrasses respiration by interference with the descent of the diaphragm. Large enemata may suffice to relieve this symptom. Turpentine should enter into the enemata. If these fail a saline laxative may be given. A stomach-tube introduced into the rectum and carried as far as practicable sometimes occasions the discharge of gas in considerable quantity. Turpentine given by the mouth is supposed to relieve this symptom. Turpentine stupes to the abdomen are useful. Charcoal given freely is sometimes effective. Mechanical compression by means of a band applied over the abdomen sometimes affords relief.

Two important events connected with the abdominal lesions are liable to occur in cases of typhoid fever—namely, perforation of the intestine followed by peritonitis, and intestinal hemorrhage. The probability of the successful treatment of the former is almost *nil*. The small chance of success to be hoped for depends on the free use of opium and measures to support the powers of life. Local depletion and blisters are not admissible. Peritonitis not dependent on perforation claims the same treatment and is not so hopeless. Intestinal hemorrhage if profuse involves danger. This event calls for hæmostatic remedies. The peristaltic movements should be arrested by opium. Ergotin may be given hypodermically. The acetate of lead, given by the mouth, is preferable to tannic acid or the astringent preparations of iron, as less likely to occasion vomiting. Cold applications should be made to the abdomen. Absolute quietude is to be enforced.

Cough, although generally existing, is seldom sufficiently troublesome to require palliation. Pneumonia should not be treated with local or general depletion, blisters, or mercury, or with nauseant sedatives. This, as well as other serious complications, contraindicates depressing measures. The position of the patient should be occasionally changed to prevent hypostatic congestion of the lungs. Epistaxis is sometimes so profuse or persisting as to indicate local astringent remedies, and sometimes even plugging of the anterior and posterior nares.

Coma is to be treated by sinapisms or blisters to the back of the neck, and if dependent on uræmia by saline hydragogues if they be not contraindicated by the abdominal symptoms. If contraindicated reliance must be had on measures to produce copious perspiration. For the latter object the pilocarpine, administered hypodermically, may prove efficient. Strong coffee is useful as a palliative of undue somnolency or semi-coma apparently due to nervous exhaustion.

It is important to prevent accumulation of urine from retention, and to resort to the timely use of the catheter if required.

Bed-sores are to be guarded against by having the position of the patient often changed and by attention to cleanliness. If they occur, the ulcerated parts are to be relieved from pressure by air-cushions and treated with appropriate local applications.

Hygienic and supporting measures constitute a most important part of the treatment of typhoid and typhus fever. Hygienic measures indicated are—complete ventilation, cleanliness, regulation of temperature (which should be about 60° F.), changing frequently the position of the body, and the varied attentions involved in good nursing.

Foremost among the hygienic conditions for passing safely through the disease is an abundance of pure air. In private practice the sick-room should be large and ventilated as completely as possible. In hospitals, fever-wards, in addition to ample provisions for ventilation, should not be crowded; 1500 cubic feet of air should be allowed to each bed. The evacuations should invariably be instantly removed. When practicable it is advisable that the bed- and body-linen should be changed daily.

The importance of fresh air is shown by the reduced rate of mortality when patients are treated in sheds and tents, as compared with the death-rate in even well-ventilated hospital wards. Striking evidence of this was afforded by the transfer of fever cases (chiefly typhus) from Bellevue Hospital to tents on Blackwell's Island. A comparison of the mortality in 520 cases treated in tents from May 17 to November 1, 1864, with the average mortality in the Bellevue Hospital wards, shows a marked difference. The average death-rate in the hospital wards was 1 in 5.97; in the tents, 1 in 16.77, excluding the cases in which death took place within forty-eight hours after admission. Making the fullest allowance for the conjecture that the cases in the tents were milder than those in the hospital wards, it can hardly be doubted that the superior ventilation in the tents was the means of saving many lives.

In a paper communicated to the New York Academy of Medicine in June, 1853, by Dr. John H. Griscom, an account is given of 82 cases of typhus transferred from an emigrant ship at Perth Amboy, N. J., to wooden shanties with sail "roofs." The ship brought over between three and four hundred passengers, and a number had died on the passage. There being no hospital or other accommodations in the town, two shanties were erected, "thirty feet long and twenty feet wide, boarded on three sides about four feet up, and over them old sails were stretched." 82 fever patients were transferred to these shanties, of whom 12 were in a state of insensibility when removed. On the night after their removal there was a violent thunder-gust accompanied by torrents of rain, and the following morning the clothes of all were saturated with water. The medical treatment consisted "of an occasional laxative or enema; vegetable acids and bitters were liberally administered, together with the free use of cold water, buttermilk, and animal broths." 4 sailors who sickened after the arrival of the vessel were removed to an ordinary dwelling-house, and of these cases 2 proved fatal. Of the 82 cases treated in the shanties, not one proved fatal.[1]

Fever patients should not only be allowed to take water as freely as they may desire, but it should be systematically given when from the blunted state of the perception they manifest no desire for it. There is reason to think that death is sometimes attributable to an insufficient supply of liquid. The importance of this should not be overlooked by the physician, inasmuch as it is a popular notion that the free use of water in fevers is hurtful. Iced water is not objectionable. It may be acidulated with lemon- or orange-juice if rendered thereby more agreeable to the patient.

The importance of support is based on the plain fact that, typhus and typhoid fever being self-limited diseases, if the patient can be kept alive, after three, four, or more weeks recovery will take place, provided there be no serious complication. In a case of severe uncomplicated fever the patient is in a situation not unlike that of a person in danger of drowning not far from or perhaps very near the shore. If he drown, it is because his strength gives way before the shore is reached. As a person in this situation requires only to be buoyed up by some support, so the fever patient in a similar emergency may only need supporting measures to live until the disease ends. Death may take place at a juncture when, could it have been averted a few days, or perchance even a single day, the period of danger might have passed.

Supporting measures embrace tonic remedies, alcoholics, and alimentation, especially the two latter. Of tonic remedies, the most efficient and convenient of administration is quinia, which may be given in doses of two or three grains twice or thrice daily.

Alimentation ranks first in importance. It is desirable for the patient to take as much nutritious food as will be digested and assimilated. Innutrition in cases of fever, as in other affections, enhances and adds to the morbid phenomena belonging to the disease. Graves uttered an important truth in saying that a great object of treatment is to prevent patients from dying from starvation. In the early part of the disease there is generally a repugnance to food, and afterward, owing to the mental state and the condition of the mouth, taste and appetite are wanting. Under the latter circumstances food is to be given, although not desired by the patient, and even when there is a disinclination to take it. The indifference to food may proceed, measurably, from an unwillingness to be disturbed and a reluctance to make any exertion.

The supporting diet should be in a liquid form, embracing the necessary variety of alimentary principles. The animal broths, eggs, and milk, with the addition of some farinaceous preparation of food, fulfil these requirements. Different articles should be given in alternation and at regular intervals. The intervals should be between two and four hours long. It is injudicious to give food every half hour or hour, or after even shorter intervals, as is not infrequently done. The amount of food given at a time must vary according to circumstances, the object being to give as much as can be taken without risk of either vomiting, discomfort from over-accumulation in the stomach, or indigestion. If the patient be in a condition to feel and express a choice for particular kinds of food, the preference should as far as practicable be consulted. The physician should have an oversight of the preparation of food, lest through ignorance or carelessness it be unpalatable, innutritious, or improperly served. Precise directions and attention to the details of dietetics form an important part of the physician's duty, not only in fevers, but in all cases of disease in which supporting treatment is indicated. Of the different articles of food just named, milk is by far the most valuable as entering into the fever diet. It is taken, if not with relish, at least with less reluctance than other articles, and it has the great advantage of embracing in proper combination all the alimentary principles required for nutrition. The "beef-tea," as it is often prepared, is little more than warm water flavored with the meat. The different so-called meat extracts contain little or no nutriment. It has been considered, indeed, that they are injurious from the amount of excrementitious principles entering into their composition. Prepared in any mode and as carefully as possible, the essence of meat and broths are vastly inferior in nutritive value to milk. They should, therefore, never be relied upon to the exclusion of the latter. If the same article of animal diet—for example, beef-broth—be given day after day, the patient acquires for it after a time an intolerable disgust. The form should therefore be varied, beef-, mutton-, and chicken-broth being given in alternation. They may be rendered more nutritious by the addition of barley or some other farinaceous article, and by adding the yolk or albumen of eggs. It is desirable that from one to two quarts of milk *per diem* should be ingested; but since the value of a milk diet in fevers has come to be appreciated its use has frequently been pushed to an injurious excess. I have met with instances in which an abundance of hard curds was passed from the bowels after much abdominal distress and general disturbance. Coffee or tea, if acceptable to patients, is allowable. It may be made nutritious by a large addition of milk.

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[1] *Transactions of the New York Academy of Medicine*, 1853, vol. i. part ii.

Alcoholics have entered largely into the treatment of fevers in this country during the last thirty years. That they have been used too freely and indiscriminately can hardly be doubted. As a natural consequence, there is perhaps at the present moment a tendency to undervalue their importance. It does not follow that they are never useful because in mild cases or in those treated under most favorable hygienic conditions the rate of mortality is small although they were not used. Observation of their immediate effects in certain cases shows their utility, often in a very striking manner. Indiscriminately and excessively used, they are not useful; but used with proper application and moderation, they form an essential part of the supporting treatment of fevers, as well as of all other diseases which destroy life by asthenia.

The supporting effect of alcoholics is directed chiefly to the circulation. Hence the indications respecting their use are derived especially from symptoms relating to the heart. Feebleness of the circulation, as denoted by the pulse and the heart-sounds, calls for their use, and they are to be given, as regards quantity, in proportion to the degree of feebleness of the circulation and according to their effect. The characters of the pulse denoting feebleness of the heart's action are smallness and compressibility, frequently combined with considerable or great frequency. A pulse in the adult exceeding 120 per minute never denotes strength, and above this number it is feeble in proportion to frequency. Above 130 the pulse always denotes a degree of feebleness of the circulation rendering supporting measures highly important. The heart-sounds also constitute a good criterion for estimating the degree of feebleness of the heart's action. In proportion as the action of the heart is weakened, the intensity of the first sound over the apex is diminished; it is shortened, and in quality it resembles the second sound. If the heart be notably weakened, the first sound may be inappreciable over the apex. Stokes was the first to direct attention to these changes as criteria for the administration of alcoholics.

Alcoholics are rarely indicated in the early part of either typhus or typhoid fever. They are oftener and earlier indicated in typhus than in typhoid fever. Brandy or some form of spirit, and wine, sherry or Madeira, may be employed. Beginning with a moderate quantity, half an ounce of spirit or an ounce of wine, the effect on the circulation and other symptoms should be observed. A good effect is shown by increased force, with diminished frequency, of the pulse, perhaps some reduction of the temperature of the body, diminution of delirium, etc. The intervals should vary, according to the urgency with which this supporting measure is indicated, between six or eight hours and each successive hour. The quantity given at a time should rarely exceed that just stated. Guided by the true indications and by the effect, alcoholics will not do harm, and will often do much good in the treatment of typhus and typhoid fever. I have repeatedly tried their suspension for a few hours in cases in which they appeared to be urgently indicated, in order to become fully satisfied of their utility, and have found the pulse becoming more frequent, with increase of delirium and other ataxic symptoms, until their use was resumed. While a certain proportion of cases do well without them, and they are therefore not always indicated, I cannot doubt that I have seen not a few cases in which life was saved by their employment.

In these fevers, as in some other diseases, the tolerance of alcohol is often notably greater than in health. A patient may take from one to two pints of spirit per day who in health could not have taken one-fourth of this quantity without inebriation. The excitant or toxic effects of alcohol should never be produced in cases of fever. If these effects be produced, the indications for alcohol are not present or it is too largely given. In extreme cases an ounce or even more of spirit, given hourly, may be necessary; but in the majority of the cases in which alcoholics are indicated not more than six or eight ounces in the twenty-four hours are needed.

During convalescence alcoholics should be given moderately. If they have been used freely during the career of the fever, when convalescence begins their excitant effect may be for the first time apparent. Frequently malt liquors or light wines are best suited to convalescence. Solid food should be allowed as soon as convalescence is declared. The processes of digestion and assimilation are usually active, and the appetite may be voracious. The cautions to be observed relate to the kinds of food and the quantity taken at one time. The patient should be restricted to plain, wholesome, well-cooked articles of diet, and excess may be avoided by taking food at shorter intervals than in health. Convalescence from typhoid, more than from typhus fever, requires care as regards diet, in view of the probable existence of intestinal ulcerations. Gestation in the open air may be allowed early in the stage of convalescence, and as soon as their strength will permit patients may walk out of doors. Care with respect to much muscular exertion is important in convalescence from typhoid fever in consequence of a liability to intestinal perforation.

Measures of the utmost importance relate to the *prophylaxis* of typhus and of typhoid fever. It is established that by the introduction of modern sanitary arrangements these diseases have been made to disappear from districts where they formerly prevailed. These measures relate chiefly to matters of public hygiene which are beyond the power of the individual physician to accomplish, but which he may do much to advance by his advice. First in importance in cities is a good system of sewerage and a supply of good water.

In the prevention of typhus fever it is important to avoid overcrowding and to secure free ventilation. Patients with typhus should not be treated in hospital wards with other cases. This statement does not apply to patients affected with typhoid fever, as there is no risk of the diffusion of the latter among patients. The custom of transferring to fever-wards with cases of typhus patients affected with typhoid fever is objectionable in view of the risk of the latter contracting typhus during convalescence. Repeated instances in which this has occurred have fallen under my observation.

In every case of typhoid fever the disease should, if possible, be traced to its source, and continued exposure of the patient and others to the cause should be prevented. Careful examination for defects or leakage in water-closets, sinks, stationary washstands, drains, and soil-pipes should be made, and such defects remedied.

Drinking-water should be boiled if there be any ground for suspecting its purity. In order to prevent diffusion of the disease the intestinal dejections should be disinfected by adding a solution of the sulphate of iron and carbolic acid, and they should not be thrown into privies or water-closets, but buried, so as to avoid the possibility of their infecting either drinking-water or the atmosphere. Articles of clothing or bed-linen soiled by intestinal dejections should be at once plunged in boiling water or disinfected.

Relapsing Fever.

A form of continued fever heretofore known by a variety of names, but now called *relapsing fever*, received very little attention in this country prior to 1870. It has never been indigenous in this country. In June, 1844, 15 cases were admitted into the Philadelphia Hospital presenting the characters of relapsing fever, this name, however, at that time not being in use. These were probably the first cases reported in this country. The patients were Irish immigrants, all coming over in the same vessel. The cases were observed by Dr. Meredith Clymer, and were reported in his treatise on fevers.[1] In addition to these, so far as I know, the only cases observed are a few reported by Dr. A. Dubois in 1848,[2] and 15 cases which came under my observation in 1850–51, an account of which is contained in my *Clinical Reports on Continued Fever.*[3] The cases reported by Dr. Dubois and by me were among recent Irish immigrants. It is highly probable that other cases have occurred among immigrants which have not been reported, and which may have been confounded with typhus and typhoid fever. In the winter of 1869–70 the disease was imported by foreign immigrants, and it prevailed to a considerable extent in New York during this winter and the following summer. It prevailed also to some extent in others of the large cities of this country. At this time the wards of Bellevue Hospital afforded an ample opportunity for the clinical study of this disease; and my observations were the basis of a lecture published in the spring of 1870, to which was appended a statistical report of 103 cases prepared at my request by Dr. Thomas J. Moore, then one of the house-physicians of the hospital.[4]

It is not long since the distinctive features of relapsing fever have been pointed out and its claims to be considered as a distinct species of fever established. It is by no means, however, a new disease, and there is evidence of its having existed from a very early period in the history of medicine. In modern times it has prevailed at different epochs, especially in Ireland, Scotland, and England. It prevailed as an epidemic in Upper Silesia, a province of Prussia, in 1847, and among the troops in the Crimea during the war of France and England with Russia. In 1863 it again appeared in Russia, and in 1865 there was a great epidemic in St. Petersburg. Since this date there have been several epidemics of relapsing fever in Russia and in North Germany, particularly its eastern part. Among the different names heretofore applied to the disease are the following: Febris recurrens, typhus recurrens, spirillum fever, "five-day fever," "seven-day fever," "short fever," "mild yellow fever," "famine fever," "hunger-pest." The name relapsing fever is based on one of the most striking of the peculiarities of the disease, and involves no hypothesis; it is therefore to be preferred to any of the other appellations. The disease described by Griesinger as bilious typhoid fever is a severe form of relapsing fever.

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[1] *Fevers, their Diagnosis, Pathology, and Treatment*, by Meredith Clymer, M. D., Philadelphia, 1846, p. 99.

[2] Vide *Transactions of American Medical Association*, vol. ii.

[3] Vide *Clinical Reports on Continued Fever, based on Analyses of 164 Cases*, by Austin Flint, M. D., 1852.

[4] Vide *New York Medical Journal*, March, 1870.

ANATOMICAL CHARACTERS.—The lesions are those of an acute infectious disease. The *spleen* is enlarged, of a dark-red color, and of soft consistence. The enlargement is in many cases extreme, and resembles that which occurs in typhoid fever. In a few instances rupture of the spleen has taken place. Infarctions in this organ are not rare. Abscesses also occur, but they are less frequent. The Malpighian follicles are indistinct, although often swollen. They are sometimes the seat of small abscesses and of necrotic softening. The enlargement of the spleen is due to congestion and hyperplasia of its lymphoid elements. The *liver* is also enlarged, and of a reddish-gray or yellowish-brown color. It is the seat of parenchymatous and often of fatty degeneration. The *kidneys* are swollen, especially the cortex, and often present a number of small hemorrhages. The epithelial cells of the cortical tubes undergo granulo-fatty degeneration. Ponfick observed lymphoid cells in the interstitial tissue. These are the changes of an acute diffuse nephritis. They are not present in all cases, but they are not rare. There is usually a moderate degree of inflammation of the *intestinal tract*, the mucous membrane being congested, swollen, and coated with mucus. The solitary follicles are somewhat enlarged. Ponfick attributes the icterus often present in relapsing fever to gastro-duodenitis. *Ecchymoses* are often met with in the stomach. Subpleural and pericardial ecchymoses are not uncommon. Small hemorrhages have also been found in the brain. Petechiæ of the skin are present in about 10 per cent. of the cases. The striated *muscular substance* is the seat of parenchymatous and of hyaline degeneration, as in typhoid fever. The degeneration is frequent, and of especial importance in the muscular substance of the *heart*. The myocardium may be affected with a fatty degeneration almost as intense as that in phosphorus-poisoning. This degeneration may be the immediate cause of death. Ponfick observed in some cases changes in the *marrow of the bones*. These changes were partly diffuse and partly circumscribed. The diffuse changes consist in the appearance of large cells filled with fatty granules, especially in the course of the small arteries, and in an increase in the number of lymphoid elements. Another alteration of the marrow occasionally found is the presence of circumscribed foci of necrotic softening. The *blood* is dark in color and fluid or imperfectly coagulated. When examined during life the number of white corpuscles is often found notably increased (1 white to 50–20 red).

In 1873, Obermeier in Berlin discovered a spiral-shaped bacterium in the blood of relapsing-fever patients. This parasitic organism is called the *spirochæte Obermeieri*, or the spirochæte of relapsing fever. It belongs to the group called spiro-bacteria (page 92). This parasite has been found in the blood in no other disease.[1] The spirochætæ are sometimes, but not usually, found in the blood after death, and if found, they are observed to be motionless. These spiral organisms are extremely delicate, and are readily seen only with the high powers of the microscope. The diameter of a spirochæte is about that of the finest fibrils of fibrin. Its length varies between ten and forty micro-millimetres, or from one and a half to six times the diameter of a red blood-corpuscle. In a drop of blood drawn during life the spirochætæ are in rapid motion, which resembles somewhat that of the spermatozoa. The parasite has been found only in the blood, and not in the other fluids or in the organs of the body. It differs from most bacteria in the readiness with which it is destroyed by the action of various reagents. It is found almost invariably during the febrile paroxysms, and disappears during the intermission. It is occasionally found during the intermission, especially for a few hours after the crisis and for a few hours preceding the relapse. We are ignorant as to the life-history of this organism. Attempts to cultivate it artificially have failed. We do not know how it gains access to the body, or in what relation it stands to the phenomena of the disease. It is held by many writers that the spirochæte develops from spores which are present in the body during the intermissions.

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[1] The spirochæte Obermeieri resembles the *Spirochæte plicatalis* discovered by Cohn in mucus from the mouth, and a spirochæte previously discovered by Ehrenberg in water. Similar spiral-shaped bacteria have been found elsewhere.

CLINICAL HISTORY.—Under this head I shall state succinctly the symptomatic features which are characteristic of this fever. These relate to two or more successive paroxysms of fever separated by an apyrexial period or intermission. The recurrence of the paroxysm of fever, or the relapse, is the distinctive feature whence the name of the disease derives its significance.

Abruptness of invasion characterizes the disease. The attack is sudden. There is no prodromic period. The seizure is almost always marked by a well-pronounced chill which is immediately followed by febrile movement. Usually, the patient at once takes to the bed; but in some cases one, two, or three days pass before there is this evidence of yielding to the disease. Moderate perspiration occurs shortly after the fever begins in a considerable proportion of cases. The perspiration in some cases is abundant, and it may recur repeatedly during the continuance of the febrile paroxysm.

The fever attains quickly either considerable or great intensity, as denoted by the pulse and axillary temperature. Thus, of two cases in which the disease was developed in Bellevue Hospital, in one the pulse on the first day was 120 and the temperature 103°; in the other case the pulse on the first day was 130 and the temperature was 103°. During the continuance of the first paroxysm the pulse and temperature generally denote a persistent intensity of fever, the pulse ranging in different cases between 100 and 140 and the temperature between 100° and 105°. The oscillations are rarely great, and those which are observed are irregular in their occurrence.

The cessation of the fever is as abrupt as its invasion. The pulse and temperature quickly fall to nearly or quite the normal standard. The transition from high fever to complete apyrexia takes place often in a few hours; and usually this is accompanied by profuse perspiration, which continues for several hours, and even an entire day. Not infrequently the pulse and temperature fall below the standard of health. I have observed the pulse to fall as low as 54 and the temperature as low as 95°. In a day or two both the pulse and temperature rise again to the normal standard. Symptoms of collapse sometimes occur soon after the fall of temperature. They are usually of short duration.

The duration of the primary paroxysm in the majority of cases is between five and seven days. Exceptionally, it is only two days, and it may be fourteen days.

During the apyrexial period or intermission the absence of fever is complete. It is incorrect to call this period a remission. The fever does not remit, but it intermits. The average duration of this period is about seven days, but it may not exceed two or three days, and it may extend to twelve days or even more.

The relapse, like the primary attack, is sudden. It is generally ushered in by chilly sensations, but not so constantly by a well-pronounced chill as in the case of the first paroxysm. The fever in the relapse quickly becomes more or less intense. The intensity may exceed that of the first paroxysm, but in the majority of cases it is less. The relapse also ends suddenly, and in most cases with profuse perspiration.

The duration of the relapse varies between three and five days. It is sometimes, however, only twenty-four hours, and it may extend to ten days.

It is to be borne in mind that the relapse does not always take place. On the other hand, a second, a third, and even a fourth and a fifth relapse have been observed. In some epidemics there have been two relapses in the majority of cases. In others a second relapse has been the exception. The duration of the second intermission is eight or nine days, and that of the third paroxysm about three days. The duration of the disease averages about twenty-five days, but is longer if more than two febrile paroxysms occur. In none of the cases which I have seen has there been more than a single relapse.

The foregoing points are those most highly distinctive of relapsing fever. It remains to notice other points belonging to the clinical history which are more or less characteristic.

Of symptoms referable to the digestive system, nausea and vomiting occur sufficiently often to be somewhat distinctive, especially when this disease is contrasted with typhus and typhoid fever. Not infrequently, these symptoms are prominent and persistent during the febrile paroxysms. The matter vomited is green or yellow from the presence of bile. The tongue is generally coated, and in most cases it is moist. This moisture of the tongue in connection with high fever is somewhat distinctive of this disease. The tongue may, however, become dry and fissured. Herpes labialis sometimes occurs. Vomiting of blood presenting the character of "black vomit" has been observed, but it is a very rare symptom. It is generally associated with hemorrhage in other situations, and it is to be considered as an accidental event, not as an element of the disease. Epistaxis is a more frequent form of hemorrhage, and is observed especially during a crisis. In some epidemics a moderate diarrhœa has been the rule, and in others constipation. There is nothing characteristic as regards the condition of the bowels. Meteorism in a moderate degree is not uncommon. Tenderness on pressure over the epigastric region is frequent. Slight tenderness in the iliac region is not uncommon. Notable tenderness exists in most cases over the liver and spleen, and enlargement of these organs is determinable by palpation and percussion. I have not observed a craving for food during the paroxysms, which, according to some writers, is distinctive of this form of fever. The appetite, however, returns during the intermission, and the digestion may be active in this stage.

The occurrence of jaundice may be mentioned in this connection. A dirty, yellowish color of the skin is present in most cases, and to it has been assigned some diagnostic value. True jaundice occurs in a small proportion of cases, but its infrequency in the other continued fevers renders it somewhat distinctive of relapsing fever. It is doubtful if the statement that this event is an element of gravity be correct. The event is much more frequent in some epidemics than in others. The name "mild yellow fever" derives whatever pertinency it has from the occurrence of jaundice sometimes in a considerable proportion of cases, and also from the occasional occurrence of black vomit.

A symptom referable to the nervous and the muscular system is highly characteristic of the disease under consideration. I refer to arthritic and muscular pains, especially the latter. During the first paroxysm pains in the loins, the calves of the legs, and the muscles in other situations are generally much complained of. They are never wanting, although, as regards intensity, they differ considerably in different cases. The muscular pains do not cease with the ending of the paroxysm, but they continue during the intermission; they are more or less prominent during the relapse, and they often persist into convalescence. The muscles, especially those of the calf, are sensitive to pressure. The pains are sometimes referred to the bones. Ponfick thinks that this may be explained by the changes in the marrow which he observed.

The mental condition perhaps, in a measure, accounts for the suffering from these pains. The perceptions are not blunted in this disease, as they are in typhus and typhoid fever. This is a negative point of distinction in contrast with the fevers just named. Another negative point is the absence of the delirium which characterizes typhus and typhoid fever. Delirium is by no means absent in all cases of relapsing fever; but the delirium is such as may occur whenever there is high febrile movement, whether the fever be essential or symptomatic, and it is generally manifested only at night. In the daytime the mental faculties are generally intact. The condition known as coma-vigil does not belong to the clinical history of relapsing fever. This statement is also true of subsultus, carphologia, and other ataxic symptoms which occur in grave cases of typhus and typhoid fever. Deafness is also a rare symptom in relapsing fever.

A distinctive point, in comparison with typhus and typhoid fever, is the absence of a characteristic eruption. In most cases there is no eruption. Sudamina or miliary vesicles are sometimes observed at the time when profuse perspiration occurs, but this eruption is incident to various affections. The same is true of petechial spots which occur in some cases of relapsing fever. Other kinds of eruption are sometimes accidentally associated. Even the rose papules of typhoid fever sometimes occur in this as they do in various other diseases.

The physiognomy presents nothing distinctive. The face is more or less flushed, as in cases of symptomatic fever. There is not that degree of capillary congestion, marked especially on the cheeks, which exists in typhoid fever, or the dingy complexion which characterizes typhus. The expression of indifference, vacuity, or stupidity which is a notable characteristic of the fevers just named is rarely observed in relapsing fever.

The urine during the paroxysms is usually concentrated, dark in color, and rich in urates. In some epidemics albuminuria has been a not uncommon symptom. Blood-corpuscles and hyaline casts have also been found, but in the majority of cases neither albumen nor casts are present. Great diminution, and even suppression, of the urine, however, are sometimes observed, uræmic coma and convulsions taking place, but these cases are exceedingly rare.

It may be stated, as a point somewhat distinctive of relapsing fever, that there is very little liability to serious complications. In this fact we have an explanation, in part at least, of the very small rate of fatality from the disease.

What has just been stated with respect to complications holds true as regards sequels. As a rule, important sequels do not occur. The most frequent are ocular affections, particularly iritis and irido-choroiditis. Otitis media has also been repeatedly observed, although less frequently. Other complications or sequels of some clinical importance are parotiditis, laryngitis, dysentery, pneumonia, and acute diffuse nephritis. Relapsing fever, when it attacks pregnant women, always leads to miscarriage or abortion. The mother almost invariably recovers, but the child, no matter how near may be the end of gestation, as a rule is either stillborn or dies shortly after birth. The spirochætæ have been found in the blood of the fœtus.

DIAGNOSIS.—The diagnosis of relapsing fever is made certain by the discovery of the characteristic spirochætæ in the blood during a paroxysm of the disease. The cases in which competent observers have failed to find the organisms after searching for them are so very few that they may be left out of consideration. Other distinctive features of relapsing fever are such as to leave hardly any room for doubt concerning the diagnosis after the disease has ended. The distinctive characters are—the abruptness of the invasion, the rapid increment of fever, the frequent occurrence of moisture on the skin or perspiration more or less abundant without any marked abatement of the fever, the prominence of muscular and arthritic pains, and in certain cases the occurrence of jaundice. These are the positive points which are diagnostic. The fevers to be excluded are the eruptive fevers, febricula, remittent fever, and typhus and typhoid fever. Scarlet fever and measles are readily excluded by the absence of the eruption and of the characters which are distinctive of these fevers during the period of invasion. Smallpox is excluded after the third day by the continuance of fever and the absence of the eruption. It is not always practicable to decide at once that the disease is not a febricula, but the intensity of the fever and the notable muscular pains do not belong to the latter. Doubt, however, is soon removed by the persistence of the fever. Typhoid fever is excluded by the absence of a prolonged access and of the abdominal symptoms before sufficient time has elapsed for the appearance of the eruption, and by absence of the characteristic mental condition. Typhus is excluded by the absence of the eruption, which appears earlier than in typhoid fever, of the dusky complexion, and of the mental condition—the latter also being apparent earlier than in typhoid fever. Remittent fever is excluded by the absence of remissions, these being determinable by a notable diminution of the axillary temperature in some cases in which they are not distinctly denoted by the pulse. Of course the prevalence of relapsing fever is taken into account in arriving at the diagnosis.

CAUSATION.—Relapsing fever is undoubtedly a communicable disease. This statement rests on facts derived from the different sources of evidence, exclusive of inoculation, which establish the contagiousness of other diseases; as, for example, typhus fever. Of those who are attacked during the prevalence of the disease, a large proportion are known to have been brought into contact with, or close proximity to, patients affected with it. The disease is diffused in hospitals among fellow-patients and those who have charge of the sick. During the period in which cases were received in Bellevue Hospital after the disease began to prevail in this city—namely, between November 14, 1869, and February 6, 1870—twelve persons contracted the fever in the hospital. These twelve persons were especially brought into contact with patients affected with the disease, and in no instance did it attack one who had not been thus exposed. One of the senior assistant physicians residing in the hospital had the disease. The orderly in one of my wards contracted it, and his wife, who came to nurse him, was attacked by it. The disease has often been diffused after the importation of a case in localities in which it did not previously exist.

Facts, however, go to show that it is not a highly contagious disease. Considerable exposure is generally necessary. The area of the infection appears to be limited, and it remains to be ascertained whether it may be transported by fomites. Some facts cited by Murchison render it probable that it may be diffused in this way. It is especially communicable when the miasm is derived from a number of patients in the wards of a hospital or in close apartments. The disease is not likely to be contracted from single patients in well-ventilated rooms. The investigations of the Metropolitan Board of Health in New York during the prevalence of the disease showed that it is diffused chiefly among those living in overcrowded, ill-ventilated tenement-houses. Of the propriety and importance of removing these patients to hospitals or wards devoted specially to cases of this disease there can be no doubt.

Destitution, deprivations, and especially deficient alimentation, are powerful auxiliary causes. Of this fact the past history of the disease at different times and places furnishes abundant evidence. The significance of the names "famine fever" and "hunger-pest" relates to this fact. Statistics do not show any notable etiological influence pertaining to age, sex, or season.

Although its diffusion involves generally, and perhaps always, a contagious principle, the history of the disease in other countries shows that it occurs as an epidemic, and after continuing for a certain time it disappears completely. Thus, in the city of London for the fourteen years preceding the winter of 1868–69 there had been no cases of the disease. The complete disappearance of the disease for so long a period tends to show the necessity of co-operating causes in conjunction with the special poison on which the production of the disease depends. The effective means for arresting the spread of the disease are—thinning the population of overcrowded tenement-houses, dispersing the occupants of insalubrious dwellings, relieving destitution, especially as regards food, and promptly removing patients to hospitals devoted to cases of relapsing fever. An attack of this disease does not secure exemption from subsequent attacks. The period of incubation in more than one-half of the cases in which it is determinable does not exceed five days. It rarely exceeds nine days. Occasionally, as in typhoid and typhus fever, the symptoms begin almost immediately after the exposure.[1]

That the spiral bacteria discovered by Obermeier in the blood during a paroxysm of relapsing fever have an essential pathological connection with the disease is universally admitted. That they or their spores constitute the contagium, and that the phenomena of the disease are due to their presence in the blood, are conclusions vastly more probable than that either are effects of the fever or accidentally associated with it. That the agent of infection is contained in the blood does not admit of doubt. Motschutkoffsky has produced relapsing fever in human beings by inoculating them with blood taken during a paroxysm from a patient affected with the disease. Koch and Carter have produced the disease in monkeys by inoculating them with blood from a case of relapsing fever. Other animals do not appear to be susceptible to the disease. Blood taken during a paroxysm only is capable of producing the disease. Nothing is known of the life-history of the spirochæte of relapsing fever. It has not hitherto been possible to cultivate it artificially. It is more probable, for various reasons, that the spores of the spirochæte are the usual agents of contagion than that the spirochæte itself is the contagium. Nothing is positively known, however, concerning these spores, although it has been suspected that small round glistening bodies frequently found in the blood of relapsing-fever patients are such spores.

In accordance with the germ theory, the first paroxysm of relapsing fever is due to a brood of spirochætæ. Their existence terminates in about a week, as a rule, and the first febrile paroxysm then ends. In a small proportion of cases the conditions required for the production of a second brood of these organisms are wanting, and the disease then consists of a single paroxysm of fever. In the great majority of cases, however, these conditions are not wanting; hence the germs remaining in the body give rise to a second brood, and there is the relapse of the fever. In rare instances a third and a fourth brood are produced, but in most persons the conditions therefor are extinguished after a second paroxysm of fever. The conditions, however, are never permanently extinguished, as in cases of typhus and typhoid fever, yellow fever, small-pox, and the other eruptive fevers, for after a certain period they who have had relapsing fever are again susceptible to the contagium. The extinction of the conditions for the development and reproduction of the spirochætæ within the body is only temporary.

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[1] Vide Murchison, *St. Thomas's Hospital Reports*, vol. ii.

The contagium is probably contained in the breath and cutaneous exhalations. There are no facts tending to show that the intestinal dejections have any part in the diffusion of the disease.

PROGNOSIS.—It is, at first view, remarkable that a fever of such intensity, and prevailing especially among a class of persons whose powers of tolerance are impaired by previous hardships and want, should have such a small rate of mortality. The mortality in different collections of cases varies between 2 and 11 per cent. Dr. Moore, in his statistical report, states that of 103 cases admitted into Bellevue Hospital between November 14, 1869, and February 6, 1870, only 2 proved fatal. In some epidemics the mortality has been as high as 10 or 11 per cent., but as a rule it has not exceeded 2 to 4 per cent. In epidemics with the higher rates of mortality the complications have been many and the disease has presented the so-called bilious type. What is the explanation of the low rate of mortality? An explanation which perhaps is sufficient to account for the fact has been already given—namely, the disease is very rarely accompanied by any serious complications. The comparatively greater fatality of typhus and typhoid fever is due mainly to complications. These fevers rarely kill *per se;* that is, death is not purely an effect of the intensity of the disease.

Of the fatal cases of relapsing fever, the death in a certain proportion is attributable either to complications, such as pneumonia and dysentery, or to antecedent affections—as, for example, disease of the kidneys and chronic alcoholism—but this fever may destroy life irrespective of any important complications or antecedent affection. Several observers have reported cases in which sudden death occurred apparently from syncope. Dr. Moore's report exemplified this fact. The death in one case was sudden and unexpected during the night on the seventh day of the disease. At the time of death the primary paroxysm seemed about to end, free perspiration having taken place. Neither coma nor convulsions occurred, and, so far as information could be obtained, the dying was by syncope. The fatty degeneration of the heart explains, in many cases at least, death by syncope.

Suppression of urine followed by uræmic coma and convulsions is sometimes a cause of death. This fact was exemplified by one of the two fatal cases occurring in Bellevue Hospital. These symptoms are due to acute diffuse nephritis, which is one of the recognized complications of relapsing fever.

TREATMENT.—There are no known means which can be relied upon for cutting short this disease. With our present knowledge there are no remedies which, employed in the intermission, will prevent the relapse. Quinia has been used freely, but with no success. Its inutility as a prophylactic has been abundantly shown by different observers. It has been recently claimed that calomel exerts a favorable influence upon the progress of the disease. The treatment, with our present knowledge, must be expectant, meaning by this term that it is to consist of palliative measures and those addressed to the particular indications in individual cases.

The intensity of the fever during the paroxysms may be lessened by sponging the body with water and by the wet sheet. Cold water may be taken into the stomach freely. Ice-cold carbonic-acid water is an acceptable and useful drink to allay thirst. Cephalalgia may be relieved by cold applications to the head. The bowels should be kept open by saline laxatives. The muscular and arthritic pains call for the use of opium, especially if it produce no unpleasant after-effects. Irrespective of the pains, opiates are indicated to relieve sleeplessness.

The dietetic treatment is important, especially in the cases in which deficient alimentation has been an auxiliary cause of the disease. In this fever, as in other fevers when alimentary support is indicated, milk is the form of diet to be preferred; and it is to be borne in mind that in this, as in most diseases, there is never any danger of the over-appropriation of nutriment, the only risk being in the ingestion of more food than can be digested. It is desirable that during the paroxysms one or two quarts of milk should be taken daily. In the intermission, when the appetite returns, as much substantial food as can be digested should be allowed; and the more food is appropriated in this stage the better is the patient enabled to tolerate the relapse. Tonic remedies are indicated throughout the disease, especially in the intermission. Quinia in small doses and some preparation of iron are the tonics to be preferred.

If the symptoms denote asthenia, alcoholics are indicated. The occasional occurrence of death from syncope renders it important to watch carefully for this indication. When there is room for supposing that alcoholics may be useful they should be given tentatively, and continued or not according to the effect, especially on the pulse and temperature. They are of course urgently indicated if the symptoms denote danger in the direction of asthenia.

Attention to the urine is important. It is to be remembered that a cause of death is suppression of urine. If the urinary excretion be deficient in quantity, or if, the quantity being sufficient, there be a deficiency of urea, diuretics are indicated. If the kidneys do not respond to diuretics, hydragogue cathartics are indicated, with a view to eliminate urea through the alimentary canal. Active hydragogues are of course indicated if uræmic coma or convulsions supervene.

It is stated that convalescence from relapsing fever is notably slow, patients remaining for a long time much enfeebled, and tolerating with difficulty affections which may occur before the recovery is complete. This has not been a marked peculiarity in the cases which have come under my observation. It is doubtless more likely to be marked in the cases in which there has been impairment of the vital powers from innutrition and hardships before the fever was contracted; and also it is more likely to be marked when abundant alimentation and the judicious use of alcohol have not entered into the treatment of the disease.

Erysipelatous Fever.

Erysipelas, considered as a local affection, belongs to surgery rather than to medicine. For an account of this form of inflammation, therefore, the reader is referred to surgical works. It occurs as an occasional complication of the continued fevers which have been considered. A form of continued fever distinct from the fevers which have been considered is characterized by the frequent occurrence of erysipelas, and hence it is called *erysipelatous fever*. As a distinct form or species of fever this is to be distinguished from erysipelas occurring as a local affection and accompanied with more or less febrile movement. The fever in the latter is symptomatic, whereas in erysipelatous fever it is primary or essential. On the other hand, erysipelatous fever is to be distinguished from typhus, typhoid, and relapsing fever with erysipelas as a complication.

Erysipelatous fever occurs as a sporadic and as an epidemic disease. As a sporadic disease it is rare, and has not been as yet sufficiently studied by means of the analysis of recorded cases. The invasion is either abrupt or there is a forming stage of variable duration. The erysipelas may be developed within a few hours from the attack or after the lapse of one, two, or three days. The head is oftenest the seat of the erysipelas, but it may be developed in any part of the body. Different parts may be successively invaded. I have known it to extend over the whole surface of the body. The febrile movement is more or less intense. Passive delirium and other ataxic symptoms are not infrequently developed; in other words, the typhoid state exists. The abdominal symptoms of typhoid fever are wanting, and the career of the disease is shorter than that of typhoid fever. The symptoms are modified, and the disease, as regards duration and danger, is affected materially by the degree of intensity, the extent, and the local results of the erysipelas. The general principles of treatment are the same as in other of the continued fevers. In the existing state of our knowledge of this fever I shall pass it by with this brief notice. It is sometimes classed among the eruptive fevers, but the erysipelas is hardly to be considered as a cutaneous eruption.

Epidemic erysipelatous fever I shall also notice very briefly. It prevailed extensively in this country between 1841 and 1846. It was not confined to a particular section, but prevailed in certain districts in the New England, Middle, Western, and Southern States. In some localities great numbers were affected and the mortality was large. It was commonly known in certain parts of the country as the "black tongue," from an appearance of the tongue occasionally observed. The medical journals of this country during the years just named and afterward contained many articles relating to the prevalence of the disease in different localities. Of these, an article by Drs. Hall and Dexter, giving an account of the disease as it prevailed in Vermont,[1] and a paper by the late Dr. Bennett of Bridgeport, Conn., are especially deserving of notice. The latter gives an analysis of various articles contained in different medical journals.[2]

The epidemic prevailed in isolated sections. It did not appear to migrate or to be transported from place to place. Its course was "irregular and erratic." Dr. Bennett, however, states that it sometimes appeared to follow the direction of rivers, small streams, and lakes. The attack was sometimes sudden and sometimes preceded by premonitions, the latter rarely existing longer than twenty-four hours, and being the same as those belonging to the access of other fevers. A pronounced and frequently a prolonged chill, with or without rigor, ushered in the disease, accompanied often by pains in the extremities and in some cases by great prostration. Pharyngitis was at once or speedily developed; and this local affection was constant. It varied much in intensity in different cases. Not infrequently it was attended with great swelling of the tonsils, and sometimes sloughing occurred. The inflammation sometimes involved the larynx, and in a certain proportion of cases death was due to either laryngitis or œdema of the glottis. The lymphatic glands of the neck became more or less swollen, the swelling in some cases being very great, and occasionally suppuration taking place.

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[1] Vide *American Journal of the Medical Sciences*, January, 1844.

[2] Vide *New York Journal of Medicine*, July, 1853.

Erysipelas was far from being constant. The frequency of its occurrence varied at different times and places. Dr. Bennett observed it in only one-sixth of one hundred and fifty cases. In view of the fact that erysipelas occurs in only a certain proportion of cases, the propriety of calling the disease erysipelatous fever rests upon the absence of any other and better name. The erysipelas was seated in different parts. It frequently led to suppuration, gangrene, and sloughing. The danger and protracted character of the disease depended on the degree, extent, and local results of the erysipelas.

The febrile movement was more or less intense. Erratic pains, like those of neuralgia and muscular rheumatism, were common during the course of the disease. Typhoid delirium and other ataxic symptoms occurred in severe cases. Petechiæ were sometimes observed. Abscesses without erysipelas occurred in some cases. The disease was not infrequently complicated with inflammation of serous membranes, as the pleura, peritoneum, and the cerebral meninges. Pneumonitis was an occasional complication.

Different cases differed much as regards the gravity of the disease. In many cases the disease was mild and ended in five or six days. The severity and danger were generally due to the complications. Of the cases complicated with laryngitis or œdema of the glottis, extensive erysipelas, serous inflammations, or pneumonitis, a large proportion ended fatally, the duration being very prolonged.

Puerperal peritonitis prevailed in conjunction with this epidemic so uniformly as to show a pathological relationship between these two affections. The opinion was generally held that the peritonitis was caused by a virus or miasm carried by the obstetrician from patients affected with the epidemic disease. Assuming this to be true in certain cases, puerperal peritonitis was by no means uniformly to be accounted for in this way. A rational explanation in many, if not in all, cases is that labor acted as an exciting cause and determined the situation of the local affection in those predisposed to the disease from the action of the epidemic influence. Many, if not most, practitioners held that the disease was communicable. Its contagiousness, however, cannot be considered as established.

As regards TREATMENT there was a diversity of opinion. Some practitioners advocated bleeding and other of the measures called antiphlogistic; but the majority, before the epidemic ceased to prevail, were convinced of the impropriety of these measures. In view of the symptoms, the nature of the disease, the tendency to suppuration and gangrene in the cases in which erysipelas existed, and the other complications which were liable to occur, there can hardly be a question as to the propriety of measures of an opposite character. Tonic remedies, alimentation, and alcoholic stimulants—in other words, supporting measures of treatment—were indicated. Mild cases required little or no treatment, but in grave cases were applicable the principles which should govern the management of other essential fevers with serious local complications.

Epidemic Fever characterized by Pharyngitis.

In the winter and spring of 1857 an epidemic fever prevailed in the western part of the State of New York, in the adjacent parts of Pennsylvania, and in Canada, characterized by inflammation of the pharynx of a mild grade of intensity, and unaccompanied by either diphtheritic exudation or much submucous infiltration. I reported the results of an analysis of 23 cases which I observed and recorded. A report of this epidemic, based upon notes of 37 cases, was at the same time made by Prof. Rochester.[1] The different cases presented great uniformity as regarded the general and local symptoms. The fever was ushered in with a chill without rigor. The febrile movement in all the cases was considerable or marked. The severity of the disease was generally sufficient to keep the patients in bed for several days. The lymphatic glands of the neck were in most cases moderately enlarged, but without suppuration. Soreness of the throat was not a prominent symptom, even when the pharyngitis was marked. There was in no case an eruption. Vomiting did not occur, and generally the bowels were constipated. There were no symptoms of importance referable to the pulmonary and nervous system. The duration of the fever was between three and six days. It affected persons of either sex and of all ages. The epidemic continued about two months, reaching its acme gradually and gradually declining. In no case under my observation did it prove fatal.

The disease was not scarlatina, as shown by its affecting persons who had had that disease, by the fact that scarlatina did not prevail at the time, by the absence of an eruption in all the cases, by the non-occurrence of dropsy as a sequel in any case, and the fact that middle-aged and aged persons were attacked as well as children and infants. There were no cases of epidemic pharyngitis with exudation or diphtheria in that part of the country. The fever resembled the mild cases of erysipelatous fever which occurred during the epidemic prevalence of that disease, but erysipelas was not developed in any case. The cases bore a close analogy to those of influenza, except that the local affection was seated in the pharynx, and not in the Schneiderian and bronchial mucous membrane.

Dr. Harvey E. Brown, Assistant Surgeon U. S. Army, kindly communicated to me an account of an epidemic similar to that just described which prevailed among the United States troops stationed at Hart's Island, Long Island Sound, in January and February, 1866.

Palliative measures of TREATMENT only were indicated.

CHAPTER IV.

PERIODICAL FEVERS.

Intermittent Fever: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Pernicious Intermittent Fever.

THE periodical are distinguished from the continued fevers by the occurrence of febrile paroxysms or marked exacerbations in a regular order of succession, thereby exemplifying a law of periodicity. This distinction applies especially to the fevers called intermittent and remittent. Yellow fever is generally included among the periodical fevers, and it belongs perhaps more appropriately here than in any other nosological division. The intermittent and remittent fevers are often distinguished as *malarial fevers*. These fevers will be found to present many striking points of difference as contrasted with the fevers which have been considered in the preceding chapters. They differ not only in the events of the clinical history, but as regards the laws governing their causation, duration, complication, sequels, and the extent to which they are controllable by known remedies.

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[1] For both reports, vide *Buffalo Med. Journal*, 1857.

Intermittent Fever.

An intermittent fever is characterized by the occurrence of febrile paroxysms in regular succession and by the absence of febrile movement between the paroxysms. The intermission is the distinctive feature of this form of fever, as its name implies. Popularly, the disease is known as "fever and ague," "chill fever," "the shakes," and by names expressive of the locality in which it is produced, as "swamp fever," "Panama fever," "Chagres fever," etc. Although essentially the same disease, intermittent fever as ordinarily presented in practice is unattended with danger to life, but occasionally it is one of the most dangerous of maladies. I shall consider first simple or ordinary intermittent fever, and afterward the grave forms of the disease under the head of *Pernicious Intermittent Fever.*

ANATOMICAL CHARACTERS.—The essential anatomical lesion in all forms of malarial fever is the presence of dark pigment in the blood and in certain organs. A certain amount of this pigment is formed in simple intermittent fever. The quantity of this pigment and other malarial anatomical changes are so much greater in the more severe forms of malarial poisoning that the consideration of these changes will be referred to the article on Remittent Fever. The spleen is more or less enlarged in simple intermittent fever.

CLINICAL HISTORY.—The clinical history will embrace an account, *first*, of the paroxysm; and *second*, of the intermission.

In the majority of cases the attack is sudden. In a certain proportion of cases, however, there are premonitions for a variable period. The premonitions are not very distinctive of this disease, consisting of pain in the head, yawnings, indisposition to exertion, loss of appetite, and general malaise. Although not very definite, they sometimes suffice to lead patients who have been repeatedly affected with intermittent fever to anticipate an impending attack.

A paroxysm, when complete, consists of three distinct periods or stages—namely, the cold, the hot, and the sweating stage.

Cold Stage.—This stage presents first a feeling of chilliness, beginning in the loins and extending thence over the back and limbs. The chill is more or less intense, in some cases consisting of a slight creeping sensation of coldness with shiverings, being in other cases extremely severe. Muscular tremor, commonly known as rigor, may or may not accompany the chill. I have met with a single example of distinct rigor without a sensation of chilliness. During the chill bristling of the hairs over the body, or horripilation, and the appearance familiar as "goose-skin," are often observed. Notwithstanding the sensation of coldness which the patient experiences, and the feeling of coldness when the hand is placed on certain parts, the thermometer in the rectum shows an increase of the temperature of the body. The thermometer, however, applied to the extremities shows a decrease of temperature below that of health. The increase of the temperature of the body begins prior to the paroxysm. The sensation of coldness is sometimes limited to a part of the body, as the back or the extremities. During the rigor frequently the teeth chatter and the movements of the body and limbs are sufficient to shake with considerable violence the bed on which the patient lies.

During this stage the patient frequently sighs, the pulse, which is usually accelerated, is small and feeble, the countenance has an expression of anxiety, the prolabia and face are pale or frequently livid, and lividity is often marked at the roots of the nails. The general appearance is as if the person had been exposed to intense cold. The circulation is sometimes completely suspended in the fingers, which are waxy in appearance, devoid of sensibility, and bleed less than usual if wounded. Other symptoms are—mental irritability, a sense of oppression referred to the præcordia, pain in the head and limbs, and palpitation.

The duration of this stage varies between a few moments and two hours or even longer. The average duration may be estimated at from a half to three-quarters of an hour. The transition to the next stage is sometimes abrupt, but is generally gradual. Flushings of heat are felt, the rigor ceases, the coldness, as it were, melts away, febrile movement is developed, and the cold stage is then ended.

The cold stage is not infrequently wanting, the paroxysm beginning with the hot stage. I have known a state of intense nervousness to take the place of the cold stage. Gastralgia or gastric irritability, denoted by pain or incessant vomiting, is a morbid condition which sometimes takes the place of this stage, as well as drowsiness or stupor and a condition resembling hysterical coma. Veritable coma is liable to occur in this stage in a form of the disease which will be noticed separately under the head of *Pernicious Intermittent Fever*. In young children convulsions are liable to occur in the cold stage.

Congestion of internal organs is necessarily involved in the cold stage, inasmuch as there is less blood in the vessels of the surface and the whole mass of blood is not diminished; but the primary and essential morbid condition underlies the congestion, and it is probable that the latter is an effect of an active contraction or spasm of the peripheral vessels, the blood accumulating in the internal organs in consequence of its being driven from the surface of the body. That the congestion incident to this stage does not stand in a causative relation to the other stages is shown by the fact that the cold stage is sometimes wanting, and that the other stages in these cases are not less marked than when the cold stage occurs.

Hot Stage.—This stage is characterized by more or less intense pyrexia. The skin becomes hot, the pulse is accelerated and full or bounding, the face is flushed, the cephalalgia continues, but the pain in the limbs and the præcordial oppression disappear. The axillary temperature rises notably, usually reaching 105° or 106° F. Thirst is usually a prominent symptom. The intensity of the pyrexia varies considerably in different cases. The duration of this stage is between three and eight hours. It is evident that in this stage the spasm of the vessels of the periphery which existed in the cold stage has ceased, and that, on the contrary, the distributing vessels of the surface of the body are abundantly dilated.

Sweating Stage.—Perspiration appears first on the face, and afterward on the trunk and extremities. The pyrexia gradually abates, and at length disappears. The heat of the surface, cephalalgia, thirst, restlessness, etc. cease. The thermometer indicates a rapid defervescence, the heat falling to the normal standard. The patient obtains refreshing sleep. With this stage the paroxysm ends. As regards the amount of sweating cases differ, it being sometimes profuse and sometimes slight. The duration of the sweating stage is variable, the average duration being three or four hours. The sweating is evidence that the pyrexia is about to end. That it is the means of bringing the paroxysm to a close is by no means certain; it is a sign of the approaching intermission, and it may be an effect rather than a cause of the decrease of the pyrexia. During and succeeding the paroxysm, urea, uric acid, and the chlorides in the urine are increased. The urine is not infrequently albuminous, and occasionally there is hæmaturia.

The intermission, called also the apyrexial period, is the space of time between two successive paroxysms. The period from the beginning of one to the beginning of the next paroxysm is called the interval in distinction from the intermission. The duration of the interval is the basis of a division into varieties commonly known as the different types of the disease. Each variety or type observes a law of periodicity in the succession of paroxysms. There are three simple types of intermittent fever—namely, the *quotidian*, the *tertian*, and the *quartan* type. In the *quotidian* type the interval is about twenty-four hours, or the paroxysm recurs daily, as the name imports. In the *tertian* type the interval is about forty-eight hours, or the paroxysm recurs on the third day, reckoning the days on which two successive paroxysms take place. In the *quartan* type the interval is about seventy-two hours, or the paroxysm recurs on the fourth day. As a rule, the paroxysms are uniform in each individual case as regards the occurrence of the cold stage, the duration of the several stages respectively, the severity of each, etc., but they present considerable diversity in different cases. Of the three simple types, the quotidian and tertian type are by far the most frequent. Examples of the quartan type are comparatively rare. Of 98,237 cases of intermittent fever in the United States army, only 1757 were of the quartan type.[1]

As regards the relative frequency of quotidian and tertian cases, either variety may predominate at particular seasons. In the same locality the majority of cases are at one season of the quotidian and at another season of the tertian type. In an aggregate of cases occurring in a series of years the quotidian type predominates. Of 98,237 cases in the United States army, 51,623 cases were of quotidian and 44,857 cases of tertian type.[2] Cases have been observed in which the paroxysm has recurred on the fifth, sixth, seventh, and eighth day, and hence there may be a quintan, a sextan, a heptan, and an octan type of intermittent fever. Such cases, however, are among the rare curiosities of clinical experience. In some cases during the continuance of the disease the type changes, the quotidian succeeding the tertian, or *vice versâ;* such cases, however, are rare.

Cases are observed occasionally in which one of the simple types is duplicated; that is, two sets of paroxysms of the same type coexist. Thus, one compound type is called a *double quotidian*, two paroxysms occurring daily. Another compound type is a *double tertian.* In a double tertian a paroxysm occurs daily, but the paroxysms on successive days take place at different hours, and may differ in various ways, whereas on alternate days they occur at the same hour and correspond in other respects. Again, a tertian is doubled or duplicated when two paroxysms occur on alternate days. Another compound type is a *double quartan.* In this type a paroxysm occurs on two successive days, and on the third day there is no paroxysm. Finally, a *triple quartan* is a compound type in which a paroxysm occurs on three successive days, the paroxysms on these days presenting notable points of difference, but each paroxysm corresponding with that which recurs on the fourth day. These compound types, except the double tertian, are extremely rare, and are interesting merely as curiosities of clinical experience. The double tertian is not very infrequent.

Paroxysms may occur at any hour of the day. They rarely occur during the night. In a large majority of the cases of the quotidian and tertian type the paroxysms occur before noon. Quartans are as likely to occur after as before the middle of the day. In infants the type is almost uniformly quotidian. As a rule, the successive paroxysms recur precisely or nearly at the same hour; but sometimes there is a variation from this rule, and the variation is in accordance with a certain law whenever the paroxysms are said to be either anticipating or retarding. They are anticipating when each successive paroxysm recurs earlier by the same period than the last. Thus, they may regularly anticipate half an hour, an hour and a half, etc. The paroxysms are retarding when each successive paroxysm is in like manner delayed. This variation in time of recurrence is generally evidence that the disease is about to end.

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[1] Vide Woodward, *Camp Diseases of the United States Army,* Philadelphia, 1863.

[2] *Ibid.*

During the intermission there is much difference in different cases as regards freedom from ailments. In some cases the patient complains only of a certain amount of debility; the appetite and digestion are good, and there is no apparent disorder of any of the functions. In other cases marked prostration follows, the appetite and digestion are more or less impaired, and various disturbances exist.

Probably more or less enlargement of the spleen occurs in all cases of malarial fever. The enlargement is often sufficient for the organ to be readily felt through the abdominal walls, constituting what is commonly known as the "ague cake" in districts where the disease prevails. The organ in some cases becomes enormously enlarged. A splenic tumor of large size is not very frequent. It occurs in some cases when the disease has been of short duration, and it is by no means developed in the majority of the cases in which the disease has existed for a long period. It may continue for a considerable period after the paroxysms cease.

Anæmia is incident to the disease, especially if it have been of considerable duration. The pallor of the face in cases in which the disease has been protracted or in which frequent relapses have taken place is often associated with a yellowish or sallow tint and with more or less œdema, giving rise to a characteristic facies. These characters denote what is called the *malarial cachexia*. General dropsy is an occasional concomitant or sequel. Notable anasarca with effusion into the serous cavities is sometimes incident to the disease, and does not, therefore, necessarily denote either a cardiac or a renal affection. An herpetic eruption about the mouth is a frequent concomitant.

The DURATION of the disease is indefinite. It not infrequently ends spontaneously after a few paroxysms, but in many cases it continues for weeks, and even months, if not arrested by curative means. Its indefiniteness of duration is a striking point of difference as contrasted with the continued fevers; and its self-limitation is not, as in the latter, restricted within a certain period. Another not less striking point of difference relates to the liability to relapses. Subsequent attacks occur in the great majority of cases. The liability to relapse remains for many years. In some cases successive attacks occur regularly after a certain period, showing that in these cases the relapses take place in accordance with an intrinsic tendency; that is, irrespective of external causes. The notion was formerly entertained that the tendency to relapse was lessened if the disease were left to end of its own accord or "wear itself out." So far from this being true, I believe that clinical experience shows a diminished tendency to relapse in proportion as the paroxysms are speedily interrupted by therapeutical interference. After the paroxysms have ceased to recur, either spontaneously or from the use of remedies, thermometric observations may show more or less increase of the heat of the body at the time when the paroxysm was to be expected. So long as these fluctuations of temperature exist the disease has not completely diappeared and relapses are liable to occur.

Physicians in malarial districts become familiar with cases which may be called cases of *latent intermittent fever*. In these cases paroxysms are not fully developed, but the patient complains of indefinite ailments, to which the term *bilious* is frequently applied. The appetite is impaired or lost; and there may be nausea and occasional vomiting, with pain in the head, indisposition to exertion, etc. The tongue is frequently covered with a thin and very white coating. A close examination will sometimes disclose evidence of periodicity in an increase of ailments at regular periods on successive days or on alternate days, and slight manifestations of the several stages of a paroxysm may be observed. The thermometer in the axilla is useful as a means of diagnosis in these cases, showing a periodical rise of temperature when the surface of the body may not show increase of heat and the pulse is but little accelerated. In a case under observation at Bellevue Hospital at the time of writing the morning temperature in the axilla was 105° F., the pulse being only 80, and at evening the temperature was 100°. Under the free use of quinia the daily increase of temperature rapidly diminished, and in a short time ceased. The prompt relief afforded by treatment addressed to a suspected malarial condition shows the correctness of the suspicion; and this treatment may be resorted to tentatively in cases of doubt. The affection in these cases is sometimes called *dumb ague*. This term is also applied to paroxysms in which rigors are wanting.

Intermittent fever is said to be *masked* in certain cases in which it is associated with other affections. It may exist in combination with various affections, such as bronchitis, pneumonitis, dysentery, etc. The association may render the periodical disease irregular and interfere with the full development of the paroxysms. In these cases it is not correct to apply to the associated affections the term malarial, meaning thereby that these affections are due to malaria. They are simply accompanied by phenomena proceeding from malarial disease existing in combination. In malarial districts the influence of malaria is liable to pervade all affections and it claims special measures of treatment. Of this fact practitioners in these districts become fully aware. Paroxysms of neuralgia sometimes appear to take the place of the paroxysms of intermittent fever, recurring with the same regularity after intervals corresponding to those in the different types of intermittent fever, and a cure is effected by the remedies which are efficacious in the latter disease.

CAUSATION.—The causation involves a special morbific agent commonly known as *malaria*. The existence of a special cause is logically inferred from the peculiar character of the disease, its limitation to certain localities called malarial, and the fact that it is controlled by remedies having a specific operation. The production of the special cause was attributed to vegetable decomposition in marshy localities and called *marsh miasm* in 1717 by Lancisi, an Italian writer. This doctrine and the name have since been very generally adopted; but that something more than ordinary vegetable decomposition is requisite for its production is sufficiently proven by the disease being indigenous in certain localities, whereas in certain districts and countries in which vegetable decomposition must take place abundantly the disease never occurs. Observation shows that it is produced especially in marshy localities, but its production is not confined to such situations, and hence the name marsh miasm is inappropriate. The production of the special cause requires a temperature above 60° F. In so-called malarial districts it is more abundantly produced when a dry follows a wet period. Turning up of the soil is often followed by an increase of its production. It often disappears after the cultivation of soil for several years, so that highly malarial districts may after a time become free from malaria. On the other hand, the area of a malarial district is sometimes greatly enlarged by a gradual extension. During the last twenty-five years the malarial district near the city of New York has extended far upward on the banks of the Hudson River, eastward into the State of Connecticut, and along the banks of the Connecticut River into Massachusetts, periodical fevers until lately not having been endemic in these situations for a long period.

The telluric source of malaria is proven by the fact of its being endemic in certain localities. Facts show that the morbific agent remains near the surface of the earth. Persons sleeping in an upper story may escape when those sleeping on a level with the ground become affected. It is more abundant in the night-air than during the day, and hence the disease may often be escaped by avoiding exposure in the evening, night, or early in the morning. The special cause of this disease is not eliminated from the bodies of those affected in such a manner that the causation ever involves a contagium. Facts abundantly prove that the disease is not communicable from one person to another. The malarial fevers therefore belong among those distinguished as purely miasmatic.

Various attempts have been made to discover parasitic organisms in the blood of persons affected with malarial fever. The so-called ague-plants or palmellæ, which Salisbury in a paper published in 1866 claimed to be the special cause of malarial fever, have at present only historical interest. The same may be said of the bacilli which Klebs and Tommasi-Crudeli found in the ground of malarial regions, and which they supposed to be the special cause of malaria. There is no proof whatever that either the palmellæ of Salisbury or the alleged bacillus malariæ of Klebs and Tommasi-Crudeli are in any way concerned in the production of malaria. Bodies which were discovered in 1881 by Laveran in malarial blood, and which have been more carefully studied since 1883 by Marchiafava and Celli in Rome, deserve more serious consideration.[1] These bodies are found usually within red blood-corpuscles, but they may exist free in the blood. They vary in size, the average being about one-third the size of red blood-corpuscles, but many are smaller and others are larger than this. Many of the bodies contain black or dark-brown pigment-granules, and others are free from pigment. When unstained the bodies are clear and homogeneous in appearance (with the exception, possibly, of included pigment), and so delicate that they are not readily detected. The bodies can be stained with fuchsin, methylene-blue and some other aniline dyes. In stained specimens the shape of the bodies varies, some being round, others elongated, crescentic, and of various other, often very irregular, shapes. In a drop of fresh blood the bodies are endowed with the power of movement, changing their shape in a manner analogous to the amœboid movements of white blood-corpuscles, and even more rapidly. Exceptionally, movable thread-like filaments are attached to the bodies. The main point in favor of these bodies being living organisms is their property of amœboid movement. They have been found only in the blood of malarial patients, and chiefly during a paroxysm of malarial fever. They may be present in only a few red corpuscles or they may be abundant. Marchiafava and Celli claim to have produced typical intermittent fever by the injection into a vein of a person free from malarial history of malarial blood containing these bodies, and to have afterward found similar bodies in the blood of the individuals experimented upon.[1] They consider these bodies to be parasitic organisms which penetrate the red blood-corpuscles, which they partly or wholly destroy, and in which they elaborate the characteristic malarial pigment. If these bodies be parasitic organisms, they are entirely different from bacteria and resemble amœbæ. It is urged, as a point in favor of the view that the parasitic organism of malaria is an amœba-like organism rather than a bacterium, that quinine exerts a much more powerful influence in arresting the vitality of the amœbæ than that of the bacteria. Attempts to cultivate these bodies have failed. It has been urged, in opposition to the view that these bodies are parasitic organisms, that they may be the result of some degenerative or other metamorphosis in the red blood-corpuscles. The possibility of such a metamorphosis cannot be denied, but it would be without analogy in any known pathological changes.[2] Should Laveran's corpuscles prove to be parasitic organisms peculiar to malarial blood, the evidence for regarding them as the specific cause of the disease would of course be very strong.[3]

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[1] Laveran, *Traité des Fièvres palustres*, Paris, 1884; Marchiafava and Celli, several articles in *Fortschritte d. Medicin* in 1883 and 1885. The latter authors call this supposed parasitic organism, Plasmodium malariæ.

Councilman and Abbot in 1885 described the presence in malarial blood of small hyaline corpuscles, most of which contain pigment. These corpuscles are probably identical with those found by Marchiafava and Celli. (See Councilman and Abbot, *Am. Journ. of the Med. Sciences*, 1885.)

In the preceding edition of this book the presence of a hyaline envelope around malarial pigment-granules was mentioned.

The period of incubation is indefinite. It may be a few days only or a few weeks, but it is not infrequently many months, and perhaps years may elapse after the reception of malaria before its morbid effects are manifested. Auxiliary conditions often appear to be necessary to give efficiency to the special cause. An attack is liable to occur after exposure to cold, over-exertion, excesses in eating or drinking, etc. The activity of the special cause is also often awakened by the occurrence of some other affection. The disease occurs especially in the spring and autumn, probably in consequence of the action of auxiliary causes in these seasons. The vernal intermittents are in general milder than the autumnal.

The disease shows no preference for either sex and it affects all ages.

If the paroxysms be caused by the development and multiplication within the body of vegetable organisms, it is to be supposed that a crop sufficient to occasion an attack either dies or is eliminated when the paroxysm ends, and that from germs remaining in the system other successive crops are produced after definite periods of time corresponding to the different types of the disease. These different types seem to imply different varieties of the ague-producing organism.

DIAGNOSIS.—In well-marked cases the diagnosis offers no difficulty. The type is to be determined by the duration of the intervals and a comparison of the paroxysms. Latent and masked intermittents are not so easily recognized; and in arriving at a positive diagnosis the effect of treatment is sometimes to be considered. Febrile paroxysms resembling those of intermittent fever are sometimes observed in connection with pulmonary phthisis. They may present the three stages well marked, and recur at regular intervals. They oftener occur in the afternoon than in the forenoon, whereas the reverse obtains in intermittent fever. The coexistence of phthisis is to be taken into account in making this differential diagnosis, as well as the known exposure to malaria or otherwise. The treatment appropriate to intermittent fever will sometimes arrest the paroxysms connected with phthisis. Chills connected with suppuration in some of the internal organs may suggest as probable the action of malaria in malarial districts. The irregularity of their occurrence, the inefficacy of treatment specially directed to a malarial affection, and the discovery of the local affection will in time clear up the diagnosis. Happily, the remedies indicated in malarial diseases are rarely hurtful in the cases in which intermittent fever is incorrectly supposed to exist. Operations on the urinary passages, such as catheterism and lithotrity, sometimes produce paroxysms resembling those of intermittent fever.

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[1] Gerhardt had previously produced intermittent fever by the injection into human beings of malarial blood.

[2] Unless such an analogy exist in the "Würmchen" found by Gaule in frog's blood.

[3] Laveran's corpuscles have been demonstrated in malarial blood in Algiers, in Italy, and in this country.

PROGNOSIS.—Ordinary or simple intermittent fever as regards immediate danger is not a grave affection. Whenever the disease involves immediate danger, it is to be considered as pernicious, and not therefore belonging under the head of ordinary or simple intermittent fever. An important fact is that an intermittent fever at first devoid of immediate danger may become pernicious. An ordinary or simple intermittent may prove remotely serious, if it continue long or recur frequently, by inducing anæmia, general dropsy, or the malarial cachexia. The remote effects of the disease, however, are rarely in themselves fatal. Death may result from the association of intermittent fever with other affections. It has been supposed that this disease exerts a protective influence against the development of pulmonary tuberculosis. So far from this being true, there is reason to believe that it promotes the development of the affection just named in those predisposed to it. It is not a small calamity to contract intermittent fever, on account of the liability to relapse and to its recurrence in connection with other diseases.

TREATMENT.—For the cure of intermittent fever medicine possesses specifics if any remedies be entitled to this appellation. This statement applies especially to the salts of quinia, of which the sulphate and the bisulphate are almost universally used in this country. The salts of quinia will promptly interrupt the recurrence of the paroxysms of intermittent fever in the vast majority of cases. It is always desirable to arrest the disease as speedily as possible. Its morbid effects are less in proportion as it is quickly arrested and the liability to relapses is diminished. There is no need of preparatory treatment. This position was taken by the author in an article published more than forty years ago.[1] An experience embracing many hundred cases in different climates since the date of that publication has abundantly confirmed the correctness of this position. Aside from the delay in arresting the disease, the measures heretofore employed to prepare the system for quinia or other special remedies were injurious. These measures were mercurial cathartics, emetics, and sometimes bleeding. They are not indicated in the treatment of intermittent fever. A consideration of no small importance, as enforcing an immediate employment of the abortive treatment, is the possibility of an intermittent fever, at first simple or ordinary, becoming after several paroxysms pernicious.

With respect to the time of giving the antiperiodic remedy, my experience has led me to the conclusion at which Bretonneau, Graves, Briquet, Trousseau, Murchison, and others have arrived—namely, that it proves most effective when given as near as possible to the paroxysm which has passed. It is probable that if it be given in the sweating stage the chances of preventing the next paroxysm are greater than if the administration be delayed until after this stage. As regards doses, the most efficient plan is to give the remedy so as to produce evidence of cinchonism as speedily as possible. This object may be accomplished by giving at a single dose the quantity supposed to be required. This quantity for an adult is between ten and twenty grains. A dose of ten or twenty grains is generally tolerated by the stomach as well as smaller doses. An objection, however, to this method is that the quantity estimated as sufficient may not be large enough or it may be larger than is required. A preferable method, therefore, is to give the remedy in smaller but not in small doses, which are to be repeated at intervals of two or three hours until cinchonism is produced. The latter is indicated by tinnitus aurium; and this effect is in general evidence that a sufficient quantity has been administered. For an adult five grains may be given at a dose, and repeated after four hours once, twice, or thrice if necessary for the effect just stated. By this method of treatment in a case of quotidian type the chances that another paroxysm will or will not occur are about even. In a case of tertian type the chances that another will not occur predominate. If another paroxysm occur the same method of treatment is to be repeated.

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[1] Vide *American Journal of the Medical Sciences*, October, 1841. This article contains a series of observations on the employment of quinia in single doses of from twenty to forty grains. At that time this treatment was novel and bold. The author believes that he was the first in this country to make observations with respect to the treatment of intermittent fever by large, single doses of quinia without preparatory measures and given at different periods in the paroxysm.

As regards the form in which the sulphate of quinia may be administered, it is most efficient when given in solution, its solvency being secured by the addition of a minim of the aromatic sulphuric acid for each grain of the salt. Owing to its greater solubility if given in pills, powders, capsules, or wafers, the bisulphate is to be preferred to the sulphate. If, owing to the irritability of the stomach, the remedy be not retained when given by the mouth, it may be given *per enema*. It is readily absorbed from the intestine, and the opinion has been held by some that it acts more efficiently when thus given. According to most observers, however, the quantity when given by the rectum should be larger than when given by the mouth.

The plan of treatment just described is almost invariably successful. If it fail, there is always reason to suspect that the preparation of quinia employed is spurious or adulterated. A difficulty in some cases relates to an intolerance of the remedy. Some persons are affected very unpleasantly by even small doses of a salt of quinia. This intolerance is not infrequently imaginary, but it is sometimes real. In such cases small doses must be given or some one of the remedies to be presently mentioned should be substituted. As a rule, small doses are curative in the cases in which the remedy is tolerated only in small quantities. On the other hand, some persons are affected by the remedy only when full or even large doses are given; and as a rule such persons require larger doses than others. Cinchonism is evidence that the requisite quantity has been given, but this need not be produced except in a slight degree. The coexistence of another affection does not contraindicate the plan of treatment which has been described. In all cases in which intermittent fever is complicated with other affections, the primary object of treatment is to interrupt the paroxysm as speedily as possible. In children and infants the doses are to be less than in adults in proportion to the age. In young children, owing to the difficulty of administration by the mouth, the remedy may be given *per enema*. It may also be employed effectively by inunction, an ointment containing it being rubbed into the axilla and groins and over the abdomen. I have met with a few instances in which moderate doses of quinia have produced menorrhagia, occurring at a period when menstruation was not due.

The hypodermic injection may be resorted to in cases of ordinary intermittents when the remedy is not retained either by the stomach or rectum. This mode of administration has the advantage of economy as regards the quantity of the remedy required, experimental observation appearing to show that the effect is three or four times greater when thus administered than when taken into the stomach. The effect is also more quickly induced. It is, however, in cases of pernicious intermittent that the hypodermic injection of a solution of quinia is of special value, by reason of the certainty and promptness with which cinchonism may be induced by this mode of administration. In simple intermittents the risk of abscesses at the places where the injections are given renders them unadvisable. Binz states that pure amorphous hydrochlorate of quinia is especially well adapted for subcutaneous injection, dissolving in an equal weight of water, this not giving rise to abscesses.

After the paroxysms are interrupted, the remedy should be continued in small doses, between four and eight grains daily, for a considerable period. It is desirable to continue it for several weeks. If anæmia exist a chalybeate should be conjoined. Relapses are prevented by this after-treatment. The diet should be nutritious, and a little wine with meals is advisable. Cathartics are to be avoided. Given before the interruption of the paroxysms, they conflict with that object. If there be constipation, it should be remedied by mild laxatives or enemas.

The germ theory affords a rational explanation of the specific efficacy of quinia in this disease. This drug is destructive to certain low organisms, and its efficacy is due to its action as a parasiticide. This statement will probably apply to other remedies which have a controlling influence over the disease.

Preparations of cinchona other than the sulphate of quinia are more or less effective in arresting intermittent fever. The sulphate of quinidia appears to possess an antiperiodic power not less than that of the sulphate of quinia.. It has less bitterness and is more soluble. The sulphate of cinchonia and cinchonidia are much less effective. Amorphous quinia, called quinoidia, chinoidine, or the precipitated extract of bark, must be given in doses of double the size of the doses of sulphate of quinia.

There are several succedanea of the preparations of cinchona which are capable of arresting the disease. Salicin is successful in a certain proportion of cases. Between 40 and 60 grains are to be given in an intermission. The sulphate of bebeerine is frequently successful, and it has been claimed in behalf of this remedy that it does not produce the unpleasant effects which are sometimes caused by the preparations of cinchona. From 15 to 30 grains are to be given in the apyrexial period. The ferrocyanide of iron, or Prussian blue, I have found an efficient antiperiodic in doses of between 1 and 2 drachms. Patients take it with reluctance, its appearance suggesting the idea of poison. Strychnia and nux vomica often succeed. Chloride of sodium, given to the extent of 8 or 10 drachms in the apyrexia, effects a cure in a certain proportion of cases. Piperine sometimes proves curative. Many practitioners give this remedy in combination with the preparations of cinchona. Arsenic is probably, next to the preparations of cinchona, the most reliable remedy. Fowler's arsenical solution is the form generally employed, given in doses of 5 to 10 drops three times daily to an adult. The muriate of ammonia, in doses of a drachm in the intermission, appears to exert an antiperiodic power. Nitric acid in doses of 8 or 10 drops continued during the intermission and paroxysm, once in six hours, has been found to be efficacious. The iodide of potassium has proved successful in some cases when the disease has resisted quinia. To the foregoing are to be added the eucalyptus globulus and hydrastia. This list might be extended. It is to be borne in mind, in judging of the effect of remedies in this disease, that it tends intrinsically to end after a few paroxysms in a certain proportion of cases. All the known remedies which may be employed as succedanea of the preparations of cinchona are much inferior to the latter. None are to be preferred except in some cases in which from an idiosyncrasy the preparations of cinchona are not tolerated. They may sometimes come into play where the preparations of cinchona cannot be procured, and they have the advantage of being less costly than the latter..

The administration of pilocarpine at or shortly before the beginning of the paroxysm has been found by Dr. Gaspar Griswold to prevent or arrest the paroxysm, and in many instances there is no recurrence.[1] The remedy acts with most certainty if administered hypodermically. The dose for hypodermic use is one-fifth of a grain of the muriate of pilocarpine dissolved in ten minims of distilled water. Given by the mouth, the dose is one-fourth of a grain. For feeble patients these doses should be somewhat reduced. This method of treatment has sometimes succeeded in effecting a cure when the disease had resisted other measures. The efficacy of this treatment establishes the fact that to prevent a paroxysm or to render it abortive may be sufficient for the cure. A full opiate at the beginning of the cold stage often appears to shorten and modify the severity of the paroxysm. It sometimes, indeed, acts as an abortive measure, and may even effect a cure of the disease. I have known a sinapism applied over the whole length of the spine at the beginning of the cold stage to arrest the paroxysm and effect a cure. This result has been obtained by dry cups applied to the spine and by friction with the spirits of turpentine. The method of McIntosh—namely, bleeding in the cold stage—was sometimes effectual, but for obvious reasons it is not to be recommended. A paroxysm appears to be sometimes warded off by taking to the bed before the hour when it is expected, and keeping up warmth of the body by bottles of hot water or other means. External warmth is useful in the cold stage, and stimulants may be given internally if tolerated by the stomach. The internal use of chloroform has been highly recommended. Given in drachm doses (either alone, followed immediately with cold water, or suspended in mucilage) at the beginning of the paroxysm, it has been found to arrest the chill and induce a refreshing sleep, from which the patient awakens without prostration. The duration and severity of the paroxysm appear to be lessened by this treatment.

During the hot stage relief is procured by sponging the body with cold or tepid water. Pieces of ice, ice-water, or carbonated water may be taken freely. During the sweating stage comfort is promoted by wiping the body with warm flannel and changing the linen and bedclothes.

Anæmia and general dropsy, occurring as sequels, claim tonic remedies, into which the preparations of cinchona and iron should enter, with nutritious diet and other hygienic measures calculated to restore the strength and invigorate the body. Enlargement of the spleen is to be treated with the sulphate of quinia or other preparations of cinchona, and a belladonna plaster or anodyne embrocations applied over the enlarged organ. If these measures fail, the iodide of potassium may be tried. Faradization and the cold douche applied to the region of the spleen have been found useful.

The preparations of cinchona and other antiperiodic remedies are efficacious as prophylactics. Taken in small doses daily by persons exposed to malaria, they ward off attacks of intermittent fever and they prevent relapses. There is no foundation for the popular notion that the prolonged use of the sulphate of quinia in small or moderate doses is prejudicial to health. I have known an instance in which two or three grains had been taken daily for twenty years, the person having found by experience that by this means he was exempt from relapses of intermittent fever. Other means of prophylaxis have been already stated in treating of the causation.

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[1] Vide *N. Y. Med. Journal*, Aug., 1880.

Pernicious Intermittent Fever.

Simple or ordinary intermittent fever is not attended with immediate danger; but the disease is sometimes presented in forms more or less dangerous, and it may prove fatal within a few days or hours. These forms of the disease are to be considered apart from simple or ordinary intermittent fever, and collectively they constitute a variety of the disease called *pernicious*, *malignant*, and *congestive*. Of these names the first is commonly adopted by writers. The term congestive, although much used in the Southern and Western portions of this country, is objectionable, as implying that the primary or essential pathological state is congestion—a doctrine which probably is incorrect.

Pernicious intermittent fever occurs in isolated cases wherever the simple or ordinary variety of the disease prevails; but, happily, such cases are extremely rare. It, however, prevails at certain epochs in malarial regions. Of the cases of intermittent fever which occur at these epochs, the proportion in which the disease is pernicious is more or less large. In some seasons the fatality from the disease is very great, and it constitutes one of the most formidable of the maladies which the physician has to encounter. In this country it has prevailed chiefly in the Southern and Western States. In view of the great danger attendant on pernicious intermittent fever, and of the fact that the success of prompt and efficient treatment is perhaps more conspicuous in this than in any other malady involving an equal amount of danger, the disease is one of great importance. It will, however, suffice to notice the symptomatic features by which the different forms are distinguished from simple or ordinary intermittent fever, together with the therapeutical indications.

Different observers agree that in many, if not in most, cases a pernicious paroxysm is preceded by one or more paroxysms not characterized by any symptoms denoting gravity of disease; hence the vast importance, especially whenever cases of the pernicious variety are known to have occurred, of resorting in every case at once to prompt and efficient measures to interrupt the paroxysms. In a certain proportion of cases, however, the pernicious character of the disease is manifested in the first paroxysm, and death may take place in that paroxysm. Drake states that the pernicious character is sometimes foreshadowed by some slight anomaly in the first paroxysms, such as "a partial numbness or coldness of the great toes instead of a regular chill, or a disposition to sleep at the access of the paroxysm." According to this author, in the cases in which the pernicious character is gradually developed the cold stage in the early paroxysms is not strongly marked, the hot stage is imperfectly developed, and in the intermissions the patient may be pursuing his usual avocations. Both patient and physician are liable to be thrown off their guard by the mildness of the paroxysms preceding the one which is pernicious.

The symptomatic features of pernicious paroxysms vary much in different cases; and on account of these diversities this variety of the disease may be considered as embracing several forms. More or less complete coma characterizes some cases, and these cases are distinguished as *comatose*, *soporose*, or *apoplectic* intermittents. Other cases are characterized by delirium preceding coma. The delirium is sometimes active, the patients requiring to be restrained. Examples have fallen under my observation. Epileptiform convulsions occur in some cases. Intense jaundice appears early in the so-called *icteric* variety. A form called *algid* is characterized by notable reduction of temperature, the extremities becoming cold as marble or the coldness being like that of a cadaver. Profuse sweating characterizes some cases. Vomiting and purging are not infrequently prominent symptoms, leading to a state of collapse like that in cases of epidemic cholera. The secretion of urine is sometimes suppressed, and albuminuria is not infrequent. In a form which may be called *asthenic* there are great restlessness and prostration, with a notably small, feeble, and irregular pulse. The development of well-marked febrile movement, with a full, regular pulse and heat of skin, is evidence that the patient will struggle through the paroxysm. The danger to be apprehended from another paroxysm is proportionate to the severity of that from which the patient emerges with safety. If the latter have involved very great risk, the next will be likely to prove fatal. The prostration in the intermission is in proportion to the severity of the paroxysm.

Hæmaturic malarial fever, which is one of the most important forms of pernicious fever, has already been considered (p. 924).

The DIAGNOSIS of all the forms of pernicious intermittent fever is aided by the fact that as a rule the dangerous paroxysm is preceded by at least one paroxysm, and it may be two or three paroxysms, devoid of any symptoms denoting danger, and by the fact that the pernicious paroxysm occurs in accordance with the type of the disease. In the forms characterized by coma, delirium, or convulsions cerebral meningitis and uræmia are to be excluded. The former is excluded by the absence of symptoms of cerebral inflammation exclusive of those just named, and the latter is excluded by an examination of the urine. Microscopical examination may show the presence in a drop of blood of black pigment and of the peculiar bodies described under the head of Causation in the preceding article. Enlargement of the spleen also has diagnostic significance.

Of the pathology of the several forms of pernicious as compared with simple or ordinary intermittent fever, all that can be said, with our present knowledge, is, that the essential morbid condition existing in the latter variety in a degree not involving immediate danger exists in the former variety in such intensity as to prove highly dangerous. Intermittent fever is not the only affection which without any essential change in the nature of the disease is in some cases extremely mild and in other cases extremely formidable. Scarlet fever may be cited as another striking example. The difference as regards symptomatic phenomena and gravity between pernicious and simple or ordinary intermittent fever is not explained by lesions found after death.

Pernicious intermittent fever is often preventable, and many lives are saved by timely efficient TREATMENT. It is doubtless prevented by promptly interrupting paroxysms before they assume a pernicious character. In seasons when pernicious cases prevail there is much risk of lives being sacrificed by the delay in arresting the disease incident to the employment of the so-called preparatory measures of treatment. In these seasons especially the curative treatment should be resorted to at once in all cases of intermittent fever, and the disease should be arrested as speedily as possible. When the disease has become pernicious there are two great objects of treatment. One of these is to carry the patient safely through an existing paroxysm; the other is to prevent another paroxysm.

With reference to the first of these objects, the indications differ according to the differences of form which pernicious paroxysms assume and according to the symptoms whatever may be the form. Enfeebled action of the heart always calls for stimulants, alcoholic, ethereal, and aromatic, severally or collectively. In many cases the danger of the disease is manifested chiefly by the symptoms referable to the circulation. In these cases the all-important indication is to strengthen the heart's action. Coma, unaccompanied by augmented force of the circulation and increased heat, does not call for bloodletting. Bloodletting has been employed in the cold stage of pernicious paroxysms of intermittent fever. That patients have recovered under this treatment by no means proves that it is devoid of danger. It is admissible only when the symptoms denote active cerebral congestion. Coldness of the surface is an indication for the external application of heat by means of warm blankets, bottles of hot water, etc., and for internal stimulants. Vomiting and purging are to be restrained by opiates given by the mouth or rectum. Opiates are also indicated by restlessness, delirium, and convulsions. In connection with the latter the kidneys are to be interrogated with reference to the existence of uræmia. Emetics given at the beginning of the paroxysm, although they have been much used, are of doubtful utility, and cathartics should never be given. Chloroform in drachm doses deserves further trial as a means of abridging the cold stage. The doses should be repeated until the hypnotic effect of the remedy is obtained. Pilocarpine, administered hypodermically, will become an invaluable remedy if it prove as successful in arresting a pernicious paroxysm as it is in cases of a simple intermittent fever.

The second object—namely, to prevent another paroxysm—should enter into the treatment before the paroxysm, attended with more or less immediate danger, is ended. With reference to this object nothing can take the place of the preparations of cinchona. The succedanea of these preparations are not to be relied upon if the latter can be obtained. The sulphate, bisulphate, or the muriate of quinia should be given during the paroxysm, and given in larger doses than in cases of ordinary intermittent fever, in view of the vastly greater importance of arresting the disease, and because the tolerance of the remedy is often much greater. It is to be given during the paroxysm, not for its immediate effect, but to render more sure the prevention of another paroxysm. A scruple of the sulphate of quinia may be given at once by the mouth to an adult, and a larger quantity if given by the rectum. After three or four hours, if there be no evidence of cinchonism, the dose may be repeated. It is to be continued after the patient has emerged from the paroxysm in doses of between 10 and 20 grains, the intervals between the doses being sufficient to indicate the effect of each dose. Cinchonism should be produced and maintained during the intermission without reference to the quantity required; but to produce this effect the enormous doses which have sometimes been given are never necessary. Promptness and boldness are important, but excessive cinchonism is not advisable, and it is to be borne in mind that permanent blindness and deafness have resulted from the use of quinia in enormous doses. By pursuing the plan just stated the remedy may be employed as freely as may be necessary to obtain to the fullest extent its remedial influence, without incurring much risk of injury from its toxical effect. In a case in which it was considered that the life of the patient depended on the prevention of another paroxysm, 120 grains of quinia were given in divided doses in about twenty-four hours. The paroxysm was prevented, but suddenly, without having previously had symptoms of much cinchonism, he became completely blind and almost completely deaf. As may be imagined, great anxiety was felt; but, happily, at the end of eight hours both the blindness and deafness had disappeared, and the patient recovered without any further untoward symptoms.

The administration of quinia by means of hypodermic injections has been found efficient in cases of pernicious intermittent characterized by coma and inability to swallow. It may be resorted to when, from irritability of the stomach, the remedy will not be retained if given by the mouth.

The nostrum called Warburg's tincture is highly recommended by many East Indian physicians in the treatment of pernicious as well as of simple intermittent fever.

The pernicious paroxysm having been experienced, at the time when another paroxysm is to be expected, provided the type be known, and on the second and third day if the type be not ascertained, the patient should be in bed at the hour when the recurrence is likely to take place, and the body should be kept warm by artificial heat and hot, stimulating drinks. An opiate will render more sure the prevention of the dreaded paroxysm.

After the interruption of the paroxysms some one of the preparations of cinchona should be continued in tonic doses, with a nutritious diet and other restorative measures. After both the pernicious and simple variety of intermittent fever, if the malarious cachexia remain in spite of appropriate measures of treatment, removal to a situation without the sphere of the malarious influence during the season when this influence is rife is to be advised.

CHAPTER V.

PERIODICAL FEVERS (CONCLUDED).

Simple Remittent and Typho-malarial Fever: Anatomical Characters; Clinical History; Pathological Character and Causation; Diagnosis; Prognosis; Treatment.—Pernicious Remittent Fever.—Yellow Fever: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment; Prevention.

A FORM of periodical fever is distinguished by the occurrence of remissions instead of intermissions, and hence is called *remittent fever*. It is also known as *bilious remittent* or *bilious fever*. The term bilious is superfluous, and, so far as it has any significance, tends to perpetuate a pathological error. Between simple remittent and intermittent fever there is a close relationship. They are mutually convertible into each other, they undoubtedly involve the same special cause, and they are controlled by the same specific remedies. The propriety of recognizing remittent as distinct from intermittent fever is denied by some writers. Bouillaud calls it a "real nosological superfœtation"! There is, however, a practical convenience in considering the two forms as separate affections, admitting that they are essentially identical.

Remittent fever and typhoid fever may be associated. Not only may the former present typhoid phenomena or the typhoid state, but the two diseases occur not infrequently in combination. This statement embodies an opinion held for a long time by practitioners in malarial sections of this country, and inculcated especially by Prof. Dickson. Dr. Drake also maintained this opinion, calling the combined affection remitto-typhous fever.[1] I have for more than thirty-five years advocated this opinion in medical teaching.[2] More recently it has been proposed to employ a name denoting the union of the two affections, and the term *typho-malarial fever* has been introduced by Dr. Joseph J. Woodward.[3] I shall adopt this name, and consider remittent and typho-malarial fever conjointly. The facts which show the blending of remittent and typhoid fever will be noticed under the heads of Pathological Character and Causation.

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[1] Vide *The Principal Diseases of the Interior Valley of North America*, Second Series, Philadelphia, 1854, p. 557.

[2] Vide article by the author, in the *Buffalo Med. Journal*, 1857.

[3] Vide *Outlines of the Chief Camp Diseases of the United States Army, etc.*, by Joseph Janvier Woodward, M. D., Assistant Surgeon U. S. Army, etc., 1863.

Simple Remittent and Typho-malarial Fever.

By the term simple remittent fever is meant remittent not associated with typhoid fever and not accompanied by symptoms of gravity entitling it to be called pernicious. Pernicious remittent fever will claim a brief separate notice. Typho-malarial fever will be considered in conjunction with simple remittent fever.

Anatomical Characters.—The characteristic lesion of remittent fever consists in the presence of dark pigment-granules in the blood and in various organs of the body. The parts chiefly affected are the spleen, the liver, and the marrow of the bones. When contained in the circulating blood the pigment may be found in the blood-vessels of any part of the body. The presence of the pigment in the blood constitutes the condition called *melanæmia.* The pigmentation of organs is sometimes called *melanosis.* Melanæmia and melanosis attend remittent fever, pernicious malarial fevers, long-continued intermittent fever, and sometimes the malarial cachexia. The degree of pigmentation depends upon the duration and the severity of the malarial affection. It is therefore most intense in connection with the severe malarial infection of warm climates.

During a paroxysm of malarial fever the spleen is enlarged from hyperæmia. This swelling at first subsides during an intermission, but under the influence of prolonged malarial infection the organ becomes permanently enlarged, constituting the so-called ague-cake. The color of the swollen spleen varies between dark-red or brownish- and bluish-black in proportion to the amount of pigment present. The consistence is soft when the enlargement is recent or when death has occurred during a febrile paroxysm. If there have been no recent attacks the consistence is firm. The swelling at first is due to hyperæmia; and subsequently there is hyperplasia of the cellular elements of the spleen, with new formation and thickening of its connective-tissue framework. The pigment is in the form of granules and of larger masses, and is contained chiefly within cells and within the hyaline bodies described on p. 1002. In recent cases its color is reddish-brown, and later it becomes black. The pigment is in the splenic pulp, and is also accumulated about the blood-vessels, especially around the veins. It is contained also within the splenic veins. Little or no pigment is found in the Malpighian bodies in most cases. The cells containing the pigment are chiefly the lymphoid cells, which constitute the principal cellular elements of the spleen. Pigment is also contained in larger and more granular cells, and in long fusiform cells which are considered to be endothelial cells of the veins.

The liver in most cases is enlarged, but in cases of long duration it may be atrophic. The consistence is usually more or less softened. The organ presents a bronze, chocolate, or slate color. It is called the bronzed liver and the pigment liver. It usually is hyperæmic. The pigment-granules are found both in the interlobular tissue and within the lobules. They are enclosed chiefly within cells. They exist partly within the blood-vessels and partly in the extravascular tissue. The pigment is chiefly found within lymphoid cells, but also in fusiform, and sometimes in stellate, cells. The fusiform cells are considered by many writers to be derived from the spleen, as similar cells exist there and as the same cells are found in the blood of the splenic and portal veins. The melanæmic pigment does not exist within the hepatic cells. These cells, however, usually contain a considerable amount of yellowish-brown pigment derived from the biliary coloring matters. According to Kelsch, there is an increased number of lymphoid cells in the interlobular tissue. This writer also maintains that a form of cirrhosis may be produced by malarial affection of the liver.

Pigment is found in the marrow of the bones, both outside of and within the blood-vessels. It is contained in lymphoid cells. Pigmentation of the brain forms a marked lesion in some cases of melanæmia. The change in color is most evident in the gray matter, which acquires a dark-gray or slate color contrasting markedly with the white matter. When the change is intense the white matter also has a dirty grayish tint and presents many puncta vasculosa. In some severe cases capillary hemorrhages have been found in large number. These extravasations have been attributed to plugging up of the small cerebral blood-vessels with pigment. In melanæmic brains the pigment is present chiefly within the small blood-vessels. It is also found in the walls of the vessels and in the perivascular spaces. The pigment does not exist in large amount in the brain unless it be also present in the circulating blood. If pigment be present in the kidney, it is met with chiefly in the glomeruli. The accumulation of pigment in other organs than those named is not great. It may be found in lymphatic glands, in the spinal cord, in the lungs, in the pancreas, in the skin, in mucous membranes, etc. In these situations it is contained within the blood-vessels.

The characters of the pigment in the blood, the source of the pigment, and the relations between the pigmentation of organs and the presence of pigment in the blood are subjects which have been discussed in the article on Melanæmia in Part I. (p. 78).

After death from remittent fever more or less congestion and inflammation of the stomach and of the intestine are frequently found. The mucous membrane of these parts may present ecchymoses. The solitary glands and Peyer's patches may be swollen. The latter often present the shaven-beard appearance.

The same parenchymatous and hyaline degenerations which have been described under the head of Typhoid Fever have also been found after death from severe malarial fevers.

Acute nephritis, dysentery, and pneumonia are rare complications of this disease.

In typho-malarial fever, in addition to appearances characteristic of malarial disease, the abdominal lesions which are essentially those characteristic of typhoid fever are found after death.[1]

CLINICAL HISTORY.—Under this head I shall present a brief sketch of simple remittent fever as regards especially its distinctive features, and afterward of typho-malarial fever.

Simple remittent fever often begins abruptly, but in a certain proportion of cases it has a brief forming stage, the prodromes being the same as those attending the development of other fevers. The febrile career begins with a chill more or less pronounced, either with or without rigor. During the chill, as in the cold stage of an intermittent paroxysm, the temperature of the body is actually raised, as shown by the thermometer in the axilla. The attack is oftener before than after mid-day. Febrile movement follows, varying in intensity in different cases, accompanied with cephalalgia, pain in the loins, etc. The thermometer in the axilla shows an increase of temperature of between 2° and 10° above the range of health. The febrile movement continues unabated for six, twelve, eighteen, twenty-four, and even forty-eight hours, and then notably subsides; the pulse falls in frequency, the skin becomes moist, the patient is comparatively comfortable, and may obtain refreshing sleep. The febrile movement, however, does not disappear; there is not apyrexia; and there is a remission, not an intermission. The temperature in the remission, as compared with the exacerbation, is notably decreased, sometimes falling nearly to the normal standard. The remission is likely to occur during the night. The degree of abatement of febrile movement varies in different cases, sometimes approximating to apyrexia, and sometimes the remission being slight. The duration of the remission varies between two or three hours and one or two days. At the end of the remission another chill may occur, but it is often wanting, and if present is generally slight. The febrile movement is renewed with as much or more intensity than before the remission. A series of remissions may ensue, recurring in regular succession, and, as regards the intervals, corresponding to the quotidian, tertian, or frequently the double tertian type of an intermittent fever. In a case under my observation the double quotidian type was represented by the remissions; that is, two distinct exacerbations occurred daily. The remissions sometimes recur irregularly. Different cases differ as regards the number of remissions. There may be but a single remission, or, on the other hand, remissions may occur regularly through the whole course of the disease. The latter is rare. In general the remissions cease to be distinct after a few days, and sooner or later the fever becomes continuous. The febrile career ends during the second or third week. It eventuates in intermittent fever in a certain proportion of cases. In some cases the disease is preceded by intermittent fever, remissions taking the place of the intermissions. During the remissions there is a marked abatement of the temperature of the body, the temperature during the exacerbations being notably increased. In this respect remittent fever differs from typhoid fever exclusive of the period of defervescence in the latter disease. The fall in temperature, as shown by the thermometer, may denote remissions which are not rendered apparent by the general symptoms.

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[1] Woodward at first believed that the intestinal ulcers of typho-malarial fever present certain peculiarities which distinguish them from those of ordinary typhoid fever. Extended studies led him to abandon this view.

Early in the fever nausea and vomiting generally occur, and are frequently prominent symptoms, continuing more or less throughout the febrile career. The matter vomited is greenish or yellowish in color. Pain or uneasiness is usually referred to the region of the stomach, and there is tenderness on pressure over the epigastrium. Diarrhœa, tympanites, and iliac tenderness are wanting. Sordes is rarely observed. Delirium is rarely a prominent feature, and when present it is due to the intensity of febrile movement. The urine is scanty, its coloring matter is increased, and its specific gravity is high. It is rarely albuminous. Jaundice occurs in a certain proportion of cases.

If simple remittent fever be protracted, certain symptoms denoting the typhoid state may appear—namely, low delirium, sordes, subsultus tendinum, etc. These symptoms are not sufficient to show the union of typhoid fever and remittent fever. They are liable to occur in all fevers and in various other affections.

In typho-malarial fever the symptoms distinctive of typhoid fever are intermingled with those of periodical fever. The symptoms referred to are those connected with the abdominal lesions of typhoid fever—namely, diarrhœa, tympanites, and iliac tenderness. These symptoms become more or less marked during the second week of the career of the disease. The characteristic eruption of typhoid fever may be observed. The ataxic symptoms belonging to the typhoid state—namely, low delirium, deafness, subsultus tendinum, etc.—occur more frequently, earlier, and in a more marked degree than in connection with simple remittent fever. Enlargement and suppuration of one or both parotids are sometimes observed in cases of typho-malarial fever.

This form of fever is preceded by a forming stage longer than in cases of simple remittent fever. The duration of the febrile career is longer and the gravity of the disease is greater. Perforation of the intestine is liable to occur. Bronchitis is a common complication, and pneumonitis is developed oftener than in simple remittent fever. Convalescence is likely to be protracted in cases of typho-malarial fever. Distinct malarial paroxysms often occur during convalescence. In different cases of the disease the phenomena of typhoid fever and of periodical fever are intermingled in varying proportions, in some cases the periodical and in other cases the typhoid phenomena preponderating. In proportion as the phenomena of the one or the other disease predominate cases will approximate, on the one hand, to simple remittent, and on the other hand to typhoid fever.

PATHOLOGICAL CHARACTER AND CAUSATION.—Simple remittent fever, as regards its essential pathological character, does not differ from intermittent fever. Both are due to the special poison known as malaria. True remittent fever is never contracted elsewhere than in malarial situations. The distinction between the two diseases is nosological rather than pathological.

Typho-malarial fever is caused by the combined action of malaria and the special cause of typhoid fever. Practitioners in malarial situations have been accustomed to say that remittent becomes converted into typhoid fever. This mode of expression is not accurate. There is not a metamorphosis of the one disease into the other, but a combination of both diseases, the phenomena of the one or of the other disease predominating in different cases. Typho-malarial fever is not a distinct type of fever. It is, however, of great importance to recognize the combination of typhoid and of malarial fever.[1]

Cases of typho-malarial fever occur in most if not in all malarial regions. This fact shows that the special cause of typhoid fever is not held in abeyance by the prevalence of malaria, and produced only after the latter disappears. The extinction of malaria is followed by cases of unmixed typhoid fever, and hence it has appeared as if the latter follows in the wake of the periodical fevers. The typho-malarial fever prevailed largely among the United States troops in certain situations during the recent civil war, especially in the Army of the Potomac in the Peninsular campaign of 1862, when it was known as the *Chickahominy fever*. It was called elsewhere during the war *camp fever*.

Simple remittent and typho-malarial fever prevail especially in the autumnal season. Persons of all ages are liable to be affected with simple remittent fever. There is no evidence that this fever is ever contagious, or that the malarial poison producing it is portable. Its occurrence affords no protection against its recurrence.

DIAGNOSIS.—Simple remittent fever is readily discriminated from simple intermittent fever by the occurrence of notable remissions instead of intermissions. The thermometer in the axilla may show the occurrence of these when they are not distinctly manifested by symptoms aside from those relating to the temperature of the body. A notable variation in temperature at different periods of the day in the early part of the disease, the mercury rising to a high point at one period and falling nearly within the range of health at another period, warrants the exclusion of typhoid fever. Exclusive of remissions the differential diagnosis involves the following points:

The abdominal symptoms of typhoid fever are wanting in simple remittent fever, and in place of these are the gastric symptoms distinctive of the latter—namely, nausea and vomiting, with tenderness over the epigastrium. The ataxic symptoms of the typhoid state are either wanting in simple remittent fever or they occur at a later period. The typhoid eruption is wanting. The access is much oftener abrupt and accompanied by a more pronounced chill. Paroxysms of intermittent fever are liable to occur at the close of a remittent fever. Finally, remittent fever is developed only in malarial districts or in persons who have been exposed to malaria. There can be no question as to this differential diagnosis in cases of disease occurring in a situation where malaria is known not to exist and when the patient is known not to have been within a malarial influence.

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[1] Vide article by Woodward on "Typho-malarial Fever," in the *Transactions of the International Medical Congress*, Philadelphia, 1877, p. 305.

Typho-malarial fever is discriminated, on the one hand, from simple remittent fever by the characteristic events of typhoid fever—namely, the abdominal symptoms, the eruption in some cases, the earlier occurrence of ataxic phenomena, epistaxis, and occasionally intestinal perforation. It is discriminated, on the other hand, from unmixed typhoid fever by the characteristic events of periodical fever—namely, remissions, gastric symptoms, jaundice, and the termination in some cases in intermittent fever. The diagnosis of typho-malarial fever by means of positive symptoms may be difficult, especially if the abdominal symptoms and the eruption of typhoid be absent, as is frequently the case. The diagnosis is to be based on the presence of more or less of the symptoms belonging to malarial and to typhoid fever, and on negative facts which serve to exclude other fevers.

PROGNOSIS.—Simple remittent fever in itself does not involve danger to life. In fatal cases the termination is due to coexisting affections. Of course it is to be understood that cases of pernicious remittent fever are not included under the head of simple remittent fever. An attack of remittent fever leaves behind it a liability to subsequent attacks of intermittent fever, and is liable to be followed by the sequels of the latter disease—namely, enlarged spleen, anæmia, and general dropsy. This disease differs much in severity at different times and places and in different cases at the same time and place. As a rule, it is likely to be more severe in tropical than in temperate climates.

Typho-malarial fever is a much graver affection than simple remittent fever, and it is certainly not less grave than typhoid disconnected from malarial fever. Like the latter, the rate of mortality differs at different times and places, owing to differences as regards the intrinsic tendency of the disease and a variety of circumstances. Data for determining the average death-rate are not available, as it has not been customary to separate cases of this disease from cases, on the one hand, of simple remittent fever in which the typhoid state occurs, and on the other hand of typhoid fever without the combined action of malaria.

TREATMENT.—The first and leading object in the treatment of simple remittent fever is the arrest of the disease by antiperiodic remedies, of which the preparations of cinchona are by far the most reliable, the sulphate, the bisulphate, and the muriate of quinia being the preparations to be preferred. As soon as the character of the disease is determined by the occurrence of a remission, one of these salts of quinia should be given in a full dose—namely, 10 or 20 grains to an adult. The remedy should be continued in doses of 5 or 10 grains after intervals of from two to four hours until it produces slight deafness or ringing in the ears, suspending the remedy when these manifestations of cinchonism appear. If other preparations of cinchona be used, they are to be given in equivalent doses in the same manner. If the remedy be not tolerated by the stomach, it should be given *per enema.*

If cinchonism be not produced during the remission, the remedy may be continued during the exacerbation of fever. This is preferable to waiting for another remission. In a case in which the practitioner is satisfied that a remission has already occurred—in other words, whenever the diagnosis is clear—the remedy should be given at once, notwithstanding the intensity of the febrile movement, without waiting for a remission. Time need not in any case be lost in resorting to cathartics or other measures preparatory to the administration of an antiperiodic remedy. In short, the treatment is essentially the same as in cases of intermittent fever. This plan of treatment will succeed in a large proportion of cases in promptly arresting the disease.

Aside from the treatment just stated, palliative measures are to be employed according to the indications in individual cases. The indications will relate to pain in the head, nausea and vomiting, vigilance and restlessness, heat and dryness of the skin, etc. The same measures are to be addressed to these symptoms as when they are present in other fevers. It is unnecessary to consider these measures in this connection. The treatment during convalescence is the same as after other fevers, except that a preparation of cinchona should be employed for some time in conjunction with a chalybeate, as after an attack of intermittent fever.

The plan of treatment which has been sketched does not embrace measures heretofore considered important in this disease—namely, emetics, cathartics, bleeding, and the use of mercury. Emetics are contraindicated by the condition of the stomach. Cathartics are not indicated by the disease *per se*. They are required only to overcome constipation, and generally simple enemata or mild laxatives will suffice for this object. Bleeding is called for only by active cerebral congestion or intense febrile movement with augmented power of the heart's action, the patient being robust and plethoric. Under the latter circumstances sedative remedies, saline laxatives, and cold to the surface will generally be sufficient without the abstraction of blood. The external use of cold water is highly useful when the skin is notably hot and dry. The wet sheet may be employed, as in cases of typhoid fever. Opium, given in pretty full doses early in the disease, appears sometimes to exert an effect beyond that of a palliative by inducing a more marked remission. The pathological views which formerly led practitioners to employ mercury freely in this disease are not tenable, and clinical observation affords no ground for regarding this remedy as specially indicated.

In typho-malarial fever the periodic element claims the treatment indicated in simple remittent fever. The object is to eliminate this element by means of antiperiodic remedies. Aside from this object, the hygienic and medicinal measures indicated are the same as in cases of unmixed typhoid fever. To discuss these measures here would be to repeat what has been presented in the chapter in which the treatment of typhoid fever is considered. The reader is therefore referred to that chapter.

It is highly important to recognize the existence of the malarial element in cases of typho-malarial fever with reference to continuing antiperiodic remedies throughout the course of the disease. I have known death to occur from a paroxysm of intermittent fever when the symptoms had denoted convalescence from the typhoid fever, the autopsy also showing that restorative processes were taking place in the intestinal ulcers. Probably in this case quinia in antiperiodic doses. continued throughout the course of the fever, would have prevented the fatal paroxysm.

Pernicious Remittent Fever.

A brief notice of pernicious remittent fever will suffice. This fever is distinguished as pernicious under circumstances similar to those which constitute grounds for the application of the same term to intermittent fever—namely, circumstances giving to the disease unusual gravity and danger. This variety of the disease is also called malignant and congestive. Pernicious remittent fever presents the same diversities as regards symptomatic phenomena as pernicious intermittent fever. In cases in which death takes place quickly it is impossible to determine whether the disease be intermittent or remittent; and a fatal result may take place before either a remission or an intermission occurs. The occurrence of an intermission or of a remission is the chief differential point. The two forms of disease, in a pathological view, are essentially the same, and they claim the same treatment. The importance of pernicious remittent fever is by no means to be measured by this brief notice of it; but inasmuch as all that is of practical importance in relation to this disease is embraced in the consideration of pernicious intermittent fever, the reader is referred to that portion of the preceding chapter which treats of the latter.

Remittent fever, especially the pernicious variety, has received in different parts of the world a variety of names denoting its geographical relations, such as Walcheren fever, African fever, Hungarian fever, jungle fever, lake fever, etc.

Yellow Fever.

The fever to be now considered has received a great variety of names, but it is at this time everywhere known as yellow fever—a name the significance of which is derived from the frequent occurrence of yellowness of the surface of the body. Although the name relates to a symptom not constantly present, and occurring occasionally in other fevers, it has the merit of not involving any hypothesis concerning the nature or causation of the disease.

ANATOMICAL CHARACTERS.—The morbid appearances which are most frequent are situated in the liver, in the stomach and intestine, and in the kidneys. The liver may be somewhat enlarged, but it generally presents no marked change as regards size. It is pale in color rather than congested. Its consistence is often diminished. The most characteristic change is the yellow color which the organ presents in most (although not in all) cases. The change in color may affect the whole liver or may exist in a larger or smaller number of scattered patches. The abnormal coloration is due to the presence of fat, as was proven by Alonzo Clark in 1853. The fat is in the form of large and small drops in the liver-cells. According to some observers, fat is found also in the interstitial tissue. The process is to be regarded as an acute fatty degeneration of the liver.[1] Some writers describe an actual disintegration of some of the hepatic cells in yellow fever. The yellow color may be due in part to jaundice of the liver. The combination of bile-staining and of fatty metamorphosis produces the so-called saffron-colored liver. Small ecchymoses are often found upon the surface of the liver. Schmidt describes the occasional presence in the liver, as well as elsewhere, of reddish-brown stains which he attributes to extravasations of hæmoglobin.[2]

The mucous membrane of the stomach is usually swollen, softened, and congested. Small hemorrhages are found in the mucous and submucous tissues. The stomach often contains a dark, bloody fluid, which is of the same nature as the black vomit to be described under the head of the Clinical History. The cells of the gastric tubules undergo parenchymatous degeneration. There may be an extensive accumulation of lymphoid cells in the submucous and other coats of the stomach. Changes similar to those of the stomach are found in the upper part of the small intestine, and they may extend downward to a variable degree. The upper part of the intestine contains in some cases black matter similar to that found in the stomach. The Peyerian and solitary glands of the intestine present no morbid alteration.

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[1] The presence of large oil-drops is not a sufficient criterion of fatty infiltration in distinction from fatty degeneration. (Consult Part I. p. 53, and the article on Fatty Liver, p. 611.) It is incorrect to regard the hepatic changes in yellow fever as comparable in degree or extent with those in acute yellow atrophy of the liver. There is still less ground for the assumption that the essential symptoms of yellow fever are referable to an acute parenchymatous or fatty degeneration of the liver (Liebermeister, Schmidtlein).

[2] Schmidt, "The Pathology of Yellow Fever," *New York Medical Journal*, 1879, vol. xxix. p. 128.

The kidneys usually are enlarged and the seat of parenchymatous degeneration. They often contain small extravasations of blood. Acute diffuse nephritis is a frequent accompaniment of yellow fever. In this condition the cortex is swollen and presents a yellowish-white color mingled with congested and hemorrhagic portions. The epithelial cells of the convoluted tubes contain fatty molecules. Red blood-corpuscles are found in the tubes as well as in the intertubular tissue. Collections of emigrated white corpuscles are met with between the tubes. Miliary abscesses have been seen exceptionally in the kidneys of those who have died of yellow fever.

It is a remarkable fact that in a disease so acutely infectious as yellow fever the spleen as a rule undergoes no enlargement. The lungs are often the seat of hypostatic congestion. In many cases hemorrhagic infarctions have been found in the lungs. The pleura as well as the pericardium and other serous membranes frequently presents small ecchymoses. Parenchymatous and fatty degeneration of the myocardium has been frequently seen in some epidemics. The brain and its membranes present nothing distinctive. No changes have been discovered in the blood other than those belonging to most of the essential fevers. The attempts to discover a specific organism in the blood and tissues have thus far met with no success.[1]

CLINICAL HISTORY.—An attack of yellow fever usually is abrupt. In a minority of cases it is preceded for one, two, or three days by languor, lassitude, loss of appetite, pain in the head, and chilly sensations. The attack is generally denoted by a chill, with or without rigor, the chill in most cases being of moderate intensity. Fever follows, differing in intensity in different cases. The temperature quickly reaches its maximum, which varies between 102° and 110° F. The pulse seldom exceeds 100. The tongue is moist and more or less coated. Vomiting occurs in a certain proportion of cases early, but it rarely is a prominent symptom until a later period. Tenderness on pressure over the epigastrium is more or less marked. The bowels, as a rule, are constipated. Cephalalgia, the pain referred especially to the supraorbital region, is usually present, and is sometimes intense. Frequently pain in the loins and pain in the lower limbs, especially in the calves of the legs, is a prominent symptom. This fever bears an analogy to smallpox in the frequent prominence of lumbar pain. The mind usually is clear, but occasionally delirium is manifested, rarely violent and active, and sometimes mirthful. The eyes are reddened, irritable, watery, or tearful. This is quite constant, and so marked as to constitute a diagnostic feature. Otherwise, the organs of sense are not disturbed. Generally there are no pulmonary symptoms.

The pyrexia continues for a period varying between a few hours and three days. Then follows either a marked abatement or entire cessation of fever. A remission is said to take place; but this term is not always accurate, inasmuch as there may be complete apyrexia, and in a certain proportion of cases there is no return of fever. The condition following the febrile paroxysm has been called "the state of calm." This is reckoned as the second stage of the disease, the first stage embracing the period of fever, the latter being called the "febrile period," the "stage of excitement," or the "paroxysm." The first stage may be regarded as constituting the disease proper, and whatever may follow as sequels of the disease. The disease is then a fever with a single paroxysm. In mild cases convalescence takes place immediately after this stage.

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[1] The published observations of Freire and of Cremona upon this point are not of a nature to inspire confidence in their conclusions.

In grave cases the cessation of fever is often deceptive. The pains, etc. may cease, and the patient may feel as if convalescence had begun; but after a period varying between a few hours and twenty-four or longer there are new symptoms more distinctive of the affection than those during the febrile paroxysm. The pulse sometimes falls below the normal frequency, and it has been observed to fall as low as 40, and even 30, per minute. It is sometimes small and weak, and sometimes vibratory. It is always notably compressible, and has been called a "gaseous pulse." The surface is usually cool, especially of the parts of the body which are exposed. In the few cases which I have noted capillary congestion of the skin existed in a marked degree. Lividity of the back, attributable to hypostatic congestion, is sometimes observed.

Of the symptoms referable to the digestive system, those most characteristic pertain to the stomach. Vomiting occurs, if not already present, and if present it becomes more prominent. In a large proportion of fatal cases and in a few of the cases ending in recovery the black vomit occurs. This, taken in connection with other symptoms, is pathognomonic of the disease. Heretofore there has been much discussion respecting the nature of the vomited matter, but it is settled that the appearances are due to blood changed by the action of the gastric fluids. As regards the gross appearances, the matter vomited is a thin liquid of a reddish, brown, claret, or blackish color, with sediment resembling coffee-grounds. Occasionally the liquid is bright red, the blood having undergone but little change. Rarely it has a greenish or yellowish tinge, from the presence of bile. The secretion is acid, and it has an acid taste without bitterness. It is sometimes acrid, excoriating the throat, tongue, and lips. The microscope shows the sediment to contain disintegrated particles of food, mucus, epithelium, blood-pigment, and deformed blood-discs, often decolorized. Fungus-spores are often found. A fluid resembling the black vomit may be produced artificially by adding an acid to blood out of the body. The bilious matter vomited in some cases of remittent fever presents somewhat similar gross characters, but chemical, microscopical, and spectroscopical examination shows the presence of bile and the absence of blood-constituents. The black vomit in different cases of yellow fever is more or less abundant. It is sometimes ejected with force, and sometimes by an act of regurgitation rather than of vomiting. It generally ceases for a period of from twelve to twenty-four hours before death. The black vomit rarely occurs until after the febrile paroxysm. It occurs at variable periods afterward, but generally not until toward the close of the disease. Blair described a matter vomited prior to the black vomit, limpid or slightly opalescent, which he termed the "white vomit." Tenderness over the epigastrium is more or less marked, and the tenderness is sometimes extreme. The tongue is frequently reddened, dry, and cracked; in some cases it is moist and covered with a creamy coating, and sometimes it preserves its natural appearance.

During the second stage the evacuations from the bowels are frequently of a brownish, approaching to a black, appearance. They resemble sometimes tar or molasses. This appearance is due to the presence of blood, so altered, however, in its passage through the intestinal canal that its morphological characters are not demonstrable. Uric acid and the triple phosphates may be found in the evacuations from the bowels. Sometimes blood but little altered is passed from the bowels. Diarrhœa in this as in the first stage is rare. The abdomen is soft and seldom meteorized. The melænic discharges generally precede the occurrence of black vomit, and they have been called the "black-vomit stools."

Albuminuria is of frequent occurrence in grave or fatal cases. Hyaline and granular casts, red blood-corpuscles, renal epithelium, and epithelium from the bladder are found in the sediment of the urine.[1] A scanty secretion of urine or suppression not infrequently precedes a fatal ending. Uræmia occurs in a certain proportion of cases. The urine in certain cases becomes of a yellow or orange color from bile-staining, and it is sometimes bloody.

Yellowness of the surface of the body—whence the disease derives its name—occurs after the febrile paroxysm. The conjunctiva becomes yellow, and this with the redness gives to the eye a striking and peculiar appearance. The yellowness of the skin is especially marked on the chest and upper extremities. The jaundice is probably hepatogenous, but it is considered by some as hæmatogenous. It exists in only a certain proportion of cases. It occurs in a much larger proportion of fatal than of non-fatal cases, and, in fact, it occurs in a small proportion of the latter. The yellowness continues after death and into convalescence.

Hemorrhage in various situations other than the stomach, intestine, and the kidneys or bladder, to which reference has been already made, is often a striking feature of the disease. It occurs from the nostrils, gums, uterus, wounds or abrasions of the skin, and sometimes from the eyes, meatus auditorius, finger-nails, holes bored in the ears for ear-rings, etc. Petechiæ and vibices are sometimes observed.

As regards symptoms referable to the nervous and the muscular system, coma and convulsions are of occasional occurrence. They are probably dependent on uræmia. Delirium is observed, but frequently the mind remains clear. Delirium may exist during the fever, and disappear afterward. Generally there is notable muscular prostration, but to this rule there are remarkable exceptions. Patients sometimes do not take to the bed, but keep about their usual avocations, not thinking themselves much ill, often a few hours before death. These have been called "walking cases." Rush relates a case in which the patient stood up before a glass and shaved himself on the day of his death. In a case cited by Bartlett the patient, a soldier, continued to do duty until black vomit took place. In another case the patient dictated and signed a letter a quarter of an hour before death. Cartwright cited an instance of a shoemaker who nearly finished a shoe the day before his death. Fenner saw a patient quietly reading a book after the black vomit had occurred. These cases exemplify a kind of cheerful delirium, as it was called by La Roche, in which the patient fancies himself well or but little ill.

The physiognomy of patients is described by different writers with a good deal of metaphor, but nearly all state that it is characteristic. The face is flushed. The appearance of the eyes is described by different writers as injected, brilliant, transparent, fiery, and glassy. To the facies are applied the following terms: suffering, dejection, anxiety, anguish, despair, terror, stupidity, vacancy, astonishment, sullenness, etc. The diversity of these terms raises a suspicion that the peculiarity of the physiognomy is less distinctive than observers have generally supposed; at all events, it appears to be difficult to convey by language a clear idea of the characteristic appearance.

The duration of the disease after the febrile paroxysm varies between twelve hours and three or four days. In cases of great gravity the duration is brief, the disease running rapidly to a fatal termination.

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[1] Vide "The Urine in Yellow Fever," by Prof. J. W. Holland, in the *London Practitioner*, July, 1879, and the *American Practitioner*, Sept., 1879.

Writers recognize a third stage in fatal cases, called the stage of collapse or the stage of exhaustion. The collapsed condition is denoted by prostration, feebleness and irregularity of the pulse, coldness of the extremities, low delirium in some cases, the intellect remaining clear in other cases, and mental indifference or apathy generally existing as in cases of epidemic cholera,—these symptoms showing tendency to death by asthenia. Coma and convulsions sometimes occur in this stage, attributable, probably, to uræmia. In some cases this period is characterized by great restlessness, jactitation, and general distress; but in other cases the patient remains quiet and seems free from suffering.

If death do not take place, the third stage is the stage of convalescence. When the disease passes to the second stage, and presents the grave symptoms belonging to this stage—namely, black vomit, hemorrhage, etc.—the recovery is always slow, and is frequently preceded by fever having more or less of the phenomena of the typhoid state. Relapses sometimes occur after convalescence appears to be declared.

In mild cases the disease ends in convalescence immediately following the paroxysm of fever. Whenever an epidemic prevails there is a certain proportion of cases devoid of any gravity. In these cases a slight chill occurs, followed by moderate pyrexia continuing for one or two days, and recovery quickly takes place. These cases would not be recognized as cases of yellow fever except from the fact of their occurrence during the prevalence of an epidemic. When this mild form of the disease occurs it is not infrequently a matter of doubt whether patients have had the disease or not.

More severe cases are distinguished by the intensity of the pyrexia. The term inflammatory, often applied to these cases, is not correct, inasmuch as the intensity of the febrile movement is not due to inflammation, but to the fever *per se*. Malignant cases are those characterized by black vomit or profuse hemorrhage in other situations, followed by collapse or exhaustion, and also cases in which coma or convulsions occur with suppression of urine.

The duration of the disease is variable. Shortness of its career is a distinguishing feature. In the shortest cases death or convalescence takes place in two or three days. According to La Roche, the duration varies between three and nine days. The average duration is less than a week.

CAUSATION.—The peculiar features of this disease, its limitation within certain geographical boundaries, and its occurrence as an epidemic, warrant the inference that it requires for its production a special cause. Does the special cause emanate from the bodies of those affected with the disease? in other words, is the disease contagious? This question has given rise to much discussion. Volumes have been written by contagionists and non-contagionists in defence of the two opposing doctrines. It is evident that the question is one of great importance in its bearing on commerce and quarantine laws. as well as on precautionary measures respecting exposure in visiting or attending upon those affected with the disease. (For a full consideration of this topic the reader is referred to works treating *in extenso* of yellow fever, and especially to the elaborate treatise by La Roche.) I shall simply state the grounds which substantiate the non-contagiousness of the disease:

1. The disease is confined within certain territorial limits. In this respect it differs from most diseases the contagiousness of which is established. Even in localities in which it is prevailing as an epidemic it is sometimes restricted to a circumscribed area.

2. The rise and progress of epidemics are not consistent with its diffusion

by contagion. For example, Fenner, with great zeal and fidelity, traced the first 30 or 40 cases in the epidemic at New Orleans in 1853, and ascertained that the disease broke out in different places among persons who could have had no communication with each other. Fenner investigated the origin and spread of the disease in New Orleans for twelve years, and stated that he never found the least proof of personal communicability. This is alike true of epidemics in other places.[1]

3. In certain places within the yellow-fever zone sporadic cases occur almost every year. According to Fenner, a summer never passes in which there is not a greater or less number of sporadic cases in New Orleans, but the disease prevails as an epidemic only in certain years. Were the disease contagious it should be diffused more or less whenever there are any cases of it.

4. When it prevails as an epidemic it spreads too rapidly to be diffused by contagion.

5. Persons going from a district where it prevails into a district where it does not exist, and becoming attacked in the latter, do not communicate the disease. There is abundant evidence that this is the rule, and the apparent exceptions are so few and of such a character that it is most logical to explain them otherwise than by the supposition of contagion.

6. Persons brought into close contact with yellow-fever patients—physicians, nurses, or other hospital patients—do not contract the disease.

7. Epidemics run a certain course as respects duration, and abruptly end, in this respect resembling epidemics of cholera. The disease should prevail longer and disappear more slowly if propagated by contagion. Like other epidemics, the disease, as a rule, becomes milder by continuance. It appears to absorb other diseases while it continues, in this respect resembling epidemic cholera, and its prevalence is arrested by cold. These facts, especially the two latter, are not consistent with the doctrine of contagion.

8. The great majority of those who have had an extensive practical acquaintance with the disease believe it to be non-contagious.

9. Experiments to test the question of communicability, by inoculation, by swallowing the black vomit, and by the utmost possible exposure, have led to negative results. To cite one among many experimental observations, Dowler gives the following account: In 1805, Don Cabanellos, a Spaniard, slept at night with his children in beds in the lazaretto in which yellow-fever victims had died. For submitting the question of contagion to this personal test he was made physician to the royal household, with an annuity of twelve hundred dollars. A number of galley-slaves who accompanied him had one year's imprisonment remitted. The whole party amounted to fifty, and no one suffered any harm.

10. Complete seclusion has proved ineffectual to prevent the disease.

In view of the foregoing considerations, to which others might be added, it seems certain that yellow fever is not produced by means of a contagium. Yellow fever, therefore, is a purely infectious miasmatic disease, using this term as already defined. (Vide p. 85.) Another question arises in connection with that of contagion—namely, May not the infection be transported in clothing, merchandise, etc., and the disease be in this way imported? Facts have established an affirmative answer to this question. The special cause can in this way be distributed by vessels, railroads, and other conveyances. The fact of the disease being portable renders judicious quarantine restrictions of vital importance, but in view of its non-contagiousness the restrictions need not include the detention of persons.

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[1] Vide article on "Epidemiological Conclusions and Suggestions," in *Brit. and For. Medico-Chirurg. Review*, Oct., 1870, p. 484.

Of the nature of the infection we have not, as yet, demonstrative proof. Something, however, is known of the conditions under which it is produced. It is indigenous in warm climates. Yellow fever is rarely developed south of 20° south or north of 40° north latitude. It prevails more in the Eastern than in the Western Hemisphere, and in certain parts of Europe and America than in Africa. In the Western World it prevails especially in the commercial towns on the Atlantic coast south of Charleston, S. C., on the Gulf of Mexico, and in the West India islands. It rarely prevails in rural situations. It prevails only in the summer season in other than tropical climates, and in the latter chiefly during the hottest months. A high temperature is essential to its causation, but in yellow-fever localities epidemics do not always occur in the years in which the temperature is highest. Humidity has been supposed to favor the development of the disease, but this is not conclusively established. There does not seem to be ground for connecting the causation with any known meteorological conditions other than a high temperature. As stated by Nott, the special cause is most active near the ground, persons on a ground-floor being more likely to be attacked than those in stories above. The miasm is more active at night than in the daytime.

In localities where the disease prevails often the conditions for its development do not uniformly exist during the hot seasons. Epidemics occur only in certain years. It seems that the efficiency of the special cause is favored by the co-operation of other causes, and hence sanitary measures—drainage, sewerage, removal of filth, avoidance of overcrowding, etc.—contribute to the prevention of yellow-fever epidemics.

Unacclimation is a condition pertaining to individual susceptibility. Natives of yellow-fever localities are rarely attacked. Particular epidemics, however, have been characterized by a considerable number of cases among the native population. Not having experienced the disease is another personal condition. The disease having been once experienced, the susceptibility to the special cause, as a rule, is extinguished. The number of persons who have the disease more than once is probably not greater than the number of exceptions to the rule that smallpox and typhus render the system insusceptible ever afterward to the special causes of these diseases. Acclimation, as regards this disease, is complete only when the disease has been experienced.

The negro race seems to be singularly exempt from a liability to this disease. Statistics show the mortality among negroes to be insignificant. It is stated, however, by Fenner that the insusceptibility is not nearly so great as mortuary statistics seem to show, in consequence of the fact that the disease in the negro is almost invariably mild and not likely to prove fatal. The susceptibility is not affected by age, persons of different ages, inclusive of infancy, being liable to the disease. Statistics show a larger proportion of cases among males than among females.

The special cause is destroyed by cold. It is a matter of common observation that an epidemic is arrested by one or two hard frosts. Epidemics, however, have a self-limited duration. Fenner noted that at New Orleans repeatedly the disease disappeared before the occurrence of frost. An epidemic rarely continues longer than 60 or 70 days. Barlow calculated the average duration of yellow-fever epidemics, and finds it to be 58.33 days, being somewhat longer than the mean duration of epidemics of cholera.

Of the nature of the special cause or infection, as already stated, positive proof has not as yet been obtained; but that the disease is produced by specific germs or organisms is as logically established as with respect to any of the infectious diseases the causes of which have not been demonstrated. Assuming the existence of yellow-fever germs, the production of the disease by filth or any local conditions without the presence of these germs can only be considered as possible by those who hold to the doctrine of spontaneous generation. While, as already stated, certain obvious local conditions may have an auxiliary agency, that these conditions are essential is disproved by instances in which the disease is developed in situations as healthy as possible in a sanitary point of view. The multiplication of germs without the body, therefore, may take place irrespective of any apparent local causes. The organisms which furnish the yellow-fever germs are exotic as regards most if not all parts of this country. The sporadic cases which appear in New Orleans in seasons when the disease is not epidemic are probably due to germs which have escaped destruction having been placed under circumstances favorable for retaining their vitality. This has been called the "laying over" or the "hibernation" of germs. Germs attached to clothing or other articles under favorable circumstances may probably retain their vitality and infective capability for a long period.

The period of incubation is variable. It varies between two and fifteen days, and in the majority of cases it is nearer the first than the second of these numbers. Campbell has reported an instance in which the period was nearly six weeks.[1]

DIAGNOSIS.—The access of the disease and the symptoms during the febrile or first stage present nothing very distinctive. All observers agree that it is often difficult to arrive at a positive diagnosis during this stage. In a considerable proportion of cases the disease at the end of this stage terminates in convalescence. These cases would not be considered as cases of yellow fever except during an epidemic. The disease in these cases presents the characters of a febricula. The symptoms which have diagnostic significance are—the abruptness of the attack, pains in the back and limbs, and suffusion or redness of the eyes.

In cases progressing beyond the febrile paroxysm and presenting the grave events which are liable to follow, the diagnostic features are sufficiently distinctive. They are—yellowness of the conjunctiva and skin, black vomit, hemorrhages elsewhere than from the stomach, epigastric tenderness, slowness of the pulse in certain cases, coldness of the extremities, suppression or a scanty secretion of urine, and the phenomena denoting collapse. All these events are not present in every case, but generally enough of them exist to render the diagnosis positive. The march of the disease to a fatal issue from the end of the febrile paroxysm or the first stage in well-marked cases is peculiar, and it can hardly be confounded with any other affection.

PROGNOSIS.—The mortality from yellow fever varies much in different epidemics. The range of variation is between 10 and 75 per cent.[2] The average mortality, according to calculations by La Roche, is 1 in 2.32. The rate of mortality differs in different periods of the same epidemics, the rule being a decrease in the rate as the epidemic approaches its termination. Bemiss gives the rate of mortality in private practice during the epidemic of 1878 in New Orleans as 10 per cent. among white patients and 3 per cent. among colored patients. The mortality during the same epidemic among hospital patients was 51.7 per cent. for white and 21 per cent. for colored patients.[3]

Unfavorable prognostics are—yellowness of the surface, black vomit, great diminution or suppression of urine, abundant hemorrhage in any situation, coldness of the extremities, jactitation, hiccough, delirium, convulsions, and coma. Of these symptoms, black vomit and suppression of urine are almost invariably forerunners of death. Coma and convulsions are fatal symptoms dependent on uræmia.

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[1] "The Yellow-Fever Germ on Coast and Inland," by Henry F. Campbell, M. D., *Trans. of the Med. Association of Georgia*, 1879.

[2] DaCosta, *Medical Diagnosis*, Philadelphia, 1881, p. 828.

[3] S. M. Bemiss, article on "Yellow Fever" in *Pepper's System of Practical Medicine, by American Authors*, Philadelphia, 1885, vol. i.

A favorable prognosis cannot be confidently entertained in any case of this disease. Unfavorable events are liable to occur when up to the time of their occurrence the symptoms appeared to be favorable. Black vomit, hemorrhage elsewhere, and uræmic convulsions or coma are liable to occur unexpectedly in cases which appear to be progressing favorably.

Exclusive of cases in which the immediate cause of death is uræmic coma, the mode of dying is by asthenia.

TREATMENT.—With reference to the treatment of yellow fever, it is to be premised that no specific remedy has been discovered. There is no known plan of treatment on which reliance can be placed to cut short the disease. Another consideration to be premised is that the disease, exclusive of malignant cases, tends to recovery. Mild yellow fever is a very mild disease, not tending to destructive lesions of either the solids or fluids, nor does it tend to continue indefinitely if not arrested, like intermittent fever.

A third preliminary consideration is that in a considerable proportion of malignant cases the disease is necessarily fatal. Hence, a large proportion of fatal cases is by no means proof of injudicious treatment. A fourth consideration is that different epidemics differ as regards the relative proportion of mild and malignant cases. In some epidemics most of the cases are mild, and in others malignant. It follows that the ratio of recoveries under a certain plan of treatment may be no test of the superiority of that plan. Measures which appear to be eminently successful in one epidemic may appear to fail in another epidemic, the difference being due to variations as regards the tendency of the disease to a fatal issue.

In cases of mild yellow fever no active interference is required. Quietude, restricted diet, ventilation, and other hygienic regulations, together with such palliative measures as particular symptoms in individual cases may indicate, suffice for the treatment. The palliative measures embrace anodyne and refrigerant remedies, cold applications to the head, sponging of the body, laxatives in some cases, etc. It is, however, to be borne in mind that the disease may become malignant in cases which are at first mild in appearance. It is impossible always to distinguish at the outset the cases which will prove to be mild, and hence it is of vast importance to take every precaution to prevent the development of grave symptoms. For this end hygienic measures are especially or chiefly important. Patients should at once give up to the disease and take to the bed. As complete rest of body and mind as practicable is to be enforced. The services of a faithful and experienced nurse are of more importance than medication. Free ventilation, cleanliness, and other sanitary measures are not less important in this than in other forms of fever.

In cases characterized by intensity of fever during the febrile paroxysm no one entertains at the present time the propriety of bloodletting. Antipyretic measures of treatment—namely, cold sponging and the wet sheet—are rationally indicated. Data are not as yet available for forming an opinion as to the efficacy of these measures in preventing the grave events of the disease and in lessening its mortality.

Purgatives have been much employed in this as in most other diseases. Drastic or active purgatives do harm by increasing the gastro-intestinal irritation and producing exhaustion. Even if well borne, it is difficult to see what indication they fulfil which may not be equally met by mild laxatives or simple enemas. The latter are indicated by constipation. Purgation, except in some cases for the purpose of eliminating urea, is to be avoided. Emetics, which have also been much used, are contraindicated by the gastric symptoms which belong to the history of yellow fever.

Mercury has been considered as important in this disease. Calomel in large doses has been much used, and it has been thought to be desirable to produce ptyalism. This practice has now but few advocates. Facts do not warrant the opinion that mercurialization is in any sense curative, and the testimony of most physicians who have had practical acquaintance with this disease is adverse to the use of mercury in any form.

Measures to relieve gastric irritability are often indicated. For this end counter-irritation over the epigastrium may be employed by means of small blisters, dry cupping, sinapisms, and stimulating liniments. Anodynes, given either by the mouth or by the hypodermic method, are indicated for this end. Other remedies are chloroform in small doses, prussic acid, and creasote. Ice swallowed in small pieces has been found useful in allaying irritability of the stomach. To prevent black vomit, and with a view to arrest gastric hemorrhage when it has occurred, the acetate of lead with opium has been much extolled. With reference to this event careful attention should be given to the ingesta. Milk with lime-water, not given in large quantity, is probably the best article of diet. If sustaining measures be not indicated, bland liquids only should be taken into the stomach.

Hemorrhages elsewhere call for local styptics if the source of the hemorrhage be accessible, and for ergot or other hæmostatics. In a case related to me by a former colleague life was apparently saved by the transfusion of blood.

Great restlessness and vigilance call for opium or other anodyne remedies. Deficiency of the urinary secretion is an indication for diuretic remedies if they be tolerated by the stomach, and for measures to produce diaphoresis if diuretics be not borne or if they prove inoperative. If symptoms be present denoting uræmia, the hot-air bath should be employed. Under these circumstances elaterium or some other hydragogue may be advisable if relief be not procured by diaphoresis.

Sustaining measures are indicated in proportion as a tendency to collapse or death by asthenia is denoted by the symptoms. The irritability of the stomach may interfere with the sustaining treatment. The forms of stimulus and nourishment are to be selected which on trial are found to be best borne. Alcoholic stimulants may be given *per enema* if not retained by the stomach. Two of my colleagues in the New Orleans School of Medicine, who had experienced this disease in its severe or malignant form, attributed their recovery to the free use of alcoholic stimulants. In one of these cases, when a fatal termination was regarded as imminent, a favorable change occurred immediately after champagne had been given freely.

PREVENTION.—Measures for the prevention of yellow fever relate—1st, to the removal of local conditions which favor the multiplication of the disease-germs; 2d, to quarantine regulations; and 3d, to disinfection. The first of these three divisions embraces all that pertains to public and private hygiene. The local conditions which are especially important as auxiliary causes are unknown, and therefore it can only be hoped that they will be reached by rendering sanitary measures as complete as practicable; but, be these measures never so complete, they do not make superfluous those of quarantine and disinfection. The object of quarantine regulations is that the disease-germs shall not be imported. To effect this object either there must be non-intercourse with places in which the disease prevails, or all articles of merchandise, clothing, etc. brought therefrom must be thoroughly disinfected. To be efficient, quarantine regulations must include not only vessels from infected parts, but inland transportation by railroads and other conveyances. There is no danger of the disease being carried by the living body after disinfection of the wearing apparel, or by the bodies of the dead. Detention of the well or the sick is therefore a needless precaution, except to prevent groundless popular apprehensions. If importation of the germs have taken place, the houses with their surroundings in which cases occur should be instantly and completely disinfected. The object now is to "stamp out" the disease. There is ground for the belief that this object may be effected if measures of disinfection be promptly and thoroughly carried out.

During the prevalence of an epidemic unacclimated persons should avoid going within the infected area except under a sense of duty, and they who are already within this area should leave it unless there be motives for remaining which render the risk of life justifiable and praiseworthy.

CHAPTER VI.

ERUPTIVE FEVERS.

Variola, or Smallpox: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Varioloid, or Modified Smallpox.—Vaccinia, or Cowpox.—Varicella, or Chicken-pox.

THE fevers which remain to be considered are characterized by an eruption or exanthem, and hence they are called eruptive or exanthematous fevers. The continued fevers, typhus and typhoid, have also an eruption, but the eruption is less constant and less prominent than in the eruptive fevers. The eruptive fevers are *variola*, or smallpox, including the modified form known as *varioloid; varicella*, commonly called chicken-pox; *scarlatina*, or scarlet fever; *rubeola*, or measles; *roseola*, or rose-rash; and *rötheln*, or German measles. The eruption in two of these fevers is moist; that is, vesicular or pustular. This is true of variola and varicella. In the remainder the eruption is dry, and is properly an efflorescence or rash. To the latter kind of eruption the term exanthem is, strictly speaking, restricted.

The eruptive fevers were formerly considered as varieties of one disease, and the individuality of each was not fully settled much before the present century. Each is a distinct species of fever, having phenomena and laws peculiar to it and its own special cause. All, with the exception of roseola, are propagated by special causes reproduced within the body; that is, they are communicable diseases.

The division of the career of the disease into stages is the same in all the eruptive fevers. The first stage is the stage of invasion or the access. This stage begins with the first manifestations of disease and ends with the first appearance of the eruption. The second stage is the stage of the eruption, and extends from the time when the eruption first appears to its disappearance. This stage is subdivided in variola. The stage of desquamation or desiccation follows the eruptive stage, and this either constitutes or is followed by the stage of convalescence. The consideration of these fevers will also embrace the periods of incubation and certain events liable to occur after the disease, or sequels.

Variola, or Smallpox.

The discovery of vaccination by the immortal Jenner toward the close of the eighteenth century has divested this disease of much of the importance which it had in the two previous centuries, when it ranked first among the acute affections destructive of human life. It is not, however, so rare, even in its unmodified form, at the present day but that cases are liable to come under the observation of every practitioner. The gravity and loathsomeness of the disease, together with its contagiousness, render highly important an early diagnosis and judicious management. I shall consider first unmodified variola, and afterward, under a separate head, the modified form of the disease known as *varioloid.* The latter head will embrace the consideration of variola produced by inoculation.

ANATOMICAL CHARACTERS.—The characteristic lesion of smallpox is the eruption. The successive stages of the eruption are the macule, the papule, the vesicle, and the pustule. The pustule undergoes desiccation. The *macule* is a reddish, slightly-elevated spot due to congestion of a circumscribed portion of the skin. The microscope shows that in this stage the capillaries of the papillæ of the corium are dilated, tortuous, and elongated. The connective tissue of the papillæ is slightly œdematous. The *papule* is due chiefly to swelling of the epithelial cells of the rete Malpighii, and to the appearance in the same situation of little cavities containing a clear fluid. It is to be noted that cavities holding fluid are present in the papule as well as in the vesicle; but in the former they are so small that the vesicular structure is not evident to the naked eye. The swelling of the epithelium and the small cavities are present chiefly in the middle layers of the rete Malpighii. Weigert[1] has shown that in the centre of the pock the lowest cells of the rete Malpighii have undergone a peculiar metamorphosis, by which they are transformed into irregularly-shaped opaque masses devoid of nuclei. To this change he has given the name *diphtheroid metamorphosis*, as it resembles the change described by Wagner as occurring in the epithelium of mucous membranes in diphtheria. The metamorphosis belongs to the group of the coagulation necroses described in Part I. pp. 52 and 53. Weigert regards this necrosis of cells in the rete Malpighii as the first and the characteristic effect of the smallpox poison upon the skin. The diphtheroid metamorphosis affects only a certain number of cells in the base of the pock.

Another alteration which many of the epithelial cells undergo is the formation in their protoplasm of spaces or cavities containing a clear, serous fluid. This alteration has been called *serous infiltration* and *vesicular or dropsical transformation* of the cells. The cells in the upper and the middle layers of the rete Malpighii are those chiefly affected. At first a clear ring forms around the nucleus; and this transparent zone increases in size until nearly the whole cell is converted into a cavity, the wall of which is formed by the outer border of the cell. In this vesicular transformation the cell becomes swollen, so that the cavity may be larger than the original cell. The boundaries separating adjacent cell-cavities may become ruptured, so that by the coalescence of several cavities larger spaces with irregular walls are formed. In this way many explain the formation of the *vesicle* out of the papule. According to this view, in the papule a large number of the epithelial cells in the upper Malpighian layers become swollen and transformed into cavities, the walls of which are represented by the condensed peripheral layers of the cell. These cavities by rupture of their walls open into each other, and when large spaces containing a clear fluid have thus been formed the appearance is that of the characteristic smallpox *vesicle.* The interior of the vesicle is subdivided by a network or reticulum composed of altered epithelial cells; but the vesicular transformation of cells is not the sole origin of the small cavities of the papule or of the larger spaces of the vesicle. The serum which transudes from the capillaries of the corium penetrates the epidermis, and not only enters the cells, but also makes its way between the cells, which are thereby pushed apart. Thus, spaces containing serum exist between the cells as well as within the cells. The extracellular accumulation of serum and the coalescence of intracellular cavities combine to form the spaces of the smallpox vesicle.

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[1] *Anatomische Beiträge zur Lehre von den Pocken*, Breslau, 1874, Theil i.

The manner in which the *reticulum* of the smallpox vesicle is formed has been, and is still, a matter of controversy. The reticulum consists of coarse and of fine bands which stretch from the floor to the roof of the vesicle, and which interlace in its interior. It is generally admitted that these bands are composed of more or less altered epithelial cells. Weigert believes that the reticulum is formed in great part of irregular and branching masses which represent cells that have undergone the diphtheroid metamorphosis or coagulation necrosis. Cornil and Leloir are of the opinion that the reticulum is formed mainly by the borders of cells in whose substance cavities have appeared, these borders representing the partitions between the intracellular cavities. All, however, admit that at least some of the bands are composed simply of epithelial cells which have been compressed, elongated, and displaced by the accumulation of fluid between them.

The roof of the smallpox vesicle is composed of the stratum corneum, the stratum lucidum, and often some of the upper cells of the rete Malpighii. The sides of the vesicle are formed of cells in the middle layers of the rete Malpighii, and the floor of the vesicle is composed of the lowest cells of the rete Malpighii, some of which have undergone the diphtheroid metamorphosis. Some of the epithelial cells in the sides and in the base of the vesicle are enlarged, and these often contain two or more nuclei. There is reason to believe that in these situations there is an active proliferation of epithelial cells.

A characteristic appearance of the smallpox vesicle is produced by a central depression in its roof. This depression constitutes the so-called *umbilication of the vesicle.* Not every smallpox vesicle is umbilicated. Vesicles in the palm of the hand and in the sole of the foot are not umbilicated. There have been various theories as to the cause of the umbilication. Some have attributed it to the existence of a sweat-gland or of a hair-follicle in the centre of the vesicle. The umbilication may, however, be present in vesicles which are not formed around hair-follicles or sudoriparous glands. The most plausible explanation is that which attributes the central depression to swelling and proliferation of the epithelial cells in the sides of the vesicles. The influence of this cause is aided by the presence of bands which pass from the centre of the roof to the floor of the vesicle. These bands, the existence of which has already been noticed, act as retinacula which hold down the central part of the roof, while the sides of the vesicle are swollen by the enlargement and proliferation of cells and by the accumulation of serum.

The smallpox vesicle is filled with serum, called, in this situation, lymph. The serum may be perfectly clear, but it usually contains a few pus-cells and some granular material and micrococci. The pus-cells are probably emigrated white blood-corpuscles. A delicate network of fibrillated fibrin is sometimes found in the lymph of the vesicle. The lymph in the vesicles becomes more and more clouded by the presence of pus-cells. By the accumulation of these cells the vesicle is gradually transformed into a *pustule*. As the pus-cells increase in number the reticulum within the vesicle is destroyed, so that the fully-formed pustule consists of a single cavity. The destruction of the reticulum causes the umbilication to disappear.

In hemorrhagic variola the vesicle becomes filled with red blood-corpuscles instead of emigrated white corpuscles. Between the corpuscles is a network of fibrin.

Pus-cells are found in the connective tissue of the corium, especially around the vessels of the papillæ. These pus-cells, which are undoubtedly emigrated white blood-corpuscles, are present in large number during the pustular stage. The pustule may extend in depth in consequence of suppuration of the upper layers of the corium. The cicatrices or pock-marks which follow an attack of variola are extensive and deep in proportion to the extent to which the corium is involved in the suppurative process during the pustular stage.

During the stage of desiccation the contents of the pustule disintegrate and dry up. After the pustule ruptures and discharges its contents brownish scabs are formed by the desiccation of the pustular contents. Beneath these scabs the epithelium is regenerated, the new cells being produced by proliferation from the intact epithelial cells.

In a number of cases of smallpox Weigert observed colonies of micrococci in blood-vessels and lymph-spaces in the corium, as well as similar colonies in the vesicles. These micrococci are to be regarded as a complication, and not as concerned in the causation of the disease.

The occurrence of the variolous eruption upon the mucous membranes of various parts of the body will be described in connection with the Clinical History. Parenchymatous degeneration of the liver, heart, and kidneys has been observed in cases of smallpox. Colonies of micrococci are often found in internal organs. The formation of abscesses is rare. The spleen is sometimes swollen. Ecchymoses are very frequently found in the mucous membrane of the renal pelvis and of the ureters. In variola hæmorrhagica, hemorrhages are present in the interior of the body as well as upon the surface. They are found in the mucous membrane of the air-passages, in the lungs, in the mucous membrane of the alimentary tract, especially of the stomach and rectum, in the uterus, and rarely in the Fallopian tubes and ovaries or in the testicles. Punctate hemorrhages are present in the serous membranes and in the pia mater. The complications of smallpox, which are many, will be noticed under the head of Clinical History.

Clinical History.—Differences pertaining to the eruption and other events embraced in the clinical history in different cases are so great that writers have made several varieties of the disease. The division into *confluent* and *discrete* or *distinct variola* is generally adopted. In the latter variety the vesicles and pustules are separate or distinct from each other. In the former variety coalescence of the vesicles or pustules takes place. In cases in which coalescence exists to a greater or less extent, but not over the whole or the greater part of the surface of the body, the disease is said to be *semi-confluent*. When the eruption has a hemorrhagic character the disease is called *hemorrhagic* variola. Without considering these varieties under separate heads, I shall notice their distinctive features in connection with the symptomatology of the several stages of the disease.

Stage of Invasion.—The disease is ushered in by a chill in the great majority of cases, and the chill is usually more marked than in the other eruptive fevers. In some cases a series of chills occurs, alternating with flushes of heat. Febrile movement follows, accompanied generally by more or less perspiration. The latter is likely to continue or to recur more or less frequently up to the time of maturity of the eruption—a feature distinctive of this as compared with the other eruptive fevers. The febrile movement is known as the primary fever. It is often intense, the thermometer sometimes showing a rise to 104° or 105°. Nausea and vomiting are often prominent symptoms in this stage. The tongue becomes coated. Pain is referred to the epigastrium, accompanied by tenderness on pressure, either with or without notable nausea and vomiting. Generally, the bowels are constipated, but diarrhœa is sometimes present, especially in children. Cephalalgia, pain in the limbs, and general debility are more or less marked, as in the early part of other essential fevers; but in this fever pain in the loins is usually a marked symptom and possesses diagnostic importance. Incomplete paraplegia is occasionally observed, generally disappearing with the development of the eruption. Paralysis of the bladder, giving rise to retention of the urine, sometimes exists without paraplegia. Convulsions often attend the development of this disease in children, and sometimes occur in adults. Delirium is an occasional symptom.

If the symptoms in this stage be mild, the eruption will probably be discrete; but if the chill be notably marked, the pyrexia intense, the lumbar pain severe, etc., it may be expected that the disease will prove to be of the confluent variety.

The duration of this stage, as a rule, is two days. The eruption begins to appear on the third day after the attack. There are, however, exceptions to this rule. In some cases the eruption appears on the second or on the fourth day. The duration is sometimes extended to the fifth or sixth day, and occasionally even longer. The eruption, if it appear on the second day, may be expected to be confluent, and on the other hand in proportion as the appearance of the eruption is protracted beyond the third day mildness of the disease is to be expected. Two events relating to the skin are of occasional occurrence in this stage. One of these is a cutaneous efflorescence or rash. The whole or the greater part of the surface of the body may be covered with this erythematic redness, resembling the appearance in scarlatina. The disease is liable to be mistaken for scarlatina when this occurs. In other cases the rash is in isolated patches, and it may then bear a resemblance to measles. The patches, however, are not elevated; that is, they are not papular. Small vesicles or sudamina are sometimes observed. The efflorescence appears on the second day and continues only eighteen or twenty-four hours, giving place to the eruption peculiar to the disease.

The other event is the appearance of petechiæ and ecchymoses. These may be more or less abundant. They are sometimes very abundant, covering both the trunk and the extremities. In some cases these foreshadow subsequent hemorrhages, but they by no means always have this significance. They do not always denote severity of the disease, and they are sometimes observed in mild cases. They continue during the greater part of the stage of the eruption.

Stage of Eruption.—The histological structure of the different forms of the eruption has already been considered. It remains to notice the clinical characters of the eruption. The eruption, as a rule, appears first on the face, especially about the lips and chin, and nearly at the same time on the neck and wrists; next on the chest and arms; then over the body; and last on the lower extremities. Its diffusion over the whole cutaneous surface occupies between one and three days. Exceptionally it is first observed on the genital organs, chiefly in young children, and on the loins or nates. It may appear first in the neighborhood of a blister or sore existing in any part of the body. Successive changes take place in the physical characters of the eruption which in different stages of its progress represent nearly all of the cutanei. At first the eruption appears in the form of small red spots or specks, sometimes having a purplish or livid color. It is now a maculated eruption. The central part of the *maculæ* becomes hard, slightly elevated, and pointed. A change has taken place from a maculated to a papular eruption. In this stage of its progress the eruption is not unlike that of measles, and the disease is liable to be mistaken for the latter. The *papulæ* are smaller, rounder, and harder, and lack the crescentic or curvilinear arrangement which characterizes the eruption of measles. They feel like small shot under the skin. Next, a clear liquid becomes visible in the *papulæ*, which now become *vesiculæ*. This change is observed twenty-four hours after the first appearance of the eruption. After twenty-four hours more—*i. e.* forty-eight hours from the first appearance of the eruption—the vesicles have acquired considerable size. On the fifth day of the stage of the eruption the vesicles have attained to nearly or quite their full development, measuring from one-quarter to one-third of an inch in diameter, and are raised two-tenths to four-tenths of an inch above the level of the skin. More or less of the vesicles present a depression in the centre. They are said to be *umbilicated.* This appearance is highly characteristic, indeed almost pathognomonic. It is observed in no eruption other than that of variola, except in the vaccine vesicle, and sometimes in ecthyma. This umbilicated appearance may be detected in some of the vesicles, often as soon as the latter are discoverable. The vesicles are multilocular, as has already been described. They contain an opalescent serum.

This is the history of the eruption up to the period when the vesicles reach the maximum of their development if the eruption be discrete. In the confluent variety a diffused redness of the surface precedes the appearance of papules and vesicles. This diffused redness is likely to lead to the error of supposing this disease to be measles. The vesicles when they first appear have not the determinate form of those in discrete smallpox, but run together or coalesce to a greater or less extent. At the end of the period for the full development of the vesicles the face and other parts are covered with patches of greater or less size, in which the cuticle is uniformly raised by opalescent serum, resembling a blister. The whole of the face and the greater part of the cutaneous surface may be covered with coalescent vesicles.

After the full development of the vesicles there is another important change in the eruption. The vesicles become pustules. In other words, suppuration takes place. With this change the external characters are altered. The pocks are more distended. The reticulum is broken up. The central depression is lost, and the pustules become pointed. This change from vesicles to pustules is accompanied by general symptoms which show it to be an important epoch in the clinical history of the disease. Hence with this change begins another stage—namely, the *suppurative stage*, or the stage of *maturation.* This will claim consideration under a distinct head. It will be observed that of the different forms of cutaneous eruption, all except the *squamæ* and *tuberculæ* are represented in the successive changes in the eruption of smallpox—namely, enumerating them in the order of their sequence, *maculæ*, *papulæ*, *vesiculæ*, *pustulæ*, and in the confluent eruption *bullæ*.

The eruption is not limited to the skin. Simultaneously with its appearance on the cutaneous surface it may be observed to a greater or less extent on the mucous membrane of the mouth and throat. It appears here in the form of round opaque spots. As no stratum corneum exists upon the mucous membrane, the formation of typical vesicles and of pustules does not take place. Little erosions and ulcers rapidly form in the site of the eruption. The membrane surrounding the spots is swollen and inflamed. The spots are observed especially on the tongue and soft palate. They are liable to occur in the larynx, trachea, and bronchi; on the conjunctiva, leading sometimes to ulcers and destruction of the eye; within the nostrils, in the external meatus auditorius, and on the prepuce or vulva. The eruption does not occur upon serous membranes, and probably not on the mucous membrane of the stomach or intestine. In the larynx the eruption may cause laryngitis, which may prove fatal. An instance of this kind has fallen under my observation.

A striking feature of the primary fever pertaining to this disease is either a notable remission or complete apyrexia on the appearance of the eruption. The pulse falls to nearly or quite the normal frequency, and the heat of the skin is but little or not at all above that of health; and the thermometer in the axilla shows a reduction to 100° F. or lower. This is a highly diagnostic feature of smallpox, provided the eruption be not confluent. In the confluent variety this feature is either wanting or is much less marked. As the eruption progresses the febrile movement is gradually developed or increased.

Stage of Suppuration.—With the change of the eruption to pustules, usually on the sixth day after the first appearance of the eruption, there is a recurrence of notable increase of pyrexia, constituting what is known as the *suppurative fever* or the *secondary fever.* The pulse becomes more or less frequent, the heat of the skin is sensibly raised, and the thermometer denotes increase of temperature, especially in the evening as compared with the morning. The perspirations, which up to this epoch are likely to occur, now cease. In mild cases of discrete smallpox the secondary fever continues only for three or four days; but it continues longer in the confluent variety irrespective of any complication. The pyrexia is symptomatic of the affection of the skin, and its intensity, other things being equal, is proportionate to the degree of cutaneous inflammation. More or less redness and œdema of the skin are apparent in the spaces between the pustules. Tumefaction of the face occurs, and is often considerable. The swelling of the eyelids is often sufficient to close the eyes, as in cases of erysipelas. Swelling of the hands and feet occurs in severe cases, especially if the eruption be confluent. The redness and swelling are accompanied by a burning pain, as in erysipelas. Salivation is a frequent symptom. In severe cases of the confluent variety it is often very profuse, and accompanied with more or less swelling of the salivary glands. It is measurably or chiefly dependent on the eruption in the mouth and fauces. It is rarely observed in children. In severe cases delirium is likely to occur in this stage. The delirium is generally passive, like that in most cases of typhoid fever, but it is sometimes active or maniacal. Other ataxic symptoms—namely, subsultus, carphologia, and coma-vigil—may also occur, denoting always a condition of gravity. Coma occurs in a certain proportion of fatal cases. Other symptoms are due either to complications or to anomalous occurrences which will presently be noticed. Diarrhœa is an occasional symptom, and is always an unfavorable omen. The duration of the stage of suppuration is four or five days.

Stage of Desiccation.—This stage begins on about the twelfth day of the disease. The exceptional cases in which it begins much earlier or later are very few. The tumefaction and redness of the skin diminish. The pustules, for the greatest part, break, and the pus concretes into a thick scab. Over patches in which the eruption is confluent there is a continuous scabby crust. The whole face in severe cases is covered as if by a mask, presenting a hideous appearance, and the greater portion of the surface of the body may present a similar aspect. The diminution of the inflammation and the formation of scabs are first observed on the face, afterward on the trunk and upper extremities, and at last on the lower extremities. During this stage the skin exhales a sickening characteristic odor. In cases in which the eruption is confluent the stench is extremely offensive and may be perceived at a considerable distance. The spectacle in this and the preceding stage, and the fetor, render smallpox the most repulsive and loathsome of diseases. In mild cases the stage of desiccation is the stage of convalescence. The scabs fall off, leaving the skin beneath unbroken, but presenting a discoloration continuing for a long time, and especially apparent when the surface is exposed to cold; but in severe cases the skin beneath the scabs is excoriated or ulcerated, the scabs are liable to be renewed, and troublesome ulcerations sometimes follow. More or less pyrexia in severe cases continues into the stage of desiccation. In mild cases there may remain no permanent traces of the eruption, but in the great majority of cases there are left cicatrices, either circular or linear, which are known as "pitting" or "pock-marks." The amount of pitting will depend on the abundance of the eruption, the size of the pocks, and the depth of the ulcerations. Some of the pocks do not break, but harden, and their contents are absorbed. It is probable that in these the corium is not involved in the suppuration or that the vesicles are not converted into pustules.

To recapitulate the relative duration of the several stages in the majority of cases: The duration of the stage of invasion is between two and four days; of the stage of eruption, about five days; of the stage of suppuration, four or five days; and of the stage of desiccation, between six and ten days—making the duration of the disease in cases ending in recovery between seventeen and twenty-four days. In mild, uncomplicated cases the recovery is usually rapid, but convalescence may be indefinitely prolonged by ulcerations of the skin and other complications.

Various complications and anomalous events are liable to occur in the several stages of the disease. Pharyngitis, dependent on the eruption within the pharynx, is sometimes sufficient to occasion much pain and difficulty of deglutition. Laryngitis, dependent on the eruption within the larynx, is of not infrequent occurrence. It is denoted by huskiness of voice, or aphonia, with, in some cases, more or less difficulty of respiration. The laryngeal inflammation is sometimes diphtheritic. Œdema of the glottis is an occasional accident. Bronchitis, also from the eruption, occurs in a certain proportion of cases. Sometimes there is inflammation of the gastric or of the intestinal mucous membrane. This is rarely of the diphtheritic variety. Pneumonitis, pleuritis, and pericarditis are rare complications. Endocarditis, including the acute ulcerative form, has been observed as a complication. Furunculi, or boils, and subcutaneous abscesses are not uncommon, both as concomitants and sequels. Phlegmonous tonsillitis and suppurative inflammation of the cervical lymphatic glands and of the salivary glands occasionally occur. Erysipelas on the face, neck, limbs, or body is not very infrequent. Gangrene may occur on parts exposed to pressure, and sometimes in other parts. Ophthalmia is an important complication, sometimes leading to permanent blindness from opacity or perforation of the cornea. The inflammation in the throat sometimes extends through the Eustachian tube and gives rise to median otitis, which is liable to eventuate in deafness.

Hemorrhage in various situations occurs in a certain proportion of cases. Epistaxis is an occasional early symptom, but does not betoken unusual gravity of the disease. Hæmaturia is sometimes an early and always a grave symptom. Hemorrhage occurring at any period of the disease from the mouth, throat, air-tubes, or bowels, accompanied by petechiæ or vibices, and sometimes by an extravasation of blood into the pocks, characterizes certain malignant cases. Hemorrhagic, scorbutic, and black variola are names applied to the disease as exhibited in these cases. The most dangerous form of hemorrhagic smallpox is that in which an acute hemorrhagic diathesis develops in an early stage of the disease. The disease has then been called *purpura variolosa.* Death may take place in these cases before any eruption characteristic of smallpox has appeared, and then a diagnosis is possible only from a knowledge of the etiology of the case. In females menstruation may occur, and if not irregular or profuse it is not an untoward event. Metrorrhagia often occurs in hemorrhagic cases.

Albuminuria is a symptom of not infrequent occurrence in severe cases. It is probable that the coma which occasionally occurs is sometimes due to uræmic poisoning dependent on acute nephritis. Orchitis and ovaritis are rare complications. Inflammation of the synovial membranes of the joints, sometimes with serous and sometimes with purulent exudation, is also an infrequent complication. Paralysis of the lower extremities and of the bladder has been known to be a sequel of smallpox. This is due, according to Westphal, to an acute disseminated myelitis.

CAUSATION.—Smallpox is a highly contagious disease. It may be communicated by means of a virus—that is, by inoculation—and by means of inappreciable emanations from the body. The disease is readily transported by means of fomites, which may retain the contagium in an effective condition for months and even years. A very transient and slight exposure often suffices for the production of the disease. Thus, it is not infrequently taken by passing in the street or meeting in public conveyances persons who either are affected or have recently been affected with it. It may be contracted in hackney-coaches which have been used for carrying patients to hospitals. The disease is generally supposed to be communicable in all its stages, but undoubtedly it is most highly so during the stages of suppuration and desiccation. The contents of the vesicles and pustules and the crusts and scabs formed by desiccation contain the contagium; and it is probably contained not only in the emanations from the cutaneous surface, but in the expired breath and perhaps in the intestinal excreta. The disease may be communicated from the dead body by means not only of a virus, but by inappreciable emanations. Thus it has been contracted in the dissecting-room from subjects dead with the disease. It is stated that a cadaver may retain the virus in a condition to communicate the disease for an indefinite period—even for several years.[1] The following instance is interesting as showing the communication of the disease from the dead body, death having taken place before the development of the eruption: "During the winter of 1848–49 a young man, a member of the medical class of the New York University, died suddenly and unexpectedly in the night under the care of a physician who had not thought him seriously ill. I was invited to the autopsy, and observed, when the corpse was uncovered, a few dark-red spots on the surface which were supposed to be petechial, the principal symptoms of his attack having been gastric with great debility, as we were informed. The coffin was taken home to a New England village for burial, where, at the funeral, some of the relatives approached and opened it to see the face of the deceased before it was inhumed. Of this number, eight were attacked with smallpox, no other persons in the neighborhood being assailed."[2] Whether the disease be ever produced otherwise than by means of a contagium is, and must always remain, an open question; for with regard to reported cases in which the disease has appeared to originate spontaneously it can always be said that there may have been some unsuspected and untraceable exposure.

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[1] Vide *Nouveau Dict.*, art. "Contagion."

[2] Vide article by Dickson in the *American Journal of the Medical Sciences*, for July, 1862.

This explanation is more rational than the supposition that the special cause is produced *de novo*.

The susceptibility to the disease exists at all ages, but it is greatest in children. Some persons are wholly insusceptible to it, exposing themselves as fully as possible with impunity. Cases have been observed in which persons have become susceptible after having been insusceptible for many years. Unknown causes peculiar to certain times and places co-operate with the contagium in the diffusion of the disease, so that it prevails as an epidemic. Reference in these statements is had to the production of the disease in the natural way; that is, exclusive of inoculation. The disease as thus produced is distinguished as *natural smallpox*. The negro and Indian races are particularly susceptible to the disease. As a rule, the occurrence of the disease once extinguishes the susceptibility ever afterward. Instances, however, are not extremely infrequent of the disease having been experienced twice, and it has been known to occur thrice.

Certain interesting facts relate to the susceptibility of the fœtus *in utero*. A pregnant woman affected with the disease may or may not communicate it to the fœtus. The development of the disease before birth generally causes the death of the child, and cases have been reported of stillborn children presenting the different stages of the eruption. The death of the fœtus, however, does not always occur. Healthy children have been born presenting the evidence of having passed safely through the disease in the uterus. In some instances the disease has been contracted before birth, but not developed until several days after birth. Recovery has taken place under these circumstances. The disease may be communicated to the unborn child by the mother after the fourth month of pregnancy, and perhaps before. Finally, the special cause may be received into the system of the mother and the fœtus become affected without the mother experiencing the disease. This fact has been observed in cases in which the susceptibility of the mother had been extinguished, either by the disease having been experienced or by vaccination.

The special cause of this disease remains for a certain period latent; that is, there is a period of incubation. The duration of this period varies between five days and nearly three weeks, the average duration probably being between twelve and fourteen days. Facts appear to show that the special cause of rubeola or of scarlatina may be in operation simultaneously with the operation of the special cause of variola; in other words, smallpox may be combined with either measles or scarlet fever. Cases, however, exemplifying these combinations are extremely rare.

During an epidemic prevalence of smallpox cases are sometimes observed in which all the symptoms of the stage of invasion occur without being followed by an eruption. The disease appears to abort spontaneously at the end of the stage of invasion. These attacks resemble febricula, but it has been observed that persons who have had these attacks during the prevalence of smallpox are thereafter insusceptible to the disease, although not protected by vaccination. Hence it has been considered that the disease sometimes occurs without an eruption; and writers have been accustomed to recognize as a variety of the disease *variola sine variolis*.

The question as to what is the essential causative principle, or the contagium, is one of great pathological interest. It was at first thought by Weigert and by Cohn that the round bacteria or micrococci which they have observed in lymph from the vesicles, as well as in other parts, represent the contagious germs of smallpox. This view is now abandoned.

DIAGNOSIS.—The diagnosis of smallpox presents no difficulty after the characters of the eruption are fully manifested; but it is not so easy at an early period of the stage of the eruption before the vesicles are distinct. In seeking to determine the character of the eruption vesicles should be looked for, and next their umbilicated appearance. This appearance, it is to be borne in mind, is almost pathognomonic. The distinctive characters of the papules, as determined by the touch, are also to be borne in mind—namely, they are granular, hard, and deep-seated. The duration of the stage of invasion is to be considered—namely, from two to three days. The prominence of lumbar pain and vomiting, and especially either a remission or a cessation of febrile movement at the time of the appearance of the eruption, are important diagnostic features. It is to be remarked that lumbar pain is not in all cases a prominent symptom, so that its absence is not entitled to much weight in the exclusion of smallpox. An examination of the throat and mouth should not be omitted. The round, whitish, or ashy spots which are characteristic of the eruption on a mucous surface are generally apparent here as soon as, or before, the eruption appears on the skin. The appearance of the cutaneous eruption successively on the face or neck, the trunk and upper extremities, and the lower extremities is to be recollected.

The diagnosis cannot be made with positiveness prior to the stage of the eruption. Pain in the loins with febrile temperature, taken in connection with the absence of the symptoms which attend the access of the other eruptive fevers and of typhoid fever, should excite a strong suspicion of smallpox, especially if the patient be not protected either by vaccination or by having once had the disease. The exclusion of rubeola and scarlatina by the absence of the symptoms characteristic of the stage of invasion in these diseases is an important step in the diagnosis prior to, and in the early part of, the stage of eruption. When the eruption first appears the differential diagnosis lies chiefly between these fevers. In rubeola the Schneiderian membrane and air-passages, and in scarlatina the fauces, are prominently and early affected, as will be seen in treating of these fevers. Moreover, in rubeola the stage of invasion is longer, and in scarlatina it is shorter, than in variola. In neither rubeola nor scarlatina does the pyrexia remit or cease when the eruption appears. Other affections liable to be confounded with smallpox are lichen accompanied with pyrexia and a secondary syphilitic eruption. The occasional occurrence of an efflorescence and of petechiæ preceding the eruption proper, to which reference has been made, is to be borne in mind. The difficulties attending the diagnosis of purpura variolosa have already been mentioned.[1]

The diagnostic points should be fixed in the memory, as, in view of the danger of the diffusion of the disease by exposure before its character is ascertained, an early diagnosis is immensely important. As another inducement for being prepared to recognize the disease promptly it may be added that the ability of the physician is thereby strikingly shown, while, on the other hand, delay or failure to recognize it brings upon him popular censure.

The attempt has sometimes been made by criminals and impostors to simulate the eruption of smallpox by rubbing the skin with croton oil or tartar-emetic ointment.[2]

PROGNOSIS.—Unmodified or natural smallpox is attended with much danger to life, the ratio of fatal cases being as 1 to 3 or 4. The mortality is very great in cases of the confluent, and considerably less in the discrete, variety, the ratio in the latter being as 1 to 10. The death-rate varies between wide limits in different epidemics. The disease is much more fatal to children than to adults, and is especially fatal in early infancy. It is also extremely fatal to aged persons. Death is sometimes attributable to the intensity of the disease *per se.* Life is then destroyed before the eruption is matured and without any important complication having taken place. Instances of this kind are rare. If death take place before the stage of suppuration, it is generally due to some important complication. Complications which are likely to prove fatal are—acute laryngitis, œdema glottidis, pneumonitis, acute ulcerative endocarditis, and an affection of the kidneys involving uræmia. The latter is to be suspected in cases in which coma or convulsions occur.

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[1] Prof. Janeway calls special attention to the danger of mistaking purpura variolosa for malignant measles, typhus fever, ulcerative endocarditis, and purpura hæmorrhagica. Other valuable suggestions are contained in his article on "The Diagnosis of Certain of the Infectious Diseases," *Medical News*, July 17, 1886.

[2] For an example vide *Report of the Proceedings of the Illinois State Board of Health*, Springfield, Ill., June 29, 1883.

Death not attributable to complications is generally due to inability of the powers of the system to support the cutaneous affection; hence the danger, other things being equal, is proportionate to the amount of the eruption. Confluent smallpox involves a very extensive suppurative inflammation which is likely to lead to a fatal result by exhaustion. Unfavorable prognostics are—great prostration, frequency and feebleness of the pulse, typhoid delirium and other ataxic symptoms, absence or subsidence of redness and tumefaction of the face and extremities, cessation of salivation, diarrhœa, hemorrhage from the mucous membranes, and extravasations on the cutaneous surface.

Death taking place during or after the stage of desiccation is attributable to ulcerations of the skin, boils, abscesses, erysipelatous inflammation, gangrene, continued diarrhœa, etc.

In pregnant women the disease in its severe form is likely to induce abortion or miscarriage, and under these circumstances the result is generally fatal.

TREATMENT.—The treatment of smallpox embraces measures having direct reference to the eruption, and those indicated by the phenomena of the disease irrespective of the eruption; in other words, the treatment is local and general.

The general treatment involves the same principles as do the continued fevers. There is no special treatment. The disease will run its course, and hence the expectant plan is to be pursued. During the stage of invasion the intensity of the febrile movement may be lessened by the use of cold drinks, ice-water, lemonade, carbonated water, allowed as freely as the condition of the stomach will permit, by refrigerant remedies and by sponging the surface of the body with cold or tepid water. If there be hyperpyrexia the wet sheet and sprinkling with cold water may be resorted to, as practised in cases of typhus and typhoid fever. Refrigeration is not objectionable on the score of interfering with the appearance of the eruption. Cathartics are not indicated, but constipation is to be removed by mild laxatives or simple enemata. Remedies to relieve nausea or vomiting and cephalalgia are frequently indicated in this stage. It has been claimed that the sulphites have a modifying effect upon the disease, lessening its severity and danger. Sansom advises the sulphite of sodium in doses of 20 or 30 grs., given every third or fourth hour. He also regards as a useful remedy the sulphocarbolate of sodium, given in the same doses and with the same intervals. I have met with practitioners of experience who from their observations attributed to the sulphites a favorable influence over the disease.

During the stage of the eruption prior to suppuration palliative measures are indicated by continued nausea or vomiting, vigilance, restlessness, diarrhœa in some cases, etc. The various complications which are liable to occur will also furnish therapeutical indications. It is probable that the mineral acids are useful in this fever as well as in the continued fevers. Alimentation and supporting measures in some cases are important in this stage. These measures are important in the suppurative stage and the stage of desiccation in proportion as the eruption is copious and confluent and the general symptoms are indicative of asthenia. The principles which should govern the use of alcoholic stimulants and tonic remedies in conjunction with concentrated nutriment are the same as in all the essential fevers as well as in other diseases in which danger in the direction of asthenia is more or less imminent.

All the hygienic conditions which are so important in the treatment of typhus and typhoid fever are not less important in the treatment of smallpox; and of these conditions the freest possible ventilation is of especial importance.

The local treatment embraces measures to render the eruption abortive on the face or to prevent the disfiguration caused by pitting. The treatment for this end is called *ectrotic*, this term signifying to miscarry. The ectrotic treatment, extending more or less over the body, has been supposed to be useful by limiting the amount of cutaneous inflammation, and thereby the degree of secondary fever and danger from exhaustion. Of the various ectrotic measures which have been tried, the following have been found measurably successful:

1. The careful evacuation of the vesicles by means of a fine needle. This plan was advocated by Rayer. It calls for much patience on the part of the operator.

2. Evacuation of the vesicles and cauterization by means of a fine-pointed stick of the nitrate of silver. This also is a very tedious operation. It is highly recommended by Velpeau, Bretonneau, and others.

3. The application of the tincture of iodine once or twice daily by means of a brush while the eruption is papular. This plan was introduced by Crawford of Montreal and Samuel Jackson of Northumberland, Pa. The effect was observed by Sargeant in thirty cases, the treatment being limited to one side of the face. There was but little swelling on the side on which the iodine was applied; the vesicles were flattened; and the pitting, although not prevented, was evidently diminished.

4. The exclusion of light and air was recommended as an ectrotic measure by the late Dr. Picton of New Orleans. This is effected by covering the face with a plaster of some kind. Covering the face with gold-leaf, as practised by the Egyptians, according to Larrey, must be useful chiefly by excluding light and air.

5. The application of a mild mercurial ointment spread on cloth has been highly recommended by authors in different countries. The risk of producing the constitutional effects of mercury is an objection of considerable moment to this plan. Mercurialization is certainly not desirable in smallpox. In view of this risk Bennett was led to substitute for the mercurial ointment calamine saturated with olive oil, and this application he found to be usually effective. Skoda and Hebra recommend the application of compresses dipped in a solution of corrosive sublimate (grs. ij–iv to water ℥vj). Niemeyer preferred a weaker solution (gr. j to ℥vj).

6. The application of the subnitrate of bismuth and prepared chalk in equal parts twice daily, after smearing the surface with sweet oil, is stated to be an effective ectrotic measure by Hamilton of Illinois.[1]

7. Stokes advised the application of poultices over the face as the surest method of preventing disfigurement. They should be applied at the earliest

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[1] *American Journal of the Medical Sciences*, Oct., 1865.

period and continued to an advanced stage of the disease. He thought linseed meal the best article for the poultice. It should be spread on a soft material, such as French wadding, and covered with gutta-percha paper or oiled silk.

8. The application of collodion as an ectrotic application was first suggested in a letter to me by the late Dr. Brinkerhoff of California.[1] It was subsequently recommended by Aran of Paris. The collodion is to be applied once or twice daily by means of a brush. To be successful, the application must be made either while the eruption is papular or while the vesicles are quite small. Applied at a later period, it has seemed to me to increase the pitting. In the few cases in which I have seen this plan tried the application was agreeable to the patient.

A solution of gutta-percha in chloroform was used by Stokes, Graves, and Wallace. The *modus operandi* of the gutta-percha is similar to that of collodion, the effect being produced by the exclusion of air and by compression.

9. Carbolic acid has been used with success by Lemaire and Sansom. The acid is to be liquefied by alcohol, and applied by means of a camel's-hair pencil brush as soon as the contents of the vesicles begin to appear puriform.

10. The application of cold wet cloths to the skin is regarded by many as an efficient ectrotic measure.

Aside from ectrotic measures, the local treatment embraces evaporating lotions to abate undue heat, a weak solution of the chloride of lime as an antiseptic and to allay pruritus, soothing unguents in the suppurative stage, and absorbent powders. If ulcerations follow the falling off of the scabs, they require the same applications as ulcers occurring under other circumstances.

It should be an object of treatment to prevent the eruption from appearing on the conjunctival membrane. The continued application of compresses wet with cold water is advisable for this object.

With a view to prevent diffusion of the disease patients should not be discharged from hospital, or if treated at home allowed to go abroad, until the surface is completely free from scabs and repeated thorough ablutions have been employed. All articles of clothing worn during the disease should be left at the hospital, or in cases in private practice destroyed. Fumigations as a means of destroying the contagium should not be relied upon. Physicians, after visiting patients affected with this disease, should not see other patients until after having been sufficiently in the open air for the contagium to be dissipated by free ventilation. The hands should be washed before leaving the hospital or house, and change of dress before visiting other patients, especially young children, is to be recommended if not enjoined.

Modified Smallpox, or Varioloid.

Thus far, smallpox has been considered as produced by infected emanations or the contagium in fomites—that is, natural smallpox—in persons unprotected by vaccination. Materially modified as respects the events of its clinical history, and its severity notably diminished, the disease is commonly known as *varioloid.* This term is not to be understood as applied to a disease essentially distinct from, although resembling, smallpox. The name would imply this; but in all cases to which the term varioloid is correctly applied the disease is essentially variola.

Smallpox is modified, as a rule, in a remarkable degree when produced by inoculation, or, as it is also called, variolation. As a means of rendering the disease so mild as to be in a great measure divested of danger, and of preventing pitting, inoculation had been practised from time immemorial in

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[1] Vide *Buffalo Medical Journal,* vol. vi., 1851.

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China and Persia. Imported into Turkey, the practice was introduced thence into Great Britain by Lady Mary Wortley Montague in 1721. It was not adopted in France until 1764. In the same year of its introduction into Great Britain a large number of persons in Boston and the vicinity were inoculated by Zabdiel Boylston. Boylston was the first to note this important fact relating to variola produced by inoculation : namely, that the average period of incubation is shorter by several days than in cases of natural smallpox. Hence the production of natural smallpox may be anticipated and prevented by inoculation after known exposure to the miasm of the disease.

The eruption in cases of inoculated smallpox or after variolation is usually slight. The pocks rarely exceed one hundred. Many of them abort; that is, they do not proceed to suppuration. The secondary fever is slight or wanting. There is very little constitutional disturbance and the disease leaves little or no pitting. These statements are true in the main. Exceptionally, when inoculation was largely practised the disease was severe. The rate of mortality was exceedingly small, varying, according to Shattuck, in 23,000 cases in Boston between 5 and 30 deaths in 1000. Transferring the virus from persons successively inoculated, the disease becomes progressively more and more modified, until at length, as a rule to which there are exceptions, the eruption consists only of the pustule formed at the point of inoculation, with a few pustules developed around this mother-pustule.[1]

As a rule, variolation affords complete protection ever thereafter against smallpox. For those who availed themselves of it, therefore, it was a great blessing prior to the discovery of vaccination; but it contributed to the diffusion of the disease by multiplying the foci of contagion, and since the discovery of vaccination it has ceased to be resorted to as a means of avoiding the danger and disfigurement of natural smallpox. Under certain circumstances, however, the physician is not only justified but it is his duty to practise inoculation. These circumstances are the known exposure of persons who have never had the disease and who are unprotected by vaccination, and an inability to obtain the vaccine virus. Inoculation should then be resorted to in order to secure the great advantages of modified smallpox. The late Prof. Dickson advocated a restoration of the practice of inoculation, in addition to vaccination, with a view to test the protective power of the latter, and to destroy as completely as possible all susceptibility to the disease.[2]

At the present day in most cases of varioloid the modification is due to vaccination. Vaccination does not always afford complete protection against smallpox, but in general the disease is materially modified.

In varioloid the primary or eruptive fever in the stage of invasion is often as marked as in cases of natural smallpox. The duration of this stage may be longer than the average duration in ordinary variola. The eruption is usually far less copious; a few pocks only may appear, and the eruption may even be limited to a single pock. The eruption passes more rapidly through the successive changes, the maturation being completed in five or six days. Frequently the eruption aborts to a greater or less extent. It may stop at the vesicular, and even at the papular, period. More or less of the vesicles dry up without breaking. The general symptoms, as a rule, are much milder than in most of the benign cases of natural smallpox. The secondary fever is either slight or wanting, and the convalescence is speedy. Exceptionally, cases are severe, and the disease proves fatal in a ratio varying between 7 and 10 per cent.

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[1] Vide Trousseau, *Clinique médicale*, tome i.

[2] *Am. Journ. of Medical Sciences*, July, 1862.

Smallpox is modified in a certain proportion of cases spontaneously; that is, irrespective of inoculation or vaccination. Cases presenting the characters of varioloid occurred prior to the practice of inoculation and the discovery of vaccination. They were, however, rare. Finally, modified smallpox or varioloid is likely to be the form of the disease when it occurs in those who have before experienced it. In all cases of varioloid it is to be borne in mind that the disease is essentially variola or smallpox, and that, however mild may be the cases, they are capable of communicating to persons unprotected a severe and fatal form of the disease.

The DIAGNOSIS in cases of varioloid offers difficulties which do not obtain in cases of unmodified smallpox. The disease most likely to be confounded with varioloid is rubeola or measles. The different points will be stated in treating presently of the latter disease.

In cases of varioloid oftener than in ordinary variola the eruption is liable to be preceded and accompanied by an efflorescence bearing considerable resemblance to scarlatina. Until the variolous eruption is developed the disease may be supposed to be scarlatina. The occurrence of papules in the midst of the efflorescence and the remission or cessation of pyrexia are the differential points. Moreover, an examination of the mouth and throat may show the characteristic eruption here before its distinctive characters on the skin are apparent. The occurrence of a scarlatiniform eruption explains in some cases the supposed coexistence of variola or varioloid and scarlatina. An eruption resembling that of varicella has been observed in some cases of varioloid (variola varicelloides).

The TREATMENT of varioloid does not claim separate consideration. The same principles are involved as in the treatment of ordinary smallpox. The comparative mildness in the great majority of cases renders active treatment unnecessary. Usually little is required except hygienic measures.

Vaccinia, or Cowpox.

Vaccinia and cowpox are names of a disease of the cow which, communicated to man, destroys in the great majority of cases for a certain period the susceptibility to smallpox, and in the cases in which it does not offer complete protection against the latter disease renders it, as a rule, mild and devoid of danger. Cowpox is transferred to man and from one person to another by the introduction of a virus, and never at a distance by infection. The operation for its communication is called *vaccination*. For the employment of vaccination as a means of preventing smallpox the world is indebted to the immortal Jenner. The discovery was promulgated by Jenner in 1798, after a series of observations and experiments which had occupied his attention for twenty years. The fruits of this transcendently important discovery have been the saving of an incalculable number of lives which would otherwise have been destroyed by one of the most loathsome of diseases, and the prevention, to an extent which cannot be computed, of the disfigurement and other distressing effects which smallpox is liable to produce when it does not prove fatal. Of all the benefactors who have ever lived, no one has conferred on mankind such immense, palpable, and time-lasting benefits. Yet outside of the ranks of the medical profession how many are ignorant of the name of the discoverer of vaccination! The practice of vaccination met at the outset with much hostility, but Jenner lived to see it triumph over all opposition, and to witness the beginning of beneficent results which will accumulate from age to age until the end of the world. The practice was introduced into this country in 1799, notwithstanding a bitter popular prejudice, by Benjamin Waterhouse of Boston, the first professor of medicine in Harvard University.

Connected with vaccinia and vaccination are questions of great interest and importance which have given rise to extended experimental and statistical researches, as well as much discussion. They have occupied a considerable share of medical literature since the time of Jenner. Certain points are still unsettled and claim further investigation. The scope and character of this work allow of only a brief statement of facts and the opinions most consistent with existing knowledge, considered with direct reference to the practice of medicine.

Directing attention first to the descriptive history of vaccinia in man, on the third day after vaccination (the operation being usually performed on the arm near the insertion of the deltoid muscle) red points slightly elevated—that is, small papules—are apparent at the spots where the vaccine virus was inserted. On the fourth day the papules are more developed and reddened. On the fifth day vesicles are discoverable. The vesicles increase, presenting an umbilicated appearance, and on the eighth day they attain their full development, being elevated two to three lines and measuring one-third of an inch in diameter. The vesicles, like those of smallpox, are multilocular, flattened at the summits, and contain a transparent viscid liquid called the vaccine lymph. On the seventh or eighth day a red areola is apparent, extending one to three inches around the pocks, increasing in redness until the ninth or tenth day, and the contents of the vesicles become more or less purulent. At this time there usually is slight pyrexia with some local pain and pruritus. The lymphatics of the arm become swollen and the glands in the axilla may become enlarged and tender. On the tenth or eleventh day the pyrexia subsides and the redness around the pocks diminishes. A dark spot soon appears in the centre and gradually extends over the whole of the pocks. The pustules desiccate, and at about the fifteenth day they are converted into black hard scabs, which fall off usually at about the twenty-fifth day from the date of the vaccination. During the progress of the vaccine affection vesicles having the distinctive characters of vaccinia have sometimes been observed on other parts of the body. It is probable that these are caused by the patient scratching the vesicles on the arm and carrying therefrom lymph containing the virus on the finger-nails to parts where, owing to the abrasions of the skin, self-vaccination is the result. Experiments show that between the fourth and the ninth or tenth day the characteristic vesicles may be multiplied at will by revaccinating with lymph from the vesicles produced by the primary vaccination. Lichenous or vesicular eruptions, the latter lacking the distinctive features of vaccinia, not infrequently accompany or follow vaccination. Finally, permanent cicatrices denote the situation of the vaccine pocks. The cicatrix, provided the vesicle have pursued a regular course and subsequent ulceration have not occurred, is characteristic, presenting a series of depressions or pits. The period of incubation in vaccinia is sometimes considerably protracted in consequence of the existence of some other affection or from causes which are not apparent.

A most important practical point connected with vaccination is the discrimination of veritable and complete vaccination from an incomplete or a spurious affection. The development of vesicles of full size, the successive stages, and the duration of the affection, together with the characteristic appearance of the vesicles and the cicatrices, are the criteria for determining this point. Simple sores having no specific character may be the result of vaccination. Incomplete vaccinia—*vaccinoid*, as it was termed by Trousseau—is denoted by the vesicles being developed more quickly and progressing to desiccation more rapidly, by their swollen size and conoidal form, and by the absence of cicatrices or of the appearance distinctive of the true vaccine cicatrix. In view of the immense importance of a true and perfect vaccination to those who suppose themselves to be protected against smallpox, the responsibility of performing vaccination should never be undertaken by those not practically familiar with vaccinia. It is obviously far better that a person should not have been vaccinated, with a knowledge of the fact, than that he should incorrectly believe himself to have had veritable and complete cowpox, since this belief involves a false security leading to exposures which might otherwise be avoided.

It is now generally believed that cowpox is not identical with smallpox, although allied to it. The virus obtained from the the cow by inoculating with the smallpox virus (retro-vaccination, as it is called) is not only unreliable as affording protection against smallpox in man, but it has been found repeatedly to communicate the latter disease. Vaccine virus thus obtained is therefore never to be employed in vaccination.[1] Experiments seem to have confirmed the correctness of the opinion held by Jenner that an affection sometimes seen in horses, known as *grease*—called by the French *des eaux jambes*—is identical with cowpox, and that vaccinia in man may be derived from this source. As to the origin of the disease in the cow, the view is entertained that cowpox is a disease *sui generis*, and there is no positive proof that it is usually or necessarily derived from any other animal. The vaccine lymph contains bacteria, but whether these organisms represent the contagium of the lymph or not is a question still undecided. The experiments of Chauveau have rendered it probable that the contagium is a molecular substance, and is not dissolved in the lymph.

Other questions, the importance of which is obvious, are, What degree of protection against smallpox is afforded by cowpox? and, What are the causes of the protection not being universal and complete? In a large majority of cases vaccination affords, for a time at least, absolute protection against smallpox. In these cases the situation of persons vaccinated, as regards susceptibility to the latter disease, is precisely as if natural or inoculated smallpox had been experienced; but in a minority of cases the protection is only partial. The susceptibility to smallpox is not destroyed, but the disease is materially modified, constituting what is called *varioloid*. At certain times and places, the causes which give rise to the epidemic prevalence of smallpox being unusually efficient, a considerable number of vaccinated persons become affected with varioloid. Owing, however, to the comparative mildness of varioloid, the rate of mortality is very small, and therefore fails to represent the relative number of vaccinated persons who become affected with modified smallpox. It is to be remarked that smallpox in a certain proportion of cases is experienced a second time. From the following statistics it would appear that not much more protection is afforded by the occurrence of variola than by vaccination: Of 5774 boys admitted into the Royal Military Asylum at Chelsea, England, in forty-eight years, 1950 had, on admission, marks of smallpox, and 3824 either had marks of vaccination or they were at once vaccinated. Of the former, 6.15 per 1000, and of the latter, 7.06 per 1000, contracted smallpox subsequently during their residence in the asylum.[2]

The opinion widely prevails, and appears to be well founded, that the extent of protection afforded by cowpox is less in the latter than in the former part of the period which has elapsed since the discovery of vaccination; that is, a larger proportion of vaccinated persons have had varioloid of late years than heretofore, and hence many have been led to entertain the belief that the vaccine virus has deteriorated in consequence of its transmission from person to person.

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[1] Vide *Journal of the Gynæcological Society of Boston*, April and June, 1872, report of discourse by Dr. Henry A. Martin.

[2] Vide *Reynolds's System of Medicine.*

Another reason for the increase of varioloid among those who have been vaccinated is, that the protective influence of vaccination diminishes after the lapse of a certain number of years. Varioloid occurs much oftener among those more or less advanced in life than among the young. The length of time which has elapsed after a vaccination may fairly be considered as entering into the etiology of varioloid. The importance of revaccination rests upon this fact. Revaccination after the lapse of a greater or less number of years is completely successful, as regards the production of the vaccine disease, in a considerable proportion of cases, and partial success, as shown by imperfect or vaccinoid vesicles, is still more common. The epidemic prevalence of smallpox, embracing many cases of varioloid among vaccinated persons, has been promptly and completely arrested by universal vaccination and revaccination.

The importance of revaccination being admitted, the inquiry arises, How often should it be resorted to? There is no known law governing the duration of the protective influence of a single vaccination. Probably the duration varies widely in different persons, and some persons are protected by one vaccination for life. Some have thought that revaccinations should be practised as often as every three years, and others that an interval of twenty years or more is allowable. It is evidently better that the period should be needlessly short than too long. With our present knowledge the propriety, if not importance, of revaccinating every five years is to be advocated. In case of known exposure or when smallpox prevails as an epidemic it is proper to revaccinate without regard to previous vaccinations. Revaccination, in fact, is always proper as the readiest and safest test of insusceptibility to smallpox.

There is ground for the belief that the frequent occurrence of varioloid, and also of unmodified smallpox, among those who suppose themselves to be protected by having had cowpox is due in no small measure to imperfect or spurious vaccinations. In this country vaccinators are often unqualified persons. The operation is performed not infrequently by those who make no pretensions to medical knowledge, and to a great extent by irregular practitioners. In many cases persons vaccinated are never seen after the operation, they or their friends undertaking to judge for themselves of its success. Hence many persons are either wholly unprotected or but partially protected.

A cause of inadequate protection is an insufficient number of vaccine vesicles. Many have supposed that a single vesicle is all that is required, and that the only advantage of inserting the virus in more than one spot is to diminish the risk of failure to procure a single vesicle. In many parts of this country it is not customary to procure more than one or two vesicles. Facts show that the number of vaccine-vesicles has a decided influence on the amount of protective influence of cowpox. The following results of the analysis, by Simon, of nearly 6000 cases of smallpox contracted after vaccination, with reference to the proportion of deaths to the number of cicatrices, are interesting connected with the present topic: The ratio of deaths among those who stated that they had been vaccinated, but who presented *no* vaccine cicatrix, was 21¾ per cent.; among those who had *one* cicatrix, 7½ per cent.; among those who had *two* cicatrices, 4⅛ per cent.; among those who had *three* cicatrices, 1¾ per cent.; and among those who had *four or more* cicatrices, ¾ of 1 per cent.[1] In a primary vaccination it is desirable that from four to six vesicles should be produced on each arm. The number should not be less, after known exposure to smallpox, in secondary vaccinations. A less number, however, may suffice when it is desired only to test the fact of protection by previous vaccinations.

Within the past few years an important practical question relating to vaccination has arisen. In 1866 a case of spontaneous cowpox was discovered at Beaugency in France. The virus obtained from this animal was perpetuated, through successive inoculations in heifers conducted by Depaul under the sanction of the French Academy of Medicine and with the aid of the French government. The virus from this stock having been imported into this country by Dr. Henry A. Martin of Boston in 1870, he has kept up the supply through a continued series of heifer vaccinations; and since that date the supply has been kept up from virus derived from this stock in New York and some other cities. The terms animal and bovine virus are used to distinguish that derived immediately from the heifer, that derived from the human subject being called humanized virus. The important practical question is whether the animal virus affords greater protection against smallpox than the humanized virus? That the latter has deteriorated in consequence of removes from the cow seems certain. Vaccination with the long-humanized virus is less constantly successful in producing vaccinia than formerly; the revaccination of persons who have been vaccinated with this virus is oftener successful, and a diminished protection is shown by the greater number of vaccinated persons who become affected with varioloid. The opinion that vaccination with the animal virus secures a more complete and a longer exemption from the danger of contracting smallpox, is shared by many, and this virus is now extensively used in this country instead of the humanized virus. It remains to be determined by the results of a long experience whether—assuming that at the present time the animal virus has a greater protective power—this power may not become deteriorated by distant removes from Beaugency stock in heifers, as there is reason to believe has been the case in the human race. This, however, is a consideration which concerns the future; and a deterioration, if it occur, would only render it important to take advantage at different periods of other instances of vaccinia of spontaneous origin[1] in the cow, in this way renewing the stock.

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[1] Aitken, *op. cit.*

Vaccinia in man produced by the animal virus differs in certain respects from the disease produced by the long-humanized virus. The full development of the vaccine pock requires a longer period; the pock continues to increase for several days after the eighth day, this being the period required for the full development of the pock after vaccination with the long-humanized virus; and the pock is of larger size. The crusts are not formed until the eighteenth or nineteenth day, and they remain adherent until between the twenty-fifth and the thirty-fifth day. The vesicle does not spontaneously rupture, and the crust preserves the form of the vesicle with the umbilicated centre. The constitutional symptoms are more marked. In a small proportion of cases (1 in 50) a vesicular eruption occurs over the body, and the vesicles on close inspection are found to be umbilicated. The development of a roseolar eruption after vaccination has also been observed, but this is rare. With respect to these points of difference, the disease as now produced by the animal virus corresponds with the disease as described and delineated by the earliest vaccinographers—a fact which in itself renders probable the deterioration of the humanized and the superiority of the animal virus.[2]

Assuming that the animal or bovine virus is equal to the humanized in its protective power (and at least this must be conceded), there are certain advantages which the former has over the latter. There is far less liability to local accidents—namely, ulcerations and erysipelas. Dr. Martin states that in April, 1872, there had been at least 400,000 revaccinations made in this country with the Beaugency virus, and no case of death, of erysipelas, or of post-vaccine variola had been reported among them. Another advantage is a more uniform success in both primary vaccinations and revaccinations. Another is that the supply of virus can always be made to meet the demand. Again, an advantage which has only to be named for its importance to be apparent is the fact that the virus from the heifer is incapable of communicating syphilis. Granting that the risk of syphilis is small when humanized virus is used, there is nevertheless a risk; and syphilis has repeatedly been communicated. It is an immense advantage to eliminate all danger in this direction.

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[1] The term *spontaneous* is here used to distinguish it from the disease produced by retro-vaccination. Whether cowpox be ever in a broader sense spontaneous is an open question.

[2] Vide Discourse by Dr. Martin in *Journal of the Gynæcological Society of Boston;* also, "Practical Remarks on Vaccination," by Dr. Frank P. Foster, in *Neumann's Hand-book of Skin Diseases*, translated by Bulkley, and in article on "Vaccination" in *Pepper's System of Practical Medicine, by American Authors*, Philadelphia, 1885, vol. i.; also, *Sur le Cowpox découvert à Passy*, par Bousquet, Paris.

Deterioration of the humanized virus is to be added tô the causes previously enumerated of the apparent inadequateness of vaccination to afford complete protection against smallpox. Assuming that this obstacle may be overcome by the use of animal virus, the other causes are removable; and were they to be entirely removed it is not improbable that the expectation which Jenner cherished of exterminating smallpox might be realized. Certain it is that were all the requisites for the most perfect vaccination to be invariably observed, and if all persons living were vaccinated and revaccinated, smallpox, if not exterminated, would be insignificant as regards its prevalence and severity. The difficulty of securing for all mankind a protection against the disease almost complete is not so much in the insufficiency of the means as in enforcing their adoption to the fullest possible extent. Vaccination and revaccination, in order to be universal, must be compulsory, and authoritative measures to secure the proper and successful performance of the operation are essential. To consider the most efficient legal provisions to accomplish these desirable ends would be to open up a wide field for discussion, which would be inconsistent with the objects of this work. Strange as it may seem, there are persons, not without intelligence, at this day who entertain toward vaccination the prejudices which Jenner had to encounter.

It remains to notice some practical points relating to the operation for vaccination. The situation for the introduction of the vaccine virus should always be that usually selected—namely, on the arms near the insertion of the deltoid muscle. Uniformity of practice in this respect enables physicians and others to ascertain without delay or doubt the fact of a previous vaccination, and to judge of its success by an examination of the cicatrices. The vaccine lymph is to be preferred to the crust. The lymph is usually taken on ivory points or pointed quills, but the virus taken in this way to be effective must be recent. With a view to preservation the lymph may be withdrawn from the vesicles in glass capillary tubes which are at once sealed by means of a spirit-lamp. Jenner considered it a "golden rule" never to take the virus from a vaccine vesicle for the purpose of vaccination after the efflorescence is formed around it. A crust enclosed in wax and covered with tin-foil retains its activity for a considerable period.

As regards the mode of vaccinating, if dried lymph be used or lymph contained in capillary tubes it may be inserted by means of slight punctures, or by scraping away the epidermis, or by minute scarifications; the latter method being preferable. Having made, with a common lancet, a number of linear and transverse scratches, not deep enough to cause the escape of more than a drop or two of blood, the lymph is to be applied over the scarified spots, which should be five to six in number and not larger than half a dime. If the incisions be sufficiently delicate, the operation gives no pain, and allowing the spots to dry before being covered by the clothing is all that is required.

If the crust be used, a small portion should be dissolved, always at the time of the operation, in a drop or two of water, and applied by means of the lancet over the scarified spots. This is preferable to making punctures and introducing small pieces of the undissolved crust. The latter mode is liable to cause sores which, aside from the annoyance they occasion, may interfere with the specific action of the virus.

The age for the performance of a primary vaccination is a point of importance. Between the second and the third month of infancy is the period to be preferred for vaccination. Any liability to exposure to smallpox, however, renders the operation advisable at an earlier period. It is desirable that the child when vaccinated should be in good health, but the operation should not be delayed on account of illness if there be a liability to exposure to smallpox. If the operation prove unsuccessful, another trial should be made, and if this fail the virus may be introduced a third time. Some persons are insusceptible to cowpox. An insusceptibility is to be inferred from two or three successive failures with virus which has proved successful in others. The attempt to vaccinate, however, should be repeated from time to time, for susceptibility may afterward exist. So long as there is insusceptibility to cowpox there is little danger of smallpox being contracted. Aside from want of susceptibility, and exclusive of inertness of the lymph or crust used, the common cause of failure is undue bleeding from the scarifications, the flow of blood preventing the absorption of the virus.

Troublesome ulcerations occasionally follow vaccination, arising from irritation by the friction of clothing or from an unhealthy condition of the system. These are to be treated like ordinary sores. Erysipelatous inflammation and abscesses may occur. These concomitants or sequels are not always evidence of any fault in the operation or impurity of the matter used. Dr. Henry G. Piffard, however, has reported cases in which the disease of the skin known as impetigo contagiosa has occurred as a sequel of vaccination with the virus in crusts both animal and humanized. The liability to the communication of this affection is a reason for preferring lymph to crusts for vaccination.[1] Ignorant persons sometimes attribute to vaccination diseases of any kind which may occur within a few weeks or months afterward, and to charge the physician with having used impure vaccine matter. This impression may be so rooted as not to be removable, and hence a vaccination may very unjustly prove the occasion for a withdrawal of the confidence which the physician had previously possessed. So far as the imputation of communicating human diseases is concerned, it is avoided by using the animal virus; and this is a recommendation of the latter to be added to the advantages already mentioned.

After a known exposure to smallpox, if the person exposed have not been recently vaccinated or if there be any reason to distrust a previous vaccination, revaccination should of course be promptly performed. It is proper to perform it without reference to previous vaccinations, with a view to test the susceptibility and render the security as complete as possible. It has occurred to me repeatedly to vaccinate, under circumstances involving the utmost possible exposure, persons who had never been vaccinated, and thereby to prevent the occurrence of smallpox. In one instance an unvaccinated child lived in the same room and slept with a person who had contracted smallpox. The first operation proving unsuccessful, it was repeated three days afterward with success, and the child escaped. In another instance a patient with measles was supposed to have smallpox and was sent to a smallpox hospital. He had never been vaccinated, and vaccination was at once performed. The vaccine disease was deferred until the measles had run its course. It then became developed and smallpox did not occur. Owing to the relatively short duration of the period of incubation in cowpox, it anticipates the development of smallpox. I have not observed the coexistence of the two affections under these circumstances.

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[1] Vide articles by Dr. Piffard in the *New York Med. Journal*, Nos. for June and July, 1872.

Varicella, or Chicken-pox.

Varicella—or, as it is commonly called, *chicken-pox*—is an affection quite insignificant as regards danger or distressing symptoms. It is important with reference, *first*, to the question as to its having pathological relations with smallpox, and *second*, to its discrimination from modified variola or varioloid.

It is an eruptive disease affecting usually children, but occasionally adults. The eruption is preceded generally by slight constitutional symptoms for about twenty-four hours. The eruption generally appears first on the body, and afterward on the head. It usually is more abundant on the neck, body, and scalp than on the face. The eruption is frequently quite small and is very rarely abundant. It is almost invariably discrete. It is a vesicular eruption from the first. The vesicles contain at first a transparent liquid, which afterward becomes opaline, but not purulent except when they are irritated by scratching. Desiccation occurs between the fifth and the seventh day. The crusts are granular and are rarely followed by cicatrices or pitting. Sometimes bullæ of greater or less size form instead of vesicles. New vesicles frequently continue to appear during the first three days of the stage of the eruption, and even after the desiccation of those which first appeared has begun. During the eruptive stage constitutional symptoms are slight or wanting. The disease is contagious as regards children, but very rarely as regards adults. Experiments made to determine its communicability by inoculation have so generally failed that it is a question whether, in the few instances in which it was apparently successful, the contagion may not have been otherwise received. The period of incubation varies between twelve and seventeen days. Writers have made several varieties based upon differences as regards the size and shape of the vesicles. The ordinary variety, in which the vesicles are about the size of split peas and flattened at the top, has been distinguished as *varicella lentiformis vel lenticularis*, or lenticular chicken-pox. The variety called *varicella coniformis vel conoides*, or *swine-pox*, is characterized by the conoidal form of the vesicles. *Varicella globularis vel globata*, is the name of a variety in which the vesicles are unusually large and are globular in form. The different vesicles in the same case not infrequently present the characters of these three varieties. The division into varieties is of not the least practical consequence.

The name varicella, which is a diminutive of variola, meaning *little smallpox*, implies that the disease is essentially variolous. It has been considered as a modified form of variola, and is so considered by some eminent teachers and writers at the present time. The non-identity of the two affections is established by the following facts: The contagium of varicella is never known to give rise to ordinary variola, whereas the severest form of the latter disease may be derived from cases of varioloid. Varioloid prevails only during the prevalence of ordinary smallpox, but varicella not infrequently prevails when there are no cases of smallpox. Varicella is very rarely communicated to adults. This is not true of varioloid. Varicella affords no protection against variola, and, conversely, persons who have had smallpox contract varicella. Vaccination does not protect against varicella, and the latter does not interfere with the success of vaccination. It is not settled that varicella is ever communicable by inoculation. Other points of difference, showing the non-identity of varicella and varioloid, relate to the clinical history, and upon these points the differential diagnosis is to be based.

Although pathologically distinct, it must be confessed that the discrimination of varicella from varioloid is not always easy. The two affections are liable to be confounded. Cases of varioloid are not infrequently at first considered as cases of varicella, and *vice versâ*. In view of the great importance of making this differential diagnosis promptly and positively, the following diagnostic points claim the careful attention of the student and practitioner:

The stage of invasion in varioloid is as long as, or longer than, in cases of ordinary smallpox—namely, from two to three days. The short duration of this stage in varicella is a distinctive feature. The constitutional symptoms in this stage are often as marked in varioloid as in cases of ordinary smallpox. The mildness of these symptoms in varicella is a diagnostic point. The vesicular character of the eruption from the start is characteristic of varicella. In varioloid the vesicles are preceded by papules. In varioloid, as in ordinary smallpox, the eruption appears at first and especially on the face. In varicella it appears first on the body, and is likely to be more abundant elsewhere than on the face. A capital diagnostic point relates to the central depression or the umbilicated appearance. This is generally discoverable in more or less of the vesicles in varioloid, and it is generally, if not invariably, wanting in varicella. The duration of the eruptive stage is less in varicella than in the great majority of cases of varioloid. Finally, traces of vesicles in varicella are rarely found on the mucous membrane of the mouth, fauces, and genitalia.

It is important, as has been urged especially by Janeway, to trace doubtful cases of all infectious diseases to their origin, and also to note the cases which are transmitted by the disease in question, as these other cases may be more distinctive in their characters.

During the prevalence of smallpox cases of varioloid in which the eruption is small are often considered as cases of varicella. The cases of so-called varicella in adults when smallpox prevails are generally cases of varioloid; hence, one source of the diffusion of smallpox.

Varicella claims no treatment. It is important to recognize it only in order to relieve apprehensions by the assurance that the disease is not varioloid.

CHAPTER VII.

ERUPTIVE FEVERS (CONTINUED).

Scarlatina, or Scarlet Fever: Anatomical Characters; Clinical History; Causation; Diagnosis; Prognosis; Treatment.

Scarlatina, or Scarlet Fever.

THE exanthematous fever called, from the color of the eruption, *scarlatina* or *scarlet fever*, is remarkable for the wide diversity which it presents in different cases as regards symptoms and fatality. In its mildest form it is a trivial affection, but in its severest form there are few diseases more appropriately styled malignant. The disease has been divided into several varieties based on differences in symptomatic characters and intensity. Three varieties are generally recognized by writers—namely, *scarlatina simplex*, *scarlatina anginosa*, and *scarlatina maligna*. Widely different as are these varieties in respect to gravity and distinctive characters, they are not distinct diseases, and essentially they are identical. A simpler division is into mild and severe scarlatina, but it will suffice to consider incidentally the events which belong to the latter, without treating of it separately.

Anatomical Characters.—The eruption on the surface and the morbid appearances in the throat constitute the leading anatomical characters in this affection. There is usually little or no trace of the eruption to be observed after death. The eruption is caused by hyperæmia, combined with more or less œdematous swelling of the cutis. In a case in which death took place at the height of an intense scarlatinal eruption, Birch-Hirschfeld observed many lymphoid cells between the epithelial cells of the rete Malpighii, as well as in the superficial and deeper layers of the corium. He also saw small extravasations of blood. Fenwick has called attention to blood-extravasations in the lumen of the sweat-glands. The cutaneous affection of scarlatina is to be regarded as a superficial dermatitis of mild degree.

In the mildest form of scarlatinal sore throat the mucous membrane covering the tonsils, soft palate, and pharynx is reddened and slightly swollen. The tonsils are frequently enlarged. A phlegmonous inflammation of the parts named may occur in severe cases of the variety of the disease designated as scarlatina anginosa. The swelling is then extreme. The submucous and the mucous layers become infiltrated with pus-cells. Abscesses form, especially in the tonsils. The phlegmonous inflammation may terminate in destructive ulceration or in gangrene. The phlegmonous inflammation may be propagated to the connective tissue in the submaxillary region, and give rise to extensive purulent collections in this situation. Erosion of the large vessels has been caused by this suppurative process in the neck. In some epidemics diphtheritic inflammation of the throat has been a frequent attendant upon scarlet fever. The fibrinous exudation begins on the tonsils and extends to the soft palate and to the pharynx. It has less tendency than primary diphtheria to extend into the larynx. There is much controversy as to the relation which this diphtheritic affection of the throat bears to scarlatina. There are three prominent views as to this relation. According to one view, the diphtheritic affection is anatomically the same disease as primary diphtheria, but it is produced by the scarlatinal poison; according to a second view, the disease, both anatomically and etiologically, is identical with primary diphtheria, and it is to be regarded as a complication of scarlet fever; according to the third view, scarlatinal diphtheria differs in its anatomical characters from primary diphtheria. Heubner has attempted to prove, from an anatomical point of view, the non-identity of scarlatinal diphtheria and primary diphtheria, but the differential criteria to which he calls attention are not sufficient to distinguish anatomically one disease from the other.[1] The first of the three views mentioned is the most probable. The inflammatory process may be propagated from the pharynx along the Eustachian tubes to the mucous membrane lining the cavity of the middle ear. Suppurative median otitis, with perforation of the tympanum, and sometimes with consecutive caries of bone, may thus be produced. Swelling and inflammation of the lymphatic glands of the neck not infrequently accompany scarlatinal angina. The glands may suppurate. The mucous membrane of the larynx, trachea, and bronchi is sometimes inflamed. The inflammation in this situation is rarely accompanied by a fibrinous exudation. Lobular pneumonia is an occasional complication of scarlatina. Lobar pneumonia is less frequent. Fenwick has observed parenchymatous degeneration of the gastric tubules in this disease. Peyer's patches and the solitary follicles of the intestine are often moderately swollen. There may be found ecchymoses in the gastric and intestinal mucous membrane, but they are rarely abundant. Dysentery is a rare complication of scarlet fever. The spleen has often been found enlarged. Swelling of the mesenteric glands, as well as of other lymphatic glands of the body, has been observed in a number of cases. Parenchymatous degenerations of the liver, kidneys, and heart occur in scarlet fever. In some cases in which the immediate cause of death has been failure of the heart-power fatty degeneration of the muscular substance of the heart has been present.

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[1] The points to which Heubner calls attention are: 1. The pseudo-membranous deposits are much thinner in scarlatinal than in primary diphtheria, so that in the former disease membranes are not expectorated. 2. The fibrin in scarlatinal diphtheria is found between the epithelial cells and in the connective tissue of the mucosa and submucosa. He could detect no differences between the micrococci present in scarlatinal diphtheria and those of primary diphtheria ("Beobachtungen über Scharlach-Diphtherie," *Jahrbuch der Kinderheilkunde*, 1879, xiv. p. 1).

The most important sequel of scarlet fever is acute Bright's disease. Some regard this occurrence as a part of the scarlatinal process, and others, as a complication. The kidneys in cases of scarlatinal nephritis present appearances which have been described in the article on Acute Bright's Disease (p. 865). Sometimes small abscesses form in the kidneys in this disease.

CLINICAL HISTORY.—The symptomatic events of the several stages separately will be first noticed, and afterward anomalous events and complications liable to occur in either of the three stages.

Stage of Invasion.—The attack may begin with a chill more or less pronounced, but not infrequently it is wanting. Vomiting is generally an early symptom, especially in children, with, in a certain proportion of cases, diarrhœa. The pyrexia, as a rule, is notably greater than in the other eruptive fevers. Epistaxis is not uncommon. Redness of the fauces more or less vivid may generally be observed in this stage, either with or without a sense of soreness and pain in swallowing. The affection of the throat is due to the efflorescence which appears in this situation before its appearance on the skin. As regards the intensity of the pyrexia, pain in the head, prostration, and general malaise, different cases differ widely, not including cases of unusual severity. In very mild cases the symptoms are so slight that the patient does not take to the bed.

The average duration of this stage is about twenty-four hours, and the eruption usually appears on the second day. Exceptionally, it appears, on the one hand, a few hours after the attack, and on the other hand it may be delayed one, two, three, or more days after the time when it usually appears. A considerable delay in the appearance of the eruption is generally attributable to some complication.

Stage of Eruption.—This stage in children is sometimes ushered in by a transient convulsion, which at this period is not a uræmic symptom. In its development and diffusion over the body the eruption does not observe the same regularity as in smallpox and measles. It oftener appears on the body and limbs before making its appearance on the face and neck, but in a certain proportion of cases it appears first in the latter situations. It is rapidly diffused, extending more or less over the whole cutaneous surface in twenty-four hours. As regards the degree and extent of the efflorescence, different cases differ much. The first appearance is in the form of minute dots or specks. These coalesce, forming irregularly distributed patches, which vary much in size and shape, having irregular or serrated margins. Exceptionally, the eruption remains punctiform; that is, in the form of distinct maculæ or spots. The redness is most vivid and remains longest in situations where the skin is delicate, as the inner surfaces of the arms and thighs and the flexures of the joints. The redness is vermilion or scarlet; it is not uniform like erysipelatous redness, but on close inspection the patches are seen to be studded with points which are distinguished by a more deeply red color. The redness momentarily disappears on pressure, and white lines are produced by drawing a pointed hard substance—for example, a pencil—over the reddened surface. In some cases the whole cutaneous surface is covered with the efflorescence, presenting an appearance which has been compared to that of a boiled lobster. Generally, on passing the fingers over the reddened surface it is felt to be smooth, but in some cases there is a sensation of minute elevations or papules. These are the papillæ abnormally developed, as in the condition commonly known as goose-skin. Occasionally, miliary vesicles are scattered more or less abundantly over the surface. The integument is slightly swollen. This is evident on the face and is apparent to the patient when the hands are closed. The feet are also evidently somewhat swollen. The eruption is sometimes accompanied by more or less burning and pruritus. The eruption attains its maximum of intensity and diffusion on the third day after its first appearance.

There is much variation in different cases in the amount of cutaneous eruption. It is sometimes slight. This is likely to be the case when the throat affection is unusually severe. The eruption then seems to be concentrated within the throat. Occasionally the efflorescence on the skin disappears shortly after its appearance, and afterward returns. It fluctuates as regards vividness, being especially marked in febrile exacerbations, and it is generally more vivid toward evening than in the early part of the day. In anomalous cases, which will presently be referred to, the eruption is wanting.

The throat is more or less affected in the vast majority of cases, but in some very mild cases there is no throat affection. If the throat be but slightly affected, there is simply redness over the tonsils, the posterior part of the pharynx, and extending more or less over the soft palate, with little or no swelling. The affection is then only an efflorescence. In most cases, however, there is more or less swelling of the tonsils, together with a white or ash-colored exudation in greater or less abundance. This exudation is generally not like that which characterizes diphtheritic inflammation. It adheres less closely, is pultaceous, and cannot be detached in strips. It may receive a dark color from matters vomited and from the oozing of blood. In proportion to the amount of throat affection there is pain in deglutition and the voice becomes nasal. The submaxillary and lymphatic glands at the angle of the jaw frequently are swollen and tender. A still greater degree of throat affection belongs among the anomalous events of the disease and characterizes the variety distinguished as scarlatina anginosa.

The tongue early in the disease generally is coated. While the coating remains, frequently the papillæ projecting through it have the appearance of a number of red points, and the surface of the tongue looks as if cayenne pepper or red sand had been sprinkled over it. This is seen in other affections. Another appearance is quite distinctive of scarlet fever. In the progress of the disease the coating exfoliates, leaving the surface of the tongue clean and reddened; and, the papillæ being enlarged, the appearance is strikingly like that of a ripe strawberry. The strawberry-like tongue is a pathognomonic symptom, and is peculiar to this disease. It is often but not uniformly present. It is due to a condition of the mucous membrane analogous to that of the skin in the cases of so-called *rough* as distinctive from *smooth* scarlatina.

The pyrexia is not diminished, but, as a rule, is increased, after the appearance of the eruption. The frequency of the pulse and the increased heat of the skin are more marked than in any other of the essential fevers. The pulse varies in different cases—which, although more or less severe, pursue a favorable course—between 100 and 130 or 140 per minute. In extremely severe cases, especially in children, it rises still higher. Aside from frequency, the pulse is quick, often vibratory or thrilling, but always compressible, these characters showing excited action without augmented power of the ventricular contractions. The sphygmograph, as in all the eruptive fevers, shows absence of dicrotism. The skin usually is dry, and the heat, as felt by the hand, is often pungent. The thermometer in the axilla shows an increase of temperature to 105° F., and in severe cases a still greater increase. It has been known to reach 112° F.

During the stage of the eruption anorexia continues, vomiting is not uncommon, constipation exists in some cases, and in some cases there is diarrhœa. Thirst usually is urgent. Delirium, manifested by incoherency, generally exists, save in very mild cases. I have known a patient, not considered ill enough to require a watcher during the night, to get up and wander out of doors in a state of delirium. A melancholy instance, illustrative of the importance of watchfulness in the care of patients in the delirium of fever, occurred many years ago at the Louisville Marine Hospital during my term of service. A female adult patient affected with scarlatina, but not considered seriously ill, had been removed to a room by herself in the uppermost story of the hospital building, with a view to isolation. During the absence of her attendant for a few moments she precipitated herself from the window, and, falling upon the pavement, was instantly killed. Restlessness, jactitation, and insomnia are likely to be more or less marked. Albuminuria occurs in a certain proportion (about one-seventh) of cases during the eruptive stage. Transient, slight albuminuria occurs in scarlatina, as in various other affections, without denoting renal disease; but disease of the kidneys forms an important complication in certain cases of scarlatina, and will presently be noticed. The duration of the stage of eruption in the majority of cases is between four and six days. It is sometimes shorter; oftener it is prolonged to eight or ten days; and occasionally it continues for a longer period.

Stage of Desquamation.—The commencement of desquamation marks the beginning of this stage. Desquamation frequently begins with the decline of the eruption and prior to its disappearance. The degree and extent of the desquamation are in general proportionate to the intensity and diffusion of the efflorescence. Desquamation is rarely wanting, save in the cases in which the efflorescence fails to appear. The cuticle is exfoliated either in the form of minute branny scales, called furfuraceous desquamation, or in pieces of epidermis of greater or less size. Occasionally the epidermis of the hands is detached entire and may be stripped off like a glove. This is true also of the epidermis of the feet. Sometimes several successive exfoliations occur.

In cases which pursue a favorable course the febrile movement diminishes with the beginning of exfoliation, and gradually ceases, corresponding improvement taking place in other symptoms. The defervescence is sometimes rapid, but in most cases it is gradual. If the disease be uncomplicated and there be no untoward events, convalescence is declared during the stage of desquamation. The desquamative process is frequently accompanied with pruritus, which in some cases is excessive. Albuminuria occurs in this stage more frequently than in the stage of eruption—namely, in the proportion of one-fourth. As in the stage of eruption, so in the desquamative stage, the presence of albumen in the urine may or may not be indicative of an important renal complication. A recurrence of the eruption may take place during the stage of desquamation.

The duration of this stage is indefinite. It may end in five or six days. The desquamation is generally completed in from ten to twelve days, but exceptionally it continues for several weeks.

The foregoing sketch of the clinical history of scarlet fever applies to cases which are either mild or not extremely severe, and in which the course of the disease is regular; that is, without anomalous events or complications. Certain cases are characterized by extreme gravity of the symptoms at the outset of the disease. The pulse becomes very frequent, numbering 140, 150, 160, or more per minute. The thermometer shows notable hyperpyrexia. Great restlessness, delirium, coma, and occasionally convulsions, are other symptoms denoting severity of the disease. Death may take place in one or two days or sometimes even within a few hours from the attack. A fatal result may take place before the eruption appears on the skin or any pharyngeal redness is apparent. The disease in such cases is truly malignant. Cases of this description are liable to occur in certain epidemics. The gravity in these cases is not due to any complication, but to an unusual intensity of the essential morbid conditions which constitute the disease. Ataxic or nervous symptoms, in like manner dependent on the intensity of the disease, are liable to be developed at a later period—namely, active delirium, typhomania, carphologia, and subsultus tendinum. These symptoms characterize certain cases of severe scarlet fever.

Convulsions, coma, and delirium may be developed as effects of uræmia. Disease of the kidneys is an important complication in a certain proportion of cases. Generally, but not always, in these cases the urine is highly albuminous. Albuminuria, as already stated, may not indicate any important affection of the kidneys if the quantity of albumen be small; and on the other hand grave disease of the kidneys may exist without albuminuria. The presence of casts with white or red corpuscles in the urine is more significant of renal disease than the presence of albumen. The danger of uræmia is proportionate to the scanty excretion of urine. Clinical facts show that scarlatina involves a special tendency to renal disease, not only as a sequel, but as a concomitant. It is a rational supposition that the lining membrane of the secretory tubes of the kidneys takes on a morbid condition analogous to that of the mucous membrane of the pharynx, constituting a local affection which is serious in proportion as the excretion of urea is interfered with. General dropsy sometimes occurs during the progress of the disease as well as afterward. Symptoms pointing to uræmia, in addition to the events already stated—namely, convulsions, coma, and delirium—are cephalalgia, disturbance of respiration occurring without either bronchitis, pneumonia, œdema of the lungs, or any appreciable pulmonary affection, defective vision, vomiting, and occasionally diarrhœa. These symptoms should direct attention to the urine as regards albuminuria, the presence of casts, and especially the deficiency of urea. Uræmia, taking place either as a concomitant or a sequel of scarlet fever, may occur without albuminuria and without dropsy. Hence the importance of examinations of the urine in all cases, with reference not alone to the presence of albumen, but of casts and the quantity of urea.

Certain cases of severe scarlatina are characterized by a great amount of throat affection. The name *scarlatina anginosa* is applied to these cases. The tonsils are greatly swollen and covered with a muco-purulent, pultaceous deposit. Deglutition is difficult, and liquids taken into the mouth are returned through the nostrils. The inflammation may extend to the posterior and anterior nares, and acrid pus flows from the nose, excoriating the face. The breath emits an intolerable fetor in some cases. Sometimes, but rarely, the inflammation extends into the larynx and bronchial tubes. Œdema of the glottis is an occasional accident in adults. The structures of the eye may become involved, and destruction of both eyes has been known to follow. An extension of the inflammation through the Eustachian tube into the middle ear is liable to occur. I have met with a case in which the tympanum in each ear was perforated and the ossicles were discharged through the external meatus. Permanent deafness, facial paralysis, and in some cases caries of the temporal bone, followed by circumscribed meningitis and abscess of the brain, are effects of an extension of the local affection to the middle ear. The submaxillary and lymphatic glands of the neck become more or less enlarged, and sometimes suppuration takes place, constituting what have been called *scarlatinous buboes*. I have met with a case of extensive sphacelation of the lips and cheeks, the patient being a child well nourished and healthy up to the date of the attack. Gangrene and ulceration within the throat are very rare events. The local events belonging to scarlatina anginosa, inclusive of inflammation and suppuration of the glands of the neck, may occur early in the disease, or, on the other hand, they may be developed during the stage of desquamation, the symptoms having previously denoted a mild form of the disease. A true diphtheritic exudation in the fauces characterizes certain cases.

In cases of so-called *scarlatina anginosa* the cutaneous eruption may be slight and it is sometimes wanting. Albuminuria is likely to exist in these cases. Uræmic phenomena are liable to occur, but, exclusive of these and of laryngitis or œdema of the glottis, the severity of the throat affection may be sufficient to lead to a fatal result.

Hemorrhages in various situations, together with petechiæ and vibices on the skin, characterize certain cases. The name *hemorrhagic scarlatina* has been applied to these cases. They are almost invariably fatal. Hæmaturia may occur in connection with renal disease, and if not accompanied by hemorrhage elsewhere is not necessarily of grave omen.

Complications other than those which have been noticed, and of rare occurrence, are articular rheumatism, usually confined to a few joints, vaginitis, pleuritis or pyothorax, pericarditis, and endocarditis (sometimes of the acute ulcerative form), the two latter affections being often associated with either rheumatism or renal disease.

In irregular cases of scarlatina the disease is often, but by no means always, severe. The only local manifestation of the disease may be a slight soreness of the throat, no cutaneous eruption taking place. In these cases the disease has been distinguished as *scarlatina faucium* and *scarlatina sine eruptione.* Cases of the throat affection more or less severe, without any eruption on the skin, not infrequently occur during the prevalence of epidemics of scarlatina. An attack of this kind appears, in some cases at least, to render the patient thereafter insusceptible to the disease, but it is not uncommon for persons who have once had scarlatina in its regular form to suffer from the throat affection when exposed to the contagium. In cases of *scarlatina sine eruptione* the nature of the disease is not infrequently overlooked. Another irregularity in very mild cases consists in the absence of any affection of the throat, the cutaneous affection being more or less marked. In these cases doubtless the disease sometimes exists without being recognized or it is confounded with roseola. It has been supposed that the disease may exist without either an affection of the throat, a cutaneous efflorescence, or febrile movement. The name *scarlatina latens* has been applied to such cases. The evidence of the existence of the disease in this latent form is the occurrence of characteristic sequels—namely, albuminuria and anasarca—in persons who had been exposed to the contagium without presenting any of the usual phenomena of scarlatina. The local manifestation of the disease in these cases is supposed to be exclusively in the kidneys. Assuming that such cases occur and that this explanation be true, it is more correct to say that the scarlatinous contagium sometimes gives rise to acute Bright's disease without causing scarlet fever. I have referred to cases exemplifying this fact in treating of acute diffuse nephritis.

Scarlatina has various sequels. The one which occurs most frequently has just been alluded to—namely, albuminuria with general dropsy. This follows the disease in a considerable proportion of cases, the proportion being found to vary at different times and places. The time of its occurrence is between ten and twenty days after the date of desquamation. The symptoms are those of acute diffuse nephritis, an affection which has been considered in connection with the diseases of the urinary system.[1] Œdema of the face and lower extremities is first observed, and anasarca frequently follows. Effusion takes place into the serous cavities if the anasarca be considerable. Hydrothorax sometimes exists to such an extent as to seriously embarrass respiration. The urine is scanty; sometimes it has a smoky appearance, and it may be distinctly bloody. It may, however, contain few or no red blood-corpuscles. It is usually loaded with albumen, but cases have been reported by Rayer and others of dropsy after scarlatina without albuminuria. Epithelial, granular, and hyaline casts are generally found in the urinary sediment. Some red, but more white, blood-corpuscles are usually present. Blood-casts are not frequent. There is notable anæmia, the appetite is deficient, the temper is irritable, and muscular debility is more or less marked. Pyrexia, varying in degree in different cases, accompanies these symptoms. Uræmic coma and convulsions, pulmonary œdema, and œdema of the glottis are grave accidents incident to the renal affection. They occur, happily, in but a small proportion of cases, and under judicious management this affection is rarely fatal. Temporary blindness is a rare event, occurring in connection with uræmic convulsions following this disease. Acute nephritis after scarlet fever rarely ends in chronic renal disease. It is more rational to consider the renal affection which follows scarlatina as a special effect of the latter than to attribute its production to cold or other causes operating after the scarlatinous poison has ceased to act.

The opinion was held by Graves and others that the kidneys are oftener affected after mild than after severe cases of scarlatina. This sequel certainly occurs after mild cases, and sometimes when, from the absence of either the throat affection or of the cutaneous eruption, scarlet fever had not been supposed to exist. A probable explanation, however, of its occurrence in a larger proportion of mild cases is afforded by the fact that of severe cases a considerable number end fatally during the progress of the fever. It is to be borne in mind that coma and convulsions may occur as sequels of scarlatina, due to uræmia not accompanied by either albuminuria or dropsy. That the renal affection has no necessary connection with the eruption on the skin is shown by its occurrence in cases in which the scarlatinous rash was wanting.

Pleuritis, pericarditis, and acute articular rheumatism are occasional sequels as well as concomitants of scarlet fever. Chorea is also to be reckoned among the occasional sequels. External otitis is not uncommon. A purulent discharge takes place from the ears, and sometimes there is considerable deafness. If the inflammation be wholly without the tympanum, the deafness will be temporary, and under judicious management the affection may generally be removed; but it is liable to return from time to time for a considerable period after recovery.

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[1] Vide p. 865.

CAUSATION.—The communicability of scarlet fever is established by irrefragable proof, and it is probable that its causation always involves a contagium. The contagium from mild is as efficient as that from severe cases. It is important that this fact be popularly known. Many persons in this country think that the disease in very mild cases differs essentially from that in severe cases, and it is a common impression that the name scarlatina applies only to the former. Physicians should take pains to correct the error of supposing that the names scarlatina and scarlet fever indicate different diseases. The contagium is contained in emanations from the body, probably in both the expired breath and the cutaneous exhalations; hence the atmosphere surrounding patients is infected. It appears to be certain that the disease has been communicated by inoculating with the serum contained in the vesicles which sometimes appear in conjunction with the cutaneous efflorescence, but such inoculation does not materially affect the duration or the severity of the disease. That the disease may be communicated by means of fomites is unquestionable. The contagium may be carried in the clothes of physicians, nurses, and others. The disease is probably often diffused in this way. Facts show that the contagium adheres to material substances and preserves its morbific power for a long period. Persons have been attacked on returning to houses in which cases have occurred weeks or months previously, even when careful efforts have been made to remove the contagium. Several instances have been recorded in which the scarlatinal virus appears to have been transported by milk. Scarlatina sometimes follows with notable frequency surgical operations and the puerperal state. In these cases, however, it is necessary to be somewhat guarded in the diagnosis, as an efflorescence which has nothing to do with scarlatina may follow these conditions and septic fevers in general.

The period of incubation is undoubtedly, as a rule, much shorter in scarlatina than in the other eruptive fevers. Its duration, however, varies in different cases. Trousseau cites a case in which the attack took place within twenty-four hours after exposure. In a case under my observation the patient, an infant of nineteen months, was attacked twenty-fours after the first symptom of illness in a young girl who assisted in taking care of the child. In another case the duration was forty-eight hours. It is probable that the period rarely exceeds six days.

Statistics show that the disease occurs most frequently in the third and fourth years of life—that the liability to it diminishes progressively after the fifth year, and becomes very small after forty. Children less than two years of age and infants at the breast are not, as has been incorrectly stated, insusceptible to it. Several instances of its occurrence at less than two years of age have come under my observation. In one of these a child only three weeks old contracted the disease from her mother, who was attacked a few days after confinement. Cases have been reported in which the disease was contracted *in utero.*

As a rule, this disease is experienced only once; but, making due allowance for cases in which roseola may have been confounded with scarlatina, the exceptions to this rule are not extremely rare. Second or third attacks are rarely fatal.

Nothing is positively known concerning any micro-organisms which can be regarded as the cause of the disease. As in the other eruptive fevers, micrococci have repeatedly been found in various organs of the body in scarlatina, particularly when these are the seat of complications, but it is not believed that these micrococci are concerned in the production of the disease.

DIAGNOSIS.—Attention to the following diagnostic points renders the discrimination of this disease easy in the great majority of cases: The short duration of the stage of invasion, the intensity of the pyrexia, the appearance of the efflorescence in the throat prior to the cutaneous eruption, the appearance of the efflorescence on the body and upper extremities often before or simultaneously with its appearance on the neck and face, the rapid extension of the efflorescence over the cutaneous surface, the scarlet color of the efflorescence, the irregular and serrated margins of patches of efflorescence, and the persistence or increase of the pyrexia after the appearance of the efflorescence on the skin. These are positive diagnostic features belonging to the clinical history of scarlet fever as contrasted with smallpox and measles. On the other hand, the two latter eruptive fevers are excluded by the shorter period of invasion in scarlatina, pain in the loins not marked in this period, absence of the characteristic eruption of variola in the mouth and fauces, the non-occurrence of variolous papules and vesicles with depression in the centre, and of the remission of fever when the eruption appears, absence of coryza and bronchitis, which belong to the history of rubeola, and of certain distinctive characters pertaining to the eruption of rubeola, which will be stated in treating of that disease.

The difficulties in the way of diagnosis relate, in the first place, to extremely mild cases with a slight eruption and without any affection of the throat. In these cases the disease is liable to be confounded with roseola. The differential points will be noticed in treating of the latter in the next chapter. In the second place, the diagnosis may be difficult in cases in which the local manifestation is confined to the throat; that is, cases in which the cutaneous efflorescence is wanting. It must be confessed that the diagnosis in some of these cases must be, in a measure, based on their occurrence during the prevalence of scarlatina, and sometimes on the occurrence of well-marked cases in members of the same family. In some cases of doubt or error the sequels of scarlatina, especially renal disease, serve either to correct or establish the diagnosis.

A highly probable diagnosis before the efflorescence appears on the skin may be based on a sudden attack of vomiting, with high febrile movement and redness of the fauces, in a subject between three and five years of age, especially during the prevalence of scarlatina.

PROGNOSIS.—There is perhaps no disease in the nosology which presents in different cases wider extremes as regards gravity than scarlatina. In malignant cases the disease is rapidly fatal. The proportion of these cases varies much at different times and places. The prevailing type of the disease in some seasons is mild, and in other seasons severe. Whether this variation be due to a difference in the contagium or to adjunctive influences we are unable to say. Aside from the intensity of the disease, the danger depends on complications liable to occur in cases which, as regards the general symptoms, may appear to be mild. The symptoms which denote imminent danger from the intensity of the disease are—excessive frequency of the pulse, hyperpyrexia, jactitation, active delirium, and prostration; the mode of dying in these cases being by asthenia.

The prognosis is unfavorable in cases in which the throat affection is unusually severe. It is extremely unfavorable if diphtheria become developed either during the progress of the disease or as a sequel. If laryngitis ensue a fatal result is to be expected. Great enlargement and suppuration of the glands of the neck irrespective of diphtheritic inflammation of the fauces denote great danger. If gangrene of the throat or mouth occur, recovery is the exception rather than the rule. Cases characterized by hemorrhage from mucous membranes and petechiæ or vibices are generally fatal. Uræmic coma and convulsions involve imminent danger, and uræmia may occur in cases which as regards the general symptoms appear to be mild. These symptoms may be developed and sudden death take place unexpectedly if examinations of the urine have been omitted. Daily examinations of the urine are highly important. The presence of albumen or casts should always excite apprehension, but the immediate danger is proportionate to the deficiency of urea. The patient is by no means safe after albumen has disappeared from the urine if casts continue to be found in the sediment, especially if the quantity of urine be small and the specific gravity low. The prognosis is extremely unfavorable in cases of scarlet fever occurring in the puerperal state. I have known a case in which the patient contracted the disease in the seventh month of pregnancy. She miscarried during the period of incubation; parenchymatous nephritis occurred as a complication and the symptoms denoted great danger. She, however, recovered, and the child was living.

TREATMENT.—Cases of mild scarlatina do not call for active measures of treatment. The often-quoted saying of the English Hippocrates, that scarlatina is only dangerous through the officiousness of the physician, is, however, hardly true even of these cases, for be the disease never so mild there is a liability to grave complications and sequels. On the other hand, in certain cases of severe scarlatina the malignancy of the disease renders any treatment unavailing. With respect to this disease as well as the other eruptive fevers, it is to be premised that there are no known specific remedies by means of which it may be arrested or controlled; but the inference by no means follows that judicious measures of treatment may not do much toward affording relief and diminishing the fatality. It is to be added that a comparison of different collections of cases with reference to the rate of mortality under different methods of treatment is of little value, on account of the wide diversity as regards the severity of the disease.

Certain measures heretofore employed in the treatment of scarlatina are now regarded as opposed by sound reasoning and experience. This statement applies to bloodletting, active purgatives, mercurialization, blisters, and emetics. These are never indicated, and must of necessity prove hurtful. The discovery of a remedy which will arrest the disease by destroying within the body the special cause—in other words, an effectual parasiticide for the specific organisms and germs on which the disease is supposed to depend—belongs to the future. This statement applies to all the eruptive fevers. Carbolic acid, sodium sulphocarbolate, and the sulphites have been tried with an apparently beneficial effect. I have heard testimony from physicians of experience in behalf of the usefulness of each of these remedies. It has not, however, been proved that they have a special curative efficacy. At the present time, therefore, the indications are to be drawn from particular symptoms, events, and complications in individual cases.

In the majority of mild cases hygienic treatment is alone required. In addition to free ventilation, cooling drinks, and light nourishment the hygienic treatment should embrace measures to maintain the functions of the skin. In view of the fact that renal disease as a complication or sequel is a source of danger in these cases, attention to the skin is important, however mild may be the disease. The tepid bath once or twice daily is to be recommended. If the bath be not practicable, the application of a sheet wet with tepid water or repeated sponging of the body may be employed in its stead. There is reason to believe that the more the functions of the skin are maintained by these measures the less is the liability to an affection of the kidneys either during or after the disease. Exposure to cold is to be avoided both while the disease is in progress and in convalescence, but care in this respect should not lead to error in the way of ventilation or of oppressing the patient with an overplus of clothing. Vigilance or restlessness, if not relieved by the use of the bath, the wet sheet, or sponging, may indicate anodyne remedies. Constipation is to be relieved by simple enemas. Other symptoms may indicate appropriate palliative remedies.

In some cases the leading indication is derived from the intensity of the fever as denoted by hyperpyrexia and the frequency of the pulse. To diminish the heat and the frequency of the heart's action is an important object of treatment. This object may be most effectually accomplished by the use of water. It was in this disease that Currie was led, nearly a century ago, to employ cold affusions with a happy effect. This mode of applying water was strongly advocated by Trousseau, who stated that he employed it with highly satisfactory results in a large number of cases in private and hospital practice. It is perhaps the most efficient measure to be employed for the relief of the active delirium which sometimes occurs in this disease. The apparent boldness of the practice renders it frequently objectionable on the score of popular prejudice. Frequent sponging of the body secures to a considerable extent the same results; but the use of the wet sheet is hardly less efficacious than the cold affusion as practised by Currie. Employed as an efficient antipyretic measure, the wet sheet is applied over the whole body without any other covering, and the surface is sprinkled with cool or cold water at short intervals in the manner described in connection with the treatment of typhoid fever and insolation. The temperature is always reduced one, two, or more degrees by this measure, and it may be repeated as often as the thermometer shows a return to great intensity of heat. There is no occasion for fear on the ground of danger of repelling the eruption. The wet pack acts efficiently as a sedative and diaphoretic measure, and it has considerable potency as an antipyretic. The patient, stripped of all clothing, is enveloped in a sheet saturated with water at about the temperature of 70° F. and closely covered with several blankets. Usually after remaining in the pack for about an hour free perspiration is induced. The pack is to be removed when this effect occurs, the body wiped dry, and the patient placed in bed. The frequency of the pulse is often notably lessened by this measure. The patient is tranquillized and obtains refreshing sleep. The measure may be repeated once or twice daily. Its effect is so agreeable that patients often desire a repetition. In this disease, as in other fevers, full doses of quinia sometimes have a notable antipyretic effect. Antipyrine is an efficient and safe antipyretic.

In malignant cases, and whenever the intensity of the disease involves a tendency to death by asthenia, the chief reliance must be on sustaining measures. Alcohol is indicated in proportion to the frequency and feebleness of the pulse, together with general prostration. I have witnessed the same striking benefit from the free use of wine or spirits in certain cases of scarlatina as in cases of typhus or typhoid fever. Alcoholics, however, are not to be given without discrimination, and the physician is to be guided in their use by watching their immediate apparent effects, precisely as in other essential fevers. In children, wine-whey, milk-punch, and egg-nog are eligible forms for the admininistration of stimulants and nourishment.

Cases of so-called scarlatina anginosa in addition to sustaining treatment call for measures addressed to the affection of the throat. The chlorate of potassa is generally considered, and probably with justice, an important remedy in these cases. From one to two drachms may be given daily. The chlorine mixture is highly recommended by Watson and others. Stimulating or caustic applications to the pharynx are of doubtful utility. The difficulty of making them in children, and the perturbation occasioned by making them forcibly in spite of their resistance, are not small objections;

but aside from these objections it may be doubted if they produce a beneficial effect. Antiseptic applications by gargling, if the patient be not too young, by means of a sponge if the resistance be not too violent, or in the form of a powder by insufflation, contribute at least to comfort. The solution of chlorinated soda may be used for this purpose, diluted with eight or ten parts of water. The permanganate of potash may also be used in the proportion of half a grain of the salt to five ounces of water. Other topical remedies are alum, tannic acid, the borate of soda, carbolic acid, and salicylic acid. The free use of iced drinks is useful as regards the condition of the throat. The patient may be allowed to take into the mouth small pieces of ice almost *ad libitum;* and for young children a convenient plan is to confine pieces of ice in a gauze bag which may be held in the mouth. Externally, compresses kept wet with cold water may be applied. It has been recommended to apply ice to the sides of the neck. It is customary to paint the neck with iodine over glandular swellings. If suppurative inflammation ensue, poultices or the water-dressing should be applied.

Extreme pruritus of the skin is in some cases a source of much annoyance, preventing sleep and increasing the constitutional disturbance. With reference especially to this symptom some years ago a plan of treatment proposed by a German physician, Schneemann, consisted in the application of lard to the surface of the body, the rind of bacon being used for this purpose. This plan was adopted to some extent in this country. It relieves, in some cases at least, the pruritus and diminishes the febrile excitement. Other applications, however, less disagreeable, are equally efficacious. Glycerin and the rose-water ointment, glycerin and cologne-water, in the proportion of one part of the former to four or five parts of the latter, or, what is still better, vaseline or lanolin, may be substituted.

Convulsions are generally due to uræmia, but occurring early in young children they may be incident to the development of the disease without denoting a renal complication. Whether uræmic or otherwise, if they continue the inhalation of chloroform is indicated. I have notes of cases in which this measure was employed with immediate relief and recovery followed.

Uræmic phenomena in addition to convulsions—namely, cephalalgia, disturbance of vision, and coma—render prompt measures of treatment vastly important. Saline purgatives or elaterium are to be employed at once to eliminate urea, and diaphoresis should, if possible, be procured by the use of the hot-air bath, the wet sheet, or by the hypodermic administration of the muriate of pilocarpine. Vomiting and purging should always suggest the inquiry whether they be not due to uræmia, and if so they are not to be arrested. Albuminuria, and especially a deficiency of urea in the urine, should lead to measures with a view of forestalling the effects of uræmia. Fomentations are to be applied over the loins, the bowels are to be kept loose with saline laxatives; and with reference to this object it is important to produce diaphoresis by means of the warm bath or the wet sheet.

Restlessness and vigilance, if not relieved by measures of treatment already noticed, may require anodyne remedies. The succedanea of opium are to be preferred—namely, belladonna, hyoscyamus, etc. If, however, these be not efficacious, opiates are to be given, but they should be administered cautiously in young children. With reference to the liability to heart-clot, Richardson advises the carbonate of ammonia given in small doses frequently repeated. Several writers have advocated this remedy as having a special salutary influence in scarlatina.

The treatment of the sequels of scarlatina need not be here considered. The most frequent of the important sequels—namely, acute diffuse nephritis—has been considered in a former part of this work, to which the reader is referred. In the treatment of pleuritis, pericarditis, rheumatism, and chorea are involved essentially the same principles as when these affections occur in other pathological connections. External otitis is often neglected, and it may therefore continue, and in the end lead to impairment of hearing. Daily cleansing of the ear by the injection of tepid water and soap will usually prove sufficient to effect a cure; but if the affection continue in spite of this measure, which from its very simplicity it is often difficult to enforce, mild astringent injections should be employed.

I shall add a remark with reference to the prevention of renal disease as a sequel of scarlatina. The occurrence of this sequel being generally attributed to cold, it is common to confine patients within doors, and sometimes even to the bed, for two or three weeks after convalescence. Regarding, however, the sequel as a remnant of scarlatina, this extreme care is unnecessary. It is sufficient to observe the same precautions with regard to exposure to cold or other morbid agencies as during convalescence from any acute affection. Gestation out of doors has seemed to me to be useful even when patients are suffering from albuminuria and general dropsy following scarlet fever.

It remains to notice the protective influence against scarlatina which has been imputed to belladonna. Satisfactory proof of its prophylactic power requires that the number of failures shall not be large; and, judged by this rule of evidence, it is extremely doubtful if there be any ground for imputing to this remedy a prophylactic power. It is, however, to be considered that to prescribe belladonna as a prophylactic is always a harmless experiment, and it is therefore objectionable only on the score of supererogation. Moreover, as the popular mind has been directed to this question, the wishes of friends are often better satisfied if the drug be prescribed. These considerations may properly influence the physician. The following are the directions for the use of the remedy: "Dissolve from one to three grains of fresh and well-prepared extract of belladonna in an ounce of cinnamon-water, adding a few drops of alcohol to prevent fermentation. Of this solution may be given, two or three times a day, one drop for each year of the child's age, to be so administered for two weeks or longer if the danger should continue."[1] The only reliable way of protection against the disease is to avoid the contagium. To do this effectually, it is necessary not only that persons do not come within an infecting range of patients, but that they avoid those who have been within this range, the houses in which there have been cases, and everything that may prove to be fomites. Cases occurring in hospitals and private houses should be at once isolated, and all articles in the apartment occupied by patients should be properly disinfected. The best means of disinfecting clothing, bed-linen, carpets, tapestries, and stuffs which cannot be destroyed by fire is to subject them for two or three hours to steam at a temperature of 212° F. Removal without the limits of the prevalence of the disease is advisable when it is found that cases are likely to prove malignant or severe. On the other hand, if cases be pretty uniformly mild it is to be considered that the period is favorable for having the disease. Voluntary exposure with the intention that the disease shall be contracted is, however, under no circumstances to be recommended.

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[1] Stillé, *op. cit.*

CHAPTER VIII.

ERUPTIVE FEVERS (CONTINUED).

Rubeola, or Measles: Clinical History; Causation; Diagnosis; Prognosis; Treatment.—Roseola.—Rötheln.—Dengue.

Rubeola, or Measles.

RUBEOLA—so called from the red color of the eruption, called also *morbilli*, and commonly known as the *measles*—is a disease of less importance than the eruptive fevers which have been considered. It is generally a mild and often a trivial disease, but it is desirable to discriminate it as early as possible from other eruptive fevers. It is of frequent occurrence, it is sometimes accompanied by grave complications, it is occasionally followed by serious sequels, and in a certain proportion of cases the disease is intrinsically severe or even malignant. Hence, rubeola is by no means an unimportant disease. By German writers the term rubeola is used to denote an eruptive fever distinct from measles, called also Rötheln, and in this country distinguished as German measles.

Rubeola has no special anatomical characters exclusive of the internal and external efflorescence which is most conveniently considered as belonging among the symptoms of the disease. I shall pass, therefore, at once to the clinical history.

CLINICAL HISTORY.—The career of this disease is subdivided, like that of the other eruptive fevers, into the stages of invasion, eruption, and desquamation, each stage claiming separate consideration.

Stage of Invasion.—The distinctive local symptoms in this stage resemble those of a common cold, or rather an attack of influenza. There is coryza, with frequent sneezing and an acrid muco-serous discharge from the nostrils. The eyes are irritable, reddened, and watery. Epiphora is sometimes marked, the tears excoriating the face, and there is more or less intolerance of light. Subacute laryngitis, denoted by hoarseness, frequently occurs, and the symptoms of bronchitis are generally present, the cough being dry, sonorous, and painful. Occasionally the pharynx is the seat of subacute inflammation. The bronchitis, laryngitis, and coryza are due to the efflorescence which takes place on the mucous membrane of the air-passages prior to its appearance on the skin. With these local affections, which vary much in intensity in different cases, there is associated moderate pyrexia, the axillary temperature rarely exceeding 102° F., accompanied by irregular chilly sensations and shivering, but rarely by a distinct chill; the appetite is impaired or lost, and in some cases nausea and vomiting occur; and pain in the head and limbs, with debility and lassitude, is in general proportionate to the pyrexia. The general are often out of proportion to the local symptoms, as in cases of influenza. They are less intense than in the stage of invasion in variola and scarlatina, and in certain cases they are slight. Constipation exists in some cases, and in other cases there is diarrhœa. Convulsions sometimes occur in this stage, chiefly in children. In general they are not indicative of danger. Epistaxis is in some cases a prominent symptom. Spasm of the glottis, or false croup, is an occasional symptom in young children. An erythematous eruption sometimes precedes the eruption proper to the disease.

The average duration of this stage is three or four days. The duration is therefore twenty-four hours longer than in variola, and forty-eight or more hours longer than in scarlatina. Cases are not very infrequent in which the duration is five, six, or seven days, and even longer, and on the other hand it may be but three or two days, and even a single day.

Stage of Eruption.—A transient convulsion may usher in this stage in children, but it is of much rarer occurrence than in scarlet fever. The efflorescence first appears on the temples and forehead. In a few hours it extends over the head and neck. It is gradually diffused over the body and extremities, its full development occupying between thirty-six and forty-eight hours. In the slowness of the extension of the rash over the cutaneous surface this fever differs from variola and scarlatina. The eruption is sometimes preceded by a day or two by the appearance of patchy redness on the hard and the soft palate. This redness is by many interpreted as the first sign of the exanthem.

The eruption has at first an appearance of minute red dots or specks, which soon enlarge, become slightly elevated, and tend to range themselves in circular or crescentic forms. The papules resemble those in smallpox prior to the appearance of vesicles, but they are larger and softer. They bear a resemblance to flea-bites. The redness momentarily disappears on pressure. Increasing in number and size, they coalesce to a greater or less extent, and form blotches of variable dimensions with curvilinear or semilunar borders, contrasting in the latter respect with the irregular or serrated patches of efflorescence in scarlatina. Aside from cases in which a petechial eruption occurs, the eruption proper to the disease sometimes shows not only hyperæmia, but infiltrated hæmatin, the redness being with difficulty removed by pressure, and a brownish color remaining after the eruption has disappeared. The eruption in some cases is everywhere confluent, being uniformly diffused over the whole or the greater part of the cutaneous surface. The color of the eruption is dull or deep red, offering a contrast to the crimson or scarlet redness of the scarlatinous efflorescence. The portions of skin not occupied by the eruption retain the normal appearance. In proportion to the abundance of the eruption there is swelling, which is most marked on the face. The eruption is sometimes attended with considerable pruritus. Occasionally vesicles are intermingled with the rubeolous papules (morbilli vesiculosi vel miliares). On the fourth day of the eruption it begins to fade successively on the face, trunk, and extremities, and the stage of desquamation commences.

During the stage of the eruption the symptoms denoting coryza, laryngitis, and bronchitis continue. The bronchitis in this stage gives rise to a more or less abundant expectoration consisting of greenish or yellowish sputa, which are frequently nummular. Dry and moist bronchial râles are often heard on auscultating the chest. The irritability of the eyes continues, and not infrequently there is conjunctivitis. Pharyngitis in some cases continues, and sometimes the inflammation extends into the Eustachian tube, occasioning partial deafness. The pyrexia does not disappear or diminish with the development of the eruption. On the first and second day of the eruption the temperature of the axilla may rise to 104° F. and even higher. From this date, in the natural course of the disease, the temperature rapidly diminishes. Other symptoms denoting constitutional disturbance are proportionate to the pyrexia. The latter, save in exceptional cases, is much less in degree than in scarlatina. The tongue is generally coated, and not infrequently the elongated papillæ, projecting through the coating, present the appearance of red points.

Stage of Desquamation.—This stage may be considered as having begun when the eruption begins to fade. The duration of the stage is between four and eight days. In uncomplicated cases pursuing a favorable course this stage is the stage of convalescence. Actual desquamation occurs in only a certain proportion of cases. It may be more or less marked. It is always furfuraceous, the epidermis being exfoliated in branny scales, not in patches or flakes. Coincident with the decline and disappearance of the eruption the pyrexia diminishes and ceases. More or less cough and expectoration are likely to continue after the cutaneous eruption has disappeared, and persisting conjunctivitis is not uncommon. Occasionally the eruption on the skin reappears after having existed for the usual period and having disappeared. Diarrhœa, usually mild, is likely to occur in this stage.

The career of the disease is thus extended between twelve and sixteen days.

The foregoing sketch of the clinical history relates to cases in which the disease pursues a typical course. Anomalous cases occur, the disease in some being very severe or malignant, and in others unusually mild. In exceptional cases the eruption appears first on some other part of the body than the face. The affections of the Schneiderian membrane, larynx, and bronchial tubes are sometimes wanting. Writers have considered these cases as constituting a variety of the disease, called *rubeola sine catarrho.* To a certain extent, doubtless, cases which have been considered as of this variety have been cases of roseola. On the other hand, it is supposed that cases occur in which the local manifestations are limited to the mucous membrane; hence there is another variety, called *rubeola sine eruptione.* It must be difficult to decide positively that rubeola exists when the cutaneous efflorescence is wanting. In the cases belonging to the two varieties just named the disease is unusually mild. A severe form of the disease is characterized by the occurrence of petechiæ and hemorrhage in various situations. The phenomena of purpura are associated with measles in these cases. Of this rare form of the disease the following case is an example: The patient was a man aged about twenty-five years. The rubeolous eruption was abundant, presenting the characteristic appearances, but was somewhat darker than usual. Scattered among the patches of efflorescence over the whole surface of the body were petechiæ varying between the size of a pin's head and that of a split pea. There was hemorrhage from the nose and mouth. Blood also was discharged from the bowels and was contained in the urine. The tongue and gums were pallid except at certain points which were stained with exuding blood. Blood was effused beneath the conjunctiva, infiltrating the whole surface of the eyeball except the cornea. The case terminated fatally. This form of the disease is distinguished as *hemorrhagic rubeola.* It has been called also *rubeola nigra*, or black measles.

Gangrene attacking the mouth, and occasionally the anus, vulva, nose, lungs, and larynx, sometimes occurs in connection with rubeola. Other morbid conditions than those belonging to the latter, it is to be presumed, must concur in giving rise to this event. It has been observed chiefly in young children in eleemosynary institutions. Aside from any such local events or important complications, this disease sometimes assumes a severe form, presenting the ataxic and adynamic symptoms of typhus or typhoid fever, and ending fatally from an intrinsic tendency to death. Such cases, however, are extremely rare.

In the great majority of the cases in which the disease is severe the gravity is dependent on complications. The important complications most likely to occur are seated in the respiratory system. Diphtheritic laryngitis or true croup is sometimes developed. Capillary bronchitis is another fatal complication liable to occur in children; and, occurring in children of less than three years of age, it generally destroys life. It is more liable to occur in hospital than in private practice. Broncho-pneumonia and lobar pneumonia are the most frequent of the serious complications. The latter in young children is likely to prove fatal, and it is by no means devoid of danger in adults. Pleuritis is an occasional complication, but it is far more infrequent than pneumonia. Croup, capillary bronchitis, and especially pneumonia, are not less likely to occur as sequels than as concomitants of rubeola.

A delay in the appearance of the eruption is generally an effect of some important complication. The occurrence of an important complication may lead to a notable diminution and sometimes to a retrocession of the eruption. It is a popular error to attribute the complications to the retrocession of the eruption; the latter is a consequence, not a cause, of the former. Irregularity as regards the situation in which the eruption first appears is sometimes observed. In rare instances the efflorescence first makes its appearance on the lower extremities, and extends thence over the body, upper extremities, and head.

After recovery from rubeola, pulmonary phthisis, acute tuberculosis, and tuberculous meningitis are observed in a proportion of cases not large, but sufficient to show that the system is left in a condition favorable for the development of these affections. Affections distinguished as scrofulous are liable to follow as sequels—namely, scrofulous ophthalmia, coryza, otorrhœa, enlargement of lymphatic glands, etc. After an extensive prevalence of measles in any place it is observed for some years that the mortality among children exceeds the average mortality at other times. The practical importance of these facts is obvious.

Nephritis is a rare complication of measles. Some albuminuria, at the height of the disease, however, is not rare.

CAUSATION.—Rubeola, like scarlatina and variola, is a contagious disease. The contagium is not only received by those brought into close proximity to persons affected with the disease, but it may be transported to a distance by means of fomites. Persons contract the disease from the contagium adherent to the clothes of those who may have recently visited rubeolous patients. Physicians may in this way diffuse the disease.

The disease may be communicated by inoculation, either with blood from an exanthematous patch or with the secretion from the eyes and nostrils. The activity of the rubeolous contagium, however, is much less persistent than that of the contagium of smallpox and scarlet fever. The communicability, not alone by inoculation, but by means of an infected atmosphere or by fomites, belongs to the stage of invasion as well as to the eruptive and desquamative stages.

The duration of the period of incubation varies within widely separated limits—namely, between one day and thirty days. In the majority of cases the attack occurs between six and ten days after exposure. The duration was within these limits in a dozen cases under my observation in which the time of exposure could be definitely fixed, with a single exception in which the duration was fourteen days. The average duration of the period of incubation may be given as ten days. When produced by inoculation the period of incubation is somewhat shorter than it is usually when the disease occurs naturally.

The susceptibility to the disease exists at all ages. Cases are much more frequent in children than in adults, but this is owing to the fact that the majority of persons have the disease in childhood, and are generally thereby made insusceptible. They who escape the disease in early life on account of not being exposed to the contagium are liable to contract it at any subsequent age. The susceptibility is very slight during the first six months of infantile life. As a rule, this disease, like scarlatina and variola, renders the system ever afterward insusceptible to it, but exceptions to this rule are not very rare. Well-authenticated cases in which the disease has occurred three and even four times have been reported. Prevailing in camps, schools, hospitals, and communities as an epidemic, this disease presents at different times a great diversity as regards mildness or severity and the tendency to particular complications; in other words, the character of different epidemics of rubeola, as of other epidemics, is by no means uniform, but varies much in different seasons and places.

DIAGNOSIS.—The diagnosis of rubeola after the appearance of the efflorescence on the skin is generally unattended with difficulty. The diagnostic characters which have been embraced in the clinical history are sufficiently distinctive. The more important of these are—the long duration of the stage of invasion; the affection of the air-passages; the appearance of the eruption almost invariably first on the face, and its gradual diffusion over the body; the color of the eruption, its papular character, the softness of the papules, and the tendency of the papules to assume a crescentic arrangement. So far as the eruption is concerned, early in the eruptive stage there is some resemblance to the papules of smallpox, but the differential points which have been presented in treating of the latter disease should enable the practitioner to make the discrimination. In a case of rubeola in which the cutaneous efflorescence is not abundant and the air-passages are unaffected the disease is liable to be confounded with roseola; but the eruption in these two affections differs, and, moreover, such cases are extremely rare. Cases in which the air-passages are alone affected—that is, in which there is no cutaneous efflorescence—hardly admit of a positive diagnosis, but such cases are extremely rare. Their occurrence during the prevalence of rubeola in persons known to have been exposed is the chief ground on which the diagnosis is to be based.

The diagnosis cannot be made with positiveness prior to the appearance of the efflorescence on the skin. The occurrence, however, of coryza, irritability of the eyes, and bronchitis, with pyrexia, in persons who have never had measles at a time when the disease is prevailing, renders a diagnosis during the period of invasion highly probable. The diagnosis is sometimes delayed by the early occurrence of complications which interfere with the regular course of the disease, especially as regards the eruption. The appearance of the latter, however, sooner or later in the great majority of cases, removes the difficulty which may previously have existed.

Of this fever, as of all the eruptive fevers, the diagnosis in the negro must, of course, be based on other points than those relating to the color of the eruption. "In the pure negro the eruption appears as yellowish spots slightly elevated and giving a sensation of roughness; in the mulatto, as a dusky-brown, ill-defined; and in the lighter shades more distinct, reddish-brown, spots, approaching the characteristics of the eruption in the white."

PROGNOSIS.—In the majority of cases, as this disease is ordinarily presented in civil practice, it is either mild or unattended with danger; but in a certain proportion of cases the disease is severe and dangerous. The severity and danger are due to the anomalous events and complications which have been noticed in connection with the clinical history. The number of cases rendered fatal by the coexistence of capillary bronchitis, diphtheritic laryngitis, pneumonitis, etc. is not inconsiderable. The rate of mortality differs greatly at different times and places, so that the statistics of any particular epidemic would not furnish results applicable generally to the disease. It may prove a serious disease in camps. Woodward states that of the 38,021 cases contained in the official returns from the armies of the United States in the first two years of the late civil war, there were 1864 deaths. Bartholow is of the opinion that these figures do not express the actual fatality due directly or indirectly—that is, by complications and sequels—to this disease during the war; and he estimates the ratio of deaths to cases as 1 to 5.[1]

During the war the prevalence of the disease was great among recruits, especially those from the country, and the sequels or the effect upon the general health incapacitated a considerable proportion for remaining in the service. Taking into view the mortality and the enfeebled condition remaining for a long time in many of the cases which did not end fatally, measles proved to be one of the most formidable of camp diseases. With reference to the efficiency of troops in active service, it is desirable to isolate cases at once wherever they occur in camp. Doubtless, the rate of mortality and the disqualifying effects of the disease were often due to the coexistence either of scorbutus or malarial poisoning.

TREATMENT.—In ordinary cases the disease calls for nothing but palliative measures and attention to hygiene. The cough may be palliated by anodynes, and these may also be indicated by restlessness or vigilance; and for the pyrexia, if it be considerable, gentle diaphoretic and refrigerant remedies may be prescribed. The object of treatment in these cases is simply to render the patient as comfortable as possible, the disease pursuing a favorable course without therapeutical interference. It is a widely-diffused popular notion that saffron (*Crocus sativus*) is a valuable remedy in measles, as well as in the other eruptive fevers, given for the purpose of promoting the eruption. There is little ground for this notion, but the remedy is harmless. Cathartics in this as in the other eruptive fevers are not indicated, and are likely to do harm. Emetics are rarely if ever indicated. The patient should be confined to a room in which the temperature is agreeable, but free ventilation should be secured. The air of the room in the treatment of cases of this fever, as of the other essential fevers, and, indeed, of all acute diseases, should be effectually changed after short intervals by opening the windows, protecting the patient against draughts by covering the face as well as the body. The body may be sponged, portions at a time, with tepid or cool water. This may be done without any fear of causing the eruption to disappear prematurely. If the degree of pyrexia call for antipyretic treatment, the wet sheet may be resorted to. Refrigeration will not render the patient more liable to pneumonia or other pulmonary complications. Quinia, salicin, and antipyrine may also be given for an antipyretic effect. As a protection for the eyes the room should be moderately darkened.

In cases in which the disease is rendered severe by complications the treatment will have reference to the latter. The general principles of treatment in these cases are the same as when the complicating affections occur in other pathological connections. It would, therefore, be a repetition to consider the indications derived from the different complications. A practical question relates to the retardation, diminution, or retrocession of the eruption in some complicated cases. Is it important to resort to active measures designed to promote the appearance of the eruption? The importance of this object of treatment has doubtless been much exaggerated. Certain measures employed for this object, such as emetics, active stimulants, the hot or vapor bath, and overloading the body with clothing, are not to be employed. This general rule may be adopted—namely, measures should not be employed for this object if, aside from the object, they are likely to be hurtful. The tepid bath.

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[1] *Sanitary Memoirs of the War*, New York, 1867–69.

stimulating pediluvia, and sinapisms or other rubefacient applications to the skin, are not excluded by this rule. It is to be considered that the complications do not occur in consequence of the eruption being delayed, or insufficient, or "striking in;" and it is more probable that the latter are effects of the former.

Cases presenting ataxic or adynamic symptoms, either with or without complications, claim the tranquillizing and supporting measures indicated in other diseases with the same symptoms.

It is important to bear in mind the liability to scrofulous and tuberculous affections after recovery from this disease. Patients should be placed under hygienic influences which will tend to invigorate the system, with a view to obviate a tendency to the development of these affections.

Protection against this disease and the prevention of its diffusion can be secured only by means of the isolation of cases and measures of disinfection. Children should be kept from school and other places in which they are brought into contact with other children whenever, from the symptoms of the stage of invasion, the nature of the disease is to be suspected. There is ground for the opinion that the communicability is greater during this stage. The clothes worn by patients, the bedding, and the apartments should be disinfected. These protective and preventive measures are especially important during the prevalence of an epidemic in which there is a tendency to grave complications. They are of less importance in mild epidemics, inasmuch as the subsequent insusceptibility which, as a rule, the disease confers, is obviously desirable.

Roseola.

The eruptive fever called *roseola* or *rose rash*, and sometimes called *false measles*, is an affection of very little importance, exclusive of its liability to be mistaken for rubeola or scarlatina.

The eruption is preceded by symptoms of constitutional disturbance for one or two days. These symptoms are—cephalalgia, loss of appetite, sometimes nausea and vomiting, and occasionally diarrhœa, chilly sensations, febrile movement, and general malaise. In young children convulsions may occur. The constitutional disturbance is more or less marked, but it frequently is slight. The affection of the throat and of the air-passages which belongs to the history of scarlatina and rubeola in the stage of invasion is slight or wanting in roseola.

The eruption appears in the form of rose-colored spots or patches which are not elevated, the redness disappearing momentarily on pressure. It appears on different parts of the body, not beginning on the head and extending over the body, as in rubeola. Frequently it appears on the body and extremities and not on the face. It continues for twenty-four or forty-eight hours only, in a majority of cases. It disappears without desquamation and is liable to return. It is sometimes accompanied with considerable pruritus. The affection has neither sequels nor complications. A roseolar eruption may, however, occur in the course of other affections, especially in the stage of invasion in varioloid, in articular rheumatism, and in epidemic cholera after reaction. Roseola is not a grave disease. One attack affords no protection against recurrences. It is oftener presented in females than in males. It prevails not infrequently as an epidemic, chiefly during the summer season. Some authors consider it to be contagious.

Attention to the foregoing points will suffice for the discrimination of the affection from scarlatina and from rubeola. It does not call for treatment.

The use of the balsam of copaiba by some persons induces an efflorescence analogous to that of roseola, and more rarely the use of cubebs, the iodide of potassium, turpentine, antipyrine, and the sulphate of quinia. A variety of the affection belongs to the history of syphilis.

Rötheln.

The affection called by German writers *rötheln* and rubeola (*German measles*), and called also rubella, is probably not a new disease in this country, but it was formerly confounded with measles, with roseola, or with scarlet fever. Prof. J. Lewis Smith studied an epidemic occurring in New York in 1874, and found the characters which distinguish this affection. It occurs as an epidemic and is probably contagious. It affects all ages, but especially children. It differs from measles in having a period of invasion rarely longer than a day; in a much less degree of pyrexia; in the more frequent appearance of the eruption first on the body and neck; and in the rapid diffusion of the eruption. The eruption, moreover, is less elevated; the spots are round, or at least usually less irregular than the papules of measles, and there is no tendency to a crescentic arrangement. There is slight coryza, but the larynx and bronchial tubes are rarely much affected. In some epidemics the cervical and to a less extent other lymphatic glands are swollen.

The color of the eruption sometimes resembles that of scarlet fever rather than that of measles. The affection bears a close resemblance to extremely mild cases of these diseases. In making the discrimination reliance is to be placed on the occurrence of other cases having the diagnostic characters of rötheln and the non-prevalence of scarlet fever or of measles. It is hardly possible to diagnosticate an isolated sporadic case of rötheln, but when a large number of cases with similar characters occurs epidemically, the diagnosis can generally be made.

It is probable that cases of rötheln and of roseola have been not infrequently supposed to be cases of measles and scarlatina, and in this way is perhaps to be explained some of the reported instances of the latter diseases occurring in the same person more than once.

That this disease is essentially distinct from measles and scarlet fever is proven by its epidemic occurrence, by its affording no protection against these diseases, and by the fact that the latter afford no protection against it. These are sufficient grounds for recognizing a separate special cause. Rötheln, like roseola, is devoid of danger, and rarely calls for more than hygienic treatment.[1]

Dengue.

Dickson's name is especially identified with the epidemic affection commonly called *dengue*, an unclassical name of uncertain derivation, supposed to be synonymous with "dandy fever." He was the historian of its visitation in this country in 1828, and he contributed more largely to what is known respecting it than any other American writer.

The affection prevailed extensively in the West India Islands in 1827 and 1828, and about the same time in many parts of the Southern States of this country. An affection supposed to be the same prevailed in Philadelphia in 1780, and was described by Rush. It was then, as since, frequently known as the *breakbone fever*. Other epidemics supposed to be identical have occurred at various periods in Egypt, India, and other parts of the world.

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[1] A valuable article on this disease by Dr. W. A. Edwards was published in the *Am. Journ. of the Med. Sciences*, October, 1884.

It prevails especially in tropical and subtropical countries. An extensive epidemic occurred along the southern coast of this country in 1880.[1]

The development of the affection is either abrupt or slow, more frequently the former. The symptoms attending its development are anorexia, chilly sensations, but rarely a pronounced chill, languor, lassitude, and general malaise. These symptoms exist in some cases for only twenty-four hours, but in other cases for several days before the affection is fully developed.

After the access or forming period follows a febrile stage or a paroxysm of fever. The duration of the pyrexia varies between nine hours and three or four days, the average duration being about thirty-six hours. During this stage, and sometimes during the access, acute, often excruciating, pains in the head, eyes, muscles of the neck, loins, and extremities are prominent traits of the affection; hence the name *breakbone fever.* The joints are frequently somewhat swollen, those of the hands and feet being usually first affected. The affection may then have much resemblance to acute articular rheumatism. The pains diminish or disappear with the cessation of the fever, and the patient, who had taken to the bed at the onset of fever, is now able to sit up, and complains only of debility; perhaps he returns to his accustomed vocation. In four or five days, however, the pain often returns, with frequently a recurrence of the pyrexia, debility, and malaise, compelling a return to the bed. During the febrile stage there are irregular remissions. The pyrexia is sometimes considerable, the axillary temperature rising to 105° F. and even higher.

In the majority of cases an eruption occurs at a variable period after the febrile paroxysm. The eruption presents in different cases a diversity of characters. It resembles in some cases very closely the efflorescence of scarlatina. In other cases it is not unlike the eruption of rubeola. It is sometimes papular, like lichen or urticaria, and it is sometimes vesicular, like sudamina or varicella. Erysipelas and purpura are occasionally observed. Hemorrhage from the nose, mouth, bowels, and uterus occurs in some cases. Coryza and pharyngitis are occasional complications, and the latter is sometimes associated with enlarged cervical glands.

The convalescence is often tedious, and the recovery of strength, appetite, etc. is likely to be slow. The average duration of the disease is about eight days. Relapses are not infrequent. As regards incidental events and sequels, convulsions in children occasionally usher in the attack; delirium like that of delirium tremens has been observed to succeed protracted vigilance, and in pregnant women miscarriage is liable to take place. A rheumatic condition of the joints, abscesses, boils, and carbuncles are not infrequent sequels.

The extent of prevalence of this epidemic is remarkable. Wragg computes the number of cases at one time in Charleston at 10,000, and during the epidemic seven- or eight-tenths of the population were affected. All classes are attacked, persons of either sex, children and octogenarians.

The duration of epidemics is brief, ceasing usually in six or eight weeks. Of the special cause nothing is known. J. W. McLaughlin of Austin, Texas, has cultivated from the blood of dengue patients a micrococcus which he regards as the special agent of infection.[2] Sufficient evidence in support of this view, however, has not been adduced. Dickson regarded the affection as contagious. This opinion is opposed by the rapid and almost simultaneous diffusion of the affection, by the limitation of its prevalence to towns or within a circumscribed area, and by the short duration of epidemics. He also was of the opinion that, as a rule, immunity from subsequent attacks is secured when the disease has been once experienced. In proof of this opinion he stated that at Charleston, where the disease prevailed in 1850, only those escaped who had experienced it during the epidemic of 1828. The occurrence of a few cases of the disease among those who had it in 1828 he accounts for by supposing that after an interval of twenty-two years the susceptibility was renewed.[1] It is an affection chiefly occurring in warm climates, and it prevails especially in cities and large towns. To the latter rule there are, however, striking exceptions.

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[1] J. Forrest, *Am. Journ. of the Med. Sciences,* April, 1881.

[2] "Researches into the Etiology of Dengue," *Journ. of the Am. Med. Association,* June 19, 1886.

This affection, although extremely distressing, and presenting not infrequently severe symptoms, is very rarely, if ever, fatal. Its apparent intensity is in striking contrast to the absence of danger.

The affection is self-limited, and there are no known means by which it can be arrested or abridged. The treatment, therefore, consists of palliative measures. Opiates to relieve the pains, restlessness, and vigilance are indicated. Rubefacients to the spine and in other situations are useful. Alcoholic stimulants, given pretty freely, have been found beneficial. A high temperature is an indication for antipyretic treatment. Charles recommends moderate doses of belladonna. During convalescence tonic remedies and hygienic measures to promote appetite and the recovery of strength are indicated.

CHAPTER IX.

DIPHTHERIA.—MILK SICKNESS.

Diphtheria: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Milk Sickness.

Diphtheria.

OF the general diseases which remain to be considered, one of the most important is the affection now commonly known as *diphtheria*. This affection has prevailed at times as an epidemic in various parts of the world from a remote period. It has been described by writers in different countries and epochs under a variety of names, such as *ulcus Egyptiacum vel Syriacum*, *cynanche maligna*, *cynanche contagiosa*, *angina maligna*, *angina gangrænosa*, *morbus suffocans vel strangulatorius*, *garotillo*, *malignant sore throat*, *epidemic croup*, etc. In this country, until within the last thirty years, it has occurred but rarely and to a limited extent during the present century. It was admirably described by Bard in 1789 under the name *angina suffocativa*. The distinctive characters of the affection were very clearly elucidated by Bretonneau in 1821, 1825, and 1826, who applied to it the term *diphthérite*, whence originated the name *diphtheria* proposed by Trousseau. This name, the significance of which relates to the most characteristic local event—namely, the formation of a false membrane—has, to say the least, the negative merit of not involving any hypothesis concerning the pathology of the affection.

Anatomical Characters.—The characteristic lesion of diphtheria is a pseudo-membranous inflammation of one or more of the mucous membranes of the body, and less frequently of parts of the skin deprived of epidermis.

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[1] *Elements of Medicine*, 2d ed., p. 748.

The tonsils, the pillars of the fauces, the soft palate, and the pharynx are the parts primarily affected in the vast majority of cases of diphtheria, and the local affection may be limited to these situations. Often, however, the process extends to the mucous membrane of surrounding parts. The larynx is often involved, and thence the fibrinous exudation may extend to the trachea and to the large and medium-sized bronchi. The pseudo-membranous inflammation not infrequently invades the nasal cavity. The Eustachian tube is sometimes, but rarely involved, and even the mucous membrane lining the cavity of the middle ear exceptionally is covered by false membrane. The mucous membrane of the cheeks, gums, and base of the tongue may present patches of false membrane. The diphtheritic inflammation may extend a variable distance down the œsophagus. Diphtheritic conjunctivitis occurs in some cases, and involves considerable danger of loss or impairment of vision, chiefly from destructive ulceration and from opacity of the cornea.

The parts communicating with the pharynx are not the only parts which may be involved in cases of diphtheria. The lips, the stomach, the anus, the vulva, the vagina, and the prepuce are sometimes affected. The mucous membrane of the puerperal uterus is sometimes attacked. The affection also appears in some cases on excoriated or ulcerated parts of the skin, as on vesicated surfaces, wounds, ulcers, and leech-bites—in short, wherever the skin is deprived of epidermis. Exceptionally it may be seated in some one or more of the parts which have been named, exclusive of the fauces, the latter remaining unaffected. Parts other than those primarily affected are liable to be invaded at any time during the career of the disease.

In the majority of cases of diphtheria the false membrane appears first on the tonsils or the soft palate. The pseudo-membranous exudation is often, although not necessarily, preceded by a simple (catarrhal) inflammation. The false membrane is observed first as a grayish-white, slightly elevated patch, which cannot be detached from the mucous membrane without leaving a bleeding surface. The white patches may remain distinct, or they may coalesce and form one membrane, which covers the greater part of the tonsils, the soft palate, and the pharynx. As the pellicular deposit increases in thickness it acquires a yellowish or dirty-gray color. Sometimes it has a reddish or a brown hue from admixture with blood. The membranous exudation is sometimes soft and pulpy in consistence; but often it is firm, almost leather-like, and elastic, and can be detached in a single layer. At autopsies the membrane is generally found to be softened. In the course of three or four days, sometimes a longer and sometimes a shorter period, the false membrane becomes loosened from its attachment to the mucous membrane, so that it can be readily separated without injury to the subjacent tissue. The separation of the membranous exudation, either as a coherent layer or as a soft, disintegrated mass, leaves the subjacent mucous membrane either smooth and intact or ulcerated. The mucous membrane is left in an ulcerated condition chiefly in those severe cases in which the fibrinous exudation extends into its substance. Ulcers may be produced also by a suppurative process in the mucous membrane.

Outside of the pseudo-membranous exudations the mucous membrane is reddened. It usually presents ecchymoses in greater or less number. It is swollen from œdematous infiltration and the presence of inflammatory products. The tonsils especially are often greatly enlarged. The uvula may be swollen to twice its normal size. The inflamed mucous membrane is usually coated with a muco-purulent secretion which may be present upon the false membranes.

The extension of the membranous exudation into the larynx is not uncommon, and this is a serious event in the clinical history of the disease. The characters of the fibrinous exudation in the larynx, the trachea, and the bronchi are the same in diphtheria as in membranous croup. (Vide p. 287.) The false membrane in the trachea and bronchi is of the typical croupous variety; that is, it lies loosely upon the mucous membrane. In most parts of the larynx it is also loosely attached, but on the epiglottis and the vocal cords it is usually adherent. As a rule, the pseudo-membrane is readily separated from mucous membranes covered with cylindrical epithelium and having a well-marked basement membrane, whereas it is more adherent to mucous membranes lined with flat epithelium.

The false membranes are composed of fibrillated fibrin, epithelial cells, leucocytes, red blood-corpuscles, and low vegetable organisms. The fibrillated fibrin is derived from the fibrinogen contained in the inflammatory exudation from the blood, the fibrin ferment being furnished by necrotic leucocytes and tissue-cells. The epithelial cells in part undergo coagulation necrosis, and thereby lose their nuclei and undergo various changes in shape. These necrotic epithelial cells may form a coarse, glistening, homogeneous network, allied both in appearance and in composition to fibrin. The leucocytes may likewise undergo coagulation necrosis and assume an irregular shape and a hyaline appearance. The structure of the false membrane varies considerably according to its situation. Upon mucous membranes covered with cylindrical epithelium, as in the trachea, the bronchi, and the greater part of the larynx, the pseudo-membrane is composed chiefly of fibrillated fibrin, containing some epithelial cells and a larger or smaller number of red and white blood-corpuscles. Upon mucous membranes covered with laminated flat epithelium, as upon the tonsils, palate, and pharynx, a considerable part of the false membrane is made up usually of epithelial cells and of leucocytes which have been transformed in the manner already described by the process of coagulation necrosis; but here there is also some fibrillated fibrin, as well as nucleated epithelial cells and leucocytes. The false membrane may be composed, in addition to the constituents mentioned, of the superficial layers of the mucous membrane which have undergone coagulation necrosis.

Beneath the false membrane the mucous membrane contains inflammatory products—namely, serum, emigrated white blood-corpuscles, escaped red blood-corpuscles, and frequently fibrillated fibrin. The walls of the blood-vessels and other parts of the mucous membrane frequently present a homogeneous, hyaline appearance, variously described as hyaline degeneration, fibrinoid degeneration, and coagulation necrosis.

In this connection it is well to refer once more to distinctions which have already been described in previous articles. The terms croupous exudation or inflammation and diphtheritic exudation or inflammation are used in a purely anatomical sense, without any necessary relation to the diseases known as croup and diphtheria.[1] A croupous exudation is a fibrinous false membrane which rests loosely upon the mucous membrane without extending into its substance. The primary necrosis which antedates all fibrinous inflammations of mucous membranes involves only the epithelial covering. A diphtheritic exudation is a fibrinous false membrane which at first is closely adherent to the mucous membrane, and which is composed wholly or in part of necrotic portions of the mucous membrane. Here the necrosis involves not only the epithelium, but also the superficial layers of the mucous membrane. Many of the false membranes on the tonsils and pharynx are closely adherent to the mucous membrane, and thus far they appear like diphtheritic exudations; but upon microscopical examination it is often found that only the epithelium, and not the mucous membrane proper, enters into the formation of the false membrane. Such apparently diphtheritic exudations are designated by Weigert as pseudo-diphtheritic. It is doubtful whether anything be gained by the distinctions which are thus made between croupous, diphtheritic, and pseudo-diphtheritic exudations, especially as they do not refer to any essential differences in the nature of the morbid process, but only to differences in degree. It is necessary, however, that the reader should be familiar with these distinctions, as they are in common use. Some writers call all fibrinous inflammations of mucous membranes croupous inflammations, without distinguishing different varieties. Such a plan avoids the manifest confusion attending the foregoing classification.

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[1] Thus we speak of diphtheritic inflammations of the intestine or of the bladder when there is no thought of the existence of the disease known as diphtheria.

As would naturally be expected, the false membranes, being exposed more or less directly to the atmosphere, contain a variety of micro-organisms. Inasmuch as diphtheria is an eminently contagious disease, there is reason to believe that it is caused by some form of micro-organism. The various attempts which have been made to discover a specific organism which can be regarded as the cause of the disease have not yet proved successful. According to Löffler,[1] whose studies in this direction are probably the most exact yet made, of the different bacteria present in the false membranes of diphtheria a chain micrococcus and a short bacillus are the two most commonly found. Of these, the micrococcus is the less constant and the less pathogenic. Löffler attaches more importance to the short bacillus which he has isolated in pure culture, but even this is not found in every case, and moreover it was discovered once in the mouth of a child not affected with diphtheria; so that, as the matter now stands, neither Löffler's bacillus (probably identical with one previously described by Klebs) nor any other form of micro-organism has been proven to be the cause of diphtheria.

In certain cases of great severity portions of the mucous membrane of the mouth and pharynx are separated in the form of sloughs which undergo decomposition and emit a foul odor. The necrotic process may extend into the deeper tissues. This malignant form of the disease is called *gangrenous* diphtheria. Sometimes thick pseudo-membranous deposits are so disintegrated and decomposed as to give a false appearance of gangrenous diphtheria.

The lymphatic glands of the neck, especially those near the angle of the lower jaw, are more or less enlarged. As a rule, the amount of enlargement corresponds to the severity of the affection within the throat. The glandular affection rarely proceeds to suppuration. A diffuse inflammation of all of the tissues of the neck may occur, but this is not common.

The lungs may present a variety of appearances, none of which are characteristic of the disease. When death has resulted from suffocation in consequence of involvement of the air-passages, the anterior and upper parts of the lungs are found to be emphysematous. Bronchitis and broncho-pneumonia are not infrequent complications of diphtheria. Lobar pneumonia is a rare complication. The extension of the fibrinous exudation to the bronchi has already been mentioned. Œdema glottidis may develop in the course of diphtheria.

When the disease has been attended with symptoms of severe blood-poisoning, ecchymoses in the serous membranes and parenchymatous degeneration of the heart, liver, kidneys, and muscles have been found after death. Acute fatty degeneration of the heart and acute myocarditis have been found in certain cases which terminated suddenly with signs of paralysis of the heart.

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[1] *Mittheilungen aus dem Kaiserlichen Gesundheitsamte*, Bd. 2, p. 421.

The kidneys may be the seat not only of parenchymatous degeneration, but also of an acute nephritis. Oertel has found colonies of micrococci in the kidney, but they are accidental and not often present. Acute ulcerative endocarditis is a rare complication of diphtheria. The spleen is not infrequently somewhat enlarged and softened. These changes in the internal organs are those which characterize acute infectious diseases in general.

CLINICAL HISTORY.—Diphtheria presents, in different cases, such differences as regards general and local symptoms—the latter incident especially to the localizations of the diphtheritic affection—that some writers have instituted several varieties of the disease. Without adopting formally any of these, it will suffice to indicate the important diversities in connection with the symptomatology.

A marked point of difference relates to the development of the disease. The attack is sometimes abrupt, beginning with a chill more or less pronounced, and followed by considerable or marked pyrexia, or the disease may begin with symptoms denoting great prostration; but not infrequently the development is gradual and insidious, the patient complaining of indefinite ailments, and the characteristic affection of the throat being discovered on inspection when there are few or no local symptoms pointing to the existence of the disease. After the development of the disease the symptoms are naturally divided into general and local, the latter being referable especially to the parts affected with the diphtheritic process.

The affection of the fauces is rarely accompanied by notable pain or soreness, and hence there is a liability to overlook its existence. The sensibility of the parts appears, in some cases, to be diminished. Deglutition is sometimes painful, but in other cases it is unattended with pain. Incomplete paralysis of the muscles concerned in deglutition, which will be noticed as a sequel, is sometimes a concomitant of the disease, giving rise to more or less difficulty of swallowing, especially liquids. These may regurgitate through the nose. If the exudation be abundant and loose, pharyngeal râles occur with respiration, especially during sleep. The breath in some cases is notably fetid, suggesting the idea of gangrene of the throat or lungs—events not frequent in the history of the disease. The extension of the false membrane over more or less of the buccal mucous surface occasions greater or less pain in connection with the introduction of food or drinks into the mouth, together with ptyalism and stiffness of the parts, involving difficulty of articulation and absence of taste. The affection of the anterior nares is preceded and accompanied by a discharge which produces excoriation and swelling of the upper lip, limited to one side of the lip if one nostril only be affected. In other situations accessible to view, on a mucous surface or on the skin, the diphtheritic process gives rise to more or less pain or soreness, and is attended by a sero-purulent discharge variable in quantity, frequently ichorous, and sometimes fetid. As a rule, the gravity of the disease is commensurate with the extent of the local affection and the abundance of the exudation. It does not follow from this fact that the gravity depends on the local affection. The latter may be a criterion, not the cause, of the former.

The symptoms denoting an invasion of the larynx are essentially those which belong to the history of fibrinous laryngitis or true croup. These have been already considered.[1] Cough, having more or less of the characters known as croupous, feebleness of the voice with elevation of pitch, or aphonia, and labored, stridulous respiration, point to this most serious localization of the affection. The embarrassment of respiration may be increased paroxysmally, as in simple laryngitis or in true croup, by the addition of spasm of the muscles of the glottis. Swelling of the tonsils and pharynx may be sufficient to occasion some obstruction to respiration without invasion of the larynx. It is to be borne in mind that laryngitis may be excluded so long as the voice remains unaffected. Diminished respiratory murmur and embarrassment of the respiratory function out of proportion to the amount of laryngeal obstruction, with the presence of moist bronchial râles, denote an extension of the affection to the bronchial tubes.

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[1] Vide p. 288.

Passing to symptoms other than those directly referable to the parts affected with the diphtheritic process, cases differ widely as respects the amount of constitutional disturbance. Irrespective of an affection of the larynx, which of necessity involves great danger, cases are either mild or more or less severe. In some mild cases the disease is almost trivial, patients not taking to the bed, and perhaps not keeping within-doors; but, however mild cases may be at the outset and during more or less of the career of the disease, there is always a liability to the occurrence of diphtheritic inflammation in new situations, to the development of grave symptoms, and to important sequels. In the cases in which the disease is mild throughout its career the local affection is confined to the fauces, or, exceptionally, to a limited space in some other situation.

In cases of greater or less severity the clinical history embraces symptoms referable to the different anatomical systems—namely, the circulatory, cutaneous, respiratory, digestive, nervous, and urinary.

The pulse in some cases becomes very frequent; in other cases the acceleration is moderate, and it is sometimes slight. In the course of the disease the pulse is sometimes at first more or less rapid, afterward suddenly falling below the average of health. Aside from frequency, the pulse is soft or compressible. It is sometimes irregular—a symptom of unfavorable omen if it be independent of pre-existing organic disease of the heart. Hemorrhage from the nostrils is not an infrequent symptom, and it occurs occasionally from the throat and mouth. Epistaxis is sometimes so profuse as to require mechanical means for its arrest. Under these circumstances it denotes great gravity and danger, and hemorrhage in other situations is to be regarded as ominous.

The skin rarely presents much increase of heat. In some cases there is no increase, and not infrequently in the course of the disease the surface becomes cool or cold. The pyrexia, as a rule, is less than in most other acute diseases. The temperature may, however, be high. Petechial spots or ecchymoses are sometimes observed, occurring generally in connection with hemorrhage from mucous surfaces. In grave cases the skin presents an anæmic aspect even when hemorrhage has not occurred. In the cases in which the air-passages become affected there is more or less lividity of the surface, especially marked on the face and prolabia. The disease has no characteristic eruption. Rose-spots are sometimes observed and other forms of eruption—namely, erythema nodosum, urticaria, and sudamina—but these are accidental.

The symptoms referable to the respiratory system are due to fibrinous inflammation of the larynx, trachea, and the bronchial tubes. These have been already referred to among the symptoms incident to the local affections characteristic of the disease. If life continue sufficiently long, false membrane in these cases is expectorated, sometimes in the form of hollow tubes like macaroni, but generally in patches of irregular size and shape.

The desire for food is generally either notably diminished or lost. Aside from pain and difficulty in deglutition or the suffering caused by food and drinks when the mouth is affected, there is often an invincible repugnance to nourishment, so that in children, if given at all, it must be forcibly administered. Vomiting is a frequent, and in some cases a prominent, symptom.

This, as well as anorexia, is serious on account of the interference with alimentation. Diarrhœa is not uncommon, and is a symptom of bad omen. In the cases in which the diphtheritic affection extends into the œsophagus false membrane is expelled by vomiting in the form of ribbons, provided death do not take place before the exfoliation is completed.

In the majority of cases the mind is unaffected. Slight passive delirium occasionally occurs, and sometimes the delirium is active. The latter denotes great gravity of the disease. Convulsions and coma are rare, but both are of occasional occurrence. Convulsions have been observed at all periods of the disease, and coma is the forerunner of a fatal termination. It is probable that coma and convulsions, occurring in this disease, are generally due to uræmia.

Albuminuria is a frequent symptom. The proportion of cases in which it occurs is variable. It varies considerably at different times and places. Generally, the albumen contained in the urine is small in amount, but it is sometimes abundant. In the cases in which it is abundant the diphtheritic exudation is generally large and the swelling of the glands of the neck is unusually great. General dropsy in connection with albuminuria is rare; it has, however, been observed in a small number of cases. There may be evidences of the development of acute Bright's disease, in which case casts and usually some red and white blood-corpuscles will be found in the urinary sediment. Hæmaturia has been observed in cases presenting the symptoms of purpura or the hemorrhagic diathesis. As in the other febrile diseases, the urine contains a larger amount of urea than in health—a fact showing an increased waste of the tissues.

The duration of the disease is between one and two weeks. In fatal cases the duration may fall short of a week, death sometimes taking place within forty-eight hours. On the other hand, the illness may continue for an indefinite period beyond the maximum duration of the career of the disease, owing to consecutive affections or sequels.

Aside from the characteristic diphtheritic affections which are properly components of the disease, complications are rare and accidental. Pneumonitis is occasionally developed, and the affection of the kidneys, giving rise to an abundance of albumen in the urine, and sometimes to general dropsy, is perhaps to be regarded as a complication. The disease, however, may be associated with other diseases. Its concurrence with scarlatina, measles, and smallpox has been repeatedly observed. Painful swelling and redness of the joints, like that in acute rheumatism, is a rare complication.

The sequels of this disease form an important part of the clinical history. Anæmia and general debility are likely to persist for a considerable period. Feebleness of the action of the heart sometimes exists to such a degree as to lead to sudden death from syncope.

Paralysis affecting more or less the voluntary and sometimes the involuntary muscles is a characteristic sequel. The muscles of the soft palate and pharynx are oftenest affected, and paralysis here generally precedes its occurrence elsewhere. The palatine muscles are sometimes affected during the course of the disease, but generally not until after a period varying between one week and four weeks from the date of convalescence. In a slight or moderate degree the paralysis in this situation is denoted by a nasal intonation and by a certain amount of difficulty in deglutition and the regurgitation of liquids through the nostrils. In a greater degree and associated with paralysis of the œsophagus—which is happily rare—there is very great difficulty in swallowing, and it may be even necessary to introduce aliment by means of the stomach-tube. Under these circumstances sudden death has been caused by the passage of aliment into the larynx. On inspection the soft palate is seen to be relaxed and immovable. There often is anæsthesia of the mucous membrane of the throat. The paralysis affects the reflex action, so that the palate remains motionless when irritated with a pointed instrument. The existence of paralysis in this situation is indicated by a nasal intonation of the voice, and sometimes by a snoring sound in respiration. The paralysis in some cases affects the tongue and muscles of the face, so that articulation is difficult and the patient may be unable to perform the acts of suction and whistling. The act of gargling is also difficult or impossible.

The paralysis is sometimes limited to the muscles of the palate, pharynx, and mouth, but not infrequently it extends to other muscles. Exceptionally, the paralysis is manifested primarily or solely elsewhere than in the throat. Paralysis of one or more of the laryngeal muscles, either unilateral or bilateral, and associated often with anæsthesia of the laryngeal mucous membrane, may occur. Paralysis of the muscles of accommodation of the eye is not uncommon, causing defective power of adjustment in the eye for distant or near objects. This disorder is generally bilateral, but it is often more marked in one eye than in the other. Other muscles of the eye may be paralyzed, not infrequently causing strabismus. Amblyopia and transitory amaurosis occasionally occur. There is reason to believe that the amaurosis is sometimes attributable to uræmia. Disordered vision is in some cases the first of the paralytic sequels, and the paralysis is sometimes limited to the organs of sight. The other special senses—namely, taste, smell, and hearing—are sometimes affected, but rarely as compared with the sense of sight.

Paralysis of the extremities is not a rare form of diphtheritic paralysis. Certain muscles only or all of the muscles of an extremity may be involved. The lower limbs are most likely to be affected, and paralysis here generally takes precedence of its occurrence in the upper extremities. The paralysis is developed gradually, and is often preceded or attended by subjective sensations of tingling, numbness, and coldness. The loss of power over the muscles may be complete or there may be different degrees of paresis. The paralysis sometimes assumes the form of paraplegia, sometimes of hemiplegia, or one of the upper extremities may be paralyzed in connection with paraplegia, or the upper limb on one side and the lower limb on the opposite side may be alone affected. Trousseau observed the paralysis to affect the different extremities successively and recurrently. Finally, the paralysis is sometimes general—that is, all of the extremities are paralyzed—and in addition the paralysis may involve the muscles of the face, the throat, mouth, neck, and trunk, and certain of the involuntary muscles. Cases in which the paralysis is so extensive are extremely rare.

The paralysis is of the atrophic variety. The paralyzed muscles are relaxed and may present the reaction of degeneration. The knee-phenomenon or patellar tendon reflex is usually abolished, and this often occurs after an attack of diphtheria, even when no paralysis is developed.

Acute ataxia, generally of the lower extremities, has been repeatedly observed as a sequel of diphtheria, without paralysis.

In connection with the paralysis of the extremities the sensibility of the affected parts, when tested objectively, is often intact; but it may be notably diminished both as regards the sense of pain and touch. Exceptionally, there is hyperæsthesia of the integuments.

The paralysis sometimes affects the intercostal muscles and the diaphragm, giving rise to dyspnœa and involving danger from inability to carry on the respiratory acts. The muscles concerned in defecation are sometimes paralyzed, causing either retention of, or inability to retain, the feces. In like manner, the paralysis may affect the bladder, causing retention and incontinence of urine. Notable impairment or loss of virile power may be mentioned as one of the rare sequels of dipththeria.

As regards the frequency of the paralytic sequels, a nasal intonation of the voice and more or less impairment of deglutition are common, but paralysis elsewhere occurs in a minority of cases. These sequels take place in cases of mild as well as of severe diphtheria.

In cases of diphtheritic paralysis lesions have been found in the central nervous system, in the spinal nerve-roots, and in the peripheral nerves. The anatomical basis of the paralysis does not seem to be the same in all cases. In several cases which have been carefully investigated the lesions of a multiple degenerative neuritis (p. 785) have been found. The characters of the paralysis, particularly the usually rapid recovery, are in favor of the peripheral nature of the lesion in many cases.

PATHOLOGICAL CHARACTER.—Diphtheria is a general disease or an essential fever. The pyrexia and constitutional symptoms are not merely symptomatic of the inflammation involved in the local diphtheritic affections. Two views are held as regards the relation between the local lesion and the constitutional affection. According to one view, the special poison of diphtheria first enters the blood, and the pseudo-membranous inflammation of the throat or of other parts is secondary to the general disease, having to the latter a relation like that of the characteristic local affections of the skin and mucous membranes in the eruptive fevers. According to the other view, the special poison. consisting, probably, of some micro-organism, enters the circulation at the seat of the local affection, which is caused by the invasion of the parasite. It is probable that both modes of invasion occur.

A question which has occasioned much discussion and concerning which opinions differ is, whether diphtheria and membranous croup be distinct diseases. Regarded from a clinical point of view, it seems to me clear that this question is to be answered in the affirmative. There is, moreover, nothing in the pathology of these diseases opposed to this view. It is established both experimentally and clinically that pseudo-membranous inflammations of the throat may be produced by a variety of causes, such as inhalation of hot vapors, the swallowing of corrosive poisons, traumatism, and the special poisons of various infectious diseases, particularly scarlatina, smallpox, and measles. There is no ground for the assumption that aside from these causes the diphtheritic virus is the only one capable of causing pseudo-membranous laryngitis. So far as known, the pseudo-membranous exudations produced by these various causes are anatomically identical, there being nothing as yet demonstrated which distinguishes the exudations of diphtheria. In addition to the pseudo-membranous laryngitis which is produced by the special poison of diphtheria (diphtheritic croup), the author recognizes a pseudo-membranous laryngitis which has been described in a previous article as true or membranous croup. The latter affection is distinguished from diphtheria by the following circumstances: In true croup the exudation occurs primarily in the larynx, or, if the tonsils and pharynx be coincidently involved, the exudation does not extend to the nares or the œsophagus, nor does it occur in other situations. A considerable enlargement of the cervical glands does not occur. Albuminuria is not a symptom. The pyrexia and other general symptoms are purely symptomatic of the local inflammation. The disease is sporadic, not epidemic. It affects children almost exclusively. It is never followed by paralytic affections. There is no evidence of its contagiousness. The relative distinctive features of the two diseases are epitomized by saying that diphtheria is a general disease or an essential fever, whereas the so-called true croup belongs among the local diseases.

A diphtheritic affection of the throat occurs as an element of scarlatina. and the two diseases may exist in combination. The combination of the two diseases is the probable explanation of reported instances in which patients with scarlet fever apparently communicated diphtheria, and *vice versâ*. The following instance came under my observation: The patient, a boy aged four years, had well-marked diphtheria and no eruption. After convalescence he became affected with complete hemiplegia and general dropsy, the urine denoting acute diffuse nephritis. During his convalescence a sister was attacked with scarlet fever, the efflorescence being abundant. The boy had the sequels of both scarlatina and diphtheria, following what appeared to be purely an attack of the latter, and he communicated to his sister the former and not the latter disease.

CAUSATION.—Diphtheria is eminently an epidemic disease. Sporadic cases, however, occur, and of late years in large cities the disease is never wholly extinguished. Diphtheria is a contagious disease. Evidence of this is found in instances in which the disease has followed the accidental introduction of some of the diphtheritic exudation into the mouth, nostrils, eyes, or wounds of the skin. Many such instances have occurred among physicians in attendance upon diphtheritic patients. These cases prove that the contagium is present in the diphtheritic exudation. It is true that Trousseau and Peter failed to produce the disease in themselves by voluntary inoculation, but a similar immunity in individuals exists toward many diseases the contagiousness of which is acknowledged. In this regard positive evidence outweighs any number of negative facts. The experimental inoculations of animals with diphtheritic exudation has not yielded decisive results.[1] Further evidence of the contagiousness of diphtheria is afforded by the introduction of the disease into situations where it had not previously existed by patients coming from situations in which it was prevailing. There are some instances recorded of the special poison being transported by individuals who do not themselves become infected with the disease, but such cases are rare. The contagium of diphtheria often adheres tenaciously to localities where it has once gained a foothold. Thus the disease has been contracted by those occupying rooms where many months before there has been a patient affected with diphtheria. Observations have been reported which show the possibility of the transmission of the diphtheritic poison by means of milk.[2]

Defective sewerage, imperfect ventilation, overcrowding, and other unsanitary conditions favor in no small degree the development of the disease in both its sporadic and epidemic form. It is probable that this effect is produced, not by the spontaneous generation of the diphtheritic contagium, but by a favorable influence upon the multiplication of the disease-germs outside of the body or by increasing the susceptibility of the persons exposed thereto. Diphtheria, however, is not infrequent under the best hygienic surroundings.

Sporadic cases occur under circumstances which seem to render it impossible that the special germ of diphtheria had been introduced from without; but a similar apparently spontaneous development of other contagious diseases occurs, so that these instances cannot overthrow the general belief in the contagiousness of diphtheria. Still, it remains true that it is often impossible to trace the source of infection in cases of diphtheria.

The susceptibility to diphtheria is not equal at all periods of life. This susceptibility is greatest between two and seven years of age. After this period the susceptibility progressively diminishes, but it exists in considerable degree even in adults. During the first year of life diphtheria is rare. Circumstances pertaining to sex, social position, occupation, etc. do not appear to be connected with the causation. Diphtheria occurs at all seasons of the year, but it is most frequent during cold, moist, and changeable weather, perhaps because then ordinary inflammations of the throat are most common, and these may increase the readiness with which the diphtheritic virus gains entrance to the system. One attack of diphtheria does not protect against subsequent attacks. The period of incubation is generally between two and eight days; it may, however, be longer.

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[1] Some experimenters have succeeded in producing a pseudo-membranous inflammation of mucous membrane by inoculation with diphtheritic exudations; but the results of the careful experiments of Curtis and Satterthwaite, and those of H. C. Wood and Formad, are opposed to the doctrine that diphtheria can be produced in animals by inoculation.

[2] Vide article by Hart in *Proc. of International Med. Congress* of 1881.

Some persons are much more susceptible to diphtheria than others. It is often impossible to explain to what this increased susceptibility is due, but sometimes it is associated with chronic enlargement of the tonsils or with a tendency to simple, catarrhal inflammations of the throat.

Epidemics of diphtheria are irregular in their occurrence. It is a remarkable fact that they were scarcely known in America from the time of the epidemic in 1771, described by Bard, to about 1856, and since the latter date they have occurred in all parts of the continent. The disease is often limited in its prevalence in particular sections to a circumscribed area. For example, it has been observed to be restricted to a few houses or a single house or to a narrow strip of land on the banks of a stream. The special cause seems to require auxiliary causes, at present unknown, which are peculiar to certain localities.

Epidemics differ as regards certain features of the disease and the rate of fatality. In some epidemics there is an unusual tendency to an invasion of the larynx, and the amount of exudation in other situations is unusually large in the majority of cases. In other epidemics the local affection is confined to the fauces, as the rule, and the disease is generally mild. In respect to variations at different times and places, diphtheria resembles other epidemic diseases.

As has already been mentioned, nothing is positively known as to the nature of the special cause of diphtheria. There is some basis for the view that we include under the general name of diphtheria diseases dependent upon different causes. It is not probable that the diphtheritic inflammations of the throat which are often secondary to various infectious diseases, particularly scarlatina, but also to measles, smallpox, typhoid fever, and erysipelas, are caused, as a rule, by the same infectious agent which produces primary diphtheria. There are also some writers who hold the opinion that even primary diphtheria may be produced by various special causes, with corresponding differences in anatomical and clinical characteristics; but this view is far from proven.

DIAGNOSIS.—It is important to define clearly the comprehensiveness of the term diphtheria in respect to diagnosis. Some physicians are accustomed during an epidemic to consider cases of simple or follicular pharyngitis as cases of diphtheria. This is sanctioned by writers who recognize as a variety of the disease "catarrhal diphtheria;" that is, diphtheria without the local diphtheritic characteristics. It is perhaps a rational supposition that the special cause may give rise to a pharyngeal inflammation and pyrexia without fibrinous exudation; but, however rational, this is only a supposition. On the other hand, it is well known that pharyngitis without diphtheritic characters is a common affection. False statistics in regard to death-rate, and erroneous conclusions respecting the effect of treatment, can be avoided only by recognizing as an essential criterion the local diphtheritic characteristics. Adopting this rule, the diagnosis cannot be positive prior to the membranous exudation, which may be preceded for a variable period by pyrexia and other general symptoms. These, from their violence, may mislead, unless an examination of the throat be made. Thus, I have known an attack with high fever and delirium shortly after confinement to be mistaken for a time for puerperal fever. When it is an intercurrent disease in the course of another fever, it is liable for some time to be overlooked. In a case related to me by a medical friend, there being notable abdominal tenderness and great prostration, the disease for twenty-four hours was supposed to be acute peritonitis.

The diagnosis is easy when the local manifestations have taken place. The only liability to error is in confounding with the diphtheritic affection pharyngitis or tonsillitis with a deposit of mucus or an abundant follicular secretion. The appearance in some cases resembles that of a true exudation. The distinctive points are the following: A deposit of mucus or of follicular secretion is generally limited to, or especially marked over, the tonsils; it can be wiped off, but is not removable in strips; it may be seen to dip into the follicular depressions; and it is often thrown off within twenty-four or thirty-six hours. On the other hand, a diphtheritic exudation has the characters of a false membrane; it generally extends more or less over the soft palate and uvula; if removed it comes away in strips; and it is never exfoliated in thirty-six hours.

PROGNOSIS.—The proportions of deaths in collections of cases reported by different observers present much variation. This is measurably due to the fact that, irrespective of treatment, the fatality in different epidemics varies within widely-separated limits. The variations in statistics are also due in a measure to the error of confounding with diphtheria pharyngitis or tonsillitis with follicular secretion, and the error of applying the name diphtheria to all cases of sore throat occurring during an epidemic. Without attempting, therefore, to express the average death-rate in figures, it must suffice to say that it is sometimes very large and sometimes comparatively small.

The danger is, first, from an invasion of the larynx. Of the cases in which the air-passages become involved the vast majority end fatally. The prognosis in these cases is always exceedingly unfavorable. Apnœa either causes or contributes largely to the fatal result whenever the larynx becomes involved. The patient cannot be considered as secure against diphtheritic laryngitis so long as the career of the disease continues, but the danger from this source progressively diminishes after the first week.

The danger is, second, from asthenia or exhaustion. The violence of the disease may destroy life within forty-eight hours from the date of the attack, or perhaps even at an earlier period. In these cases the disease is truly called malignant. Using the language of metaphor for the want of precise knowledge of the general conditions on which the fatality in these cases depends, the vital powers are said to be overwhelmed with the force of the disease; but in the larger proportion of the cases in which the disease proves fatal by asthenia the powers of life are gradually exhausted, and death takes place in the second week. In the cases in which the larynx is unaffected the prognosis is always unfavorable if the diphtheritic membrane extend from the fauces into the posterior and anterior nares, if it pervade extensively the buccal membrane, or if it exist abundantly in other situations. The septic and gangrenous forms of diphtheria offer an especially unfavorable prognosis. Other unfavorable prognostics are—frequent vomiting, diarrhœa, copious epistaxis or hemorrhage in other situations, great frequency, irregularity, or feebleness of the pulse, coldness of the surface, abundance of albumen in the urine, convulsions, delirium, and coma.

A fact important to be borne in mind is the liability to sudden, fatal syncope in this disease. It may occur in cases which, as regards the general symptoms, do not present an appearance of great gravity. It generally follows some unusual exertion, as in getting out of bed; and it has been known to occur even during convalescence. It is probable that in some of the cases in which a fatal result occurs suddenly or unexpectedly the death is occasioned by the formation of a thrombus in the right cavities of the heart. It may also be referable to parenchymatous degeneration of the heart.

The paralytic affections which are liable to supervene do not, in general, involve danger. The danger incident to these affections relates to innutrition in the cases in which deglutition is extremely difficult or impossible, and to apnœa when the respiratory muscles are involved in the paralysis.

TREATMENT.—With reference to therapeutical indications it is convenient to divide cases into those with and those without a diphtheritic affection of the larynx. The treatment of cases in which the larynx does not become affected will be considered first. The indications in these cases relate to the general condition and to the local affection.

It is obvious that views which relate to the pathological connection between the pharyngeal affection and the general condition must have great influence upon the rational indications as regards local treatment. If the disease be produced by bacterial germs or organisms, which, received into the mouth or nostrils, find a habitat generally in the mucous membrane of the throat, and that generated here are broods of parasites which migrate into adjacent parts and into the blood, it is a reasonable supposition that local treatment is first in importance. According to this view, the object is to destroy the parasites as early, quickly, and effectually as possible. There is sufficient probability of the correctness of this view to dictate the propriety of making trial of different articles and methods with reference to a parasiticidal effect. Another view renders local treatment rationally important—namely, that which attributes the general symptoms, in a greater or less degree, to the absorption of septic matter, not accepting the theory that the morbific agency of this matter is due to a specific diphtheritic organism. According to this view, it is a reasonable supposition that to prevent septicæmia is an important object of local treatment. Rational considerations, however, no matter how strongly they may seem to be supported by morbid anatomy, together with accepted doctrines of etiology and pathology, are never sufficient for the establishment of therapeutical rules without the results of clinical experience. Making proper allowance for the variableness of the disease in different epidemics, we have not, at present, sufficient clinical data for determining the relative value of the different kinds of either the local or the general treatment of diphtheria, and we lack the important standpoint for deciding upon the influence, collectively, of all kinds of treatment—namely, the mortality when the disease is allowed to pursue its course unaffected by any therapeutical measures.

Caustic and irritating applications to the throat—namely, the nitrate of silver, hydrochloric acid, the sulphate of copper, and the strong solutions of the pernitrate or persulphate of iron—have heretofore been in vogue. They are still employed by some practitioners, and their use is sanctioned by some standard authors. They are objected to on the following rational grounds: Impairing the continuity of tissue facilitates the entrance of organisms or septic matter into the system; and observation teaches that secondary diphtheritic exudation is liable to occur in any situation on the mucous surface or skin in which there is abrasion; as, for example, after the application of a blister. Many physicians have testified to the apparent hurtfulness of these applications. On the other hand, testimony of their usefulness is not wanting. A medical friend of much experience is convinced that by the prompt and thorough application of nitric acid, if the part primarily affected can be seen and reached, the progress of the disease may with certainty be arrested. He claims that want of success can only be explained by lack of thoroughness in the application. It should be made by bringing into contact over the whole of the affected part either lint or several thicknesses of linen cloth holding the acid, with sufficient pressure for the acid to penetrate the exudation and act upon the underlying tissue.

Cauterizing applications have of late years, to a great extent, given place to those supposed to be useful by means of a disinfectant or antiseptic effect. Remedies applied for this effect are carbolic acid, salicylic acid, permanganate of potassa, chloral hydrate, sulphite of soda, benzoate of soda, subsulphate of iron, bromine, oil of turpentine, and the chlorate of potassa. Two or more of these are sometimes combined. The applications in a liquid form may be made by means of a probang, a camel's-hair brush, or a spray-producer. The latter is preferable, especially in children. The salicylic acid is conveniently applied in the form of a dry powder combined with bismuth, or it may be used in the form of a solution of salicylate of sodium (7 parts to 100 of water). The chlorate of potassa may be taken dry into the mouth, combined with pulverized sugar. It is to be remembered that the absorption of large quantities of chlorate of potassa is often attended with danger. The applications are to be made by means of injections into the nostrils if the exudation be seated in the Schneiderian membrane. Whatever be the disinfecting or antiseptic articles employed, they should not be concentrated sufficiently to cause local irritation, and they should be repeated at short intervals. Oertel advises a spray from a solution of the chlorate of potassa and salicylic acid, with, in severe cases, the addition of the permanganate of potassa, to be continued for a quarter of an hour and repeated every hour or even every half hour. The thorough cleansing of the affected parts by gargling or injections with tepid water holding some disinfectant in solution is an important part of the local treatment. Oertel attaches much importance to the promotion of suppuration, and advises for this end the frequent inhalation of hot steam. Various solutions have been used as local applications with the view of dissolving the false membrane. For this purpose lime-water, lactic acid, pepsin, trypsin, and papayotin have been employed, but it is uncertain whether they exert any decidedly favorable influence. When there is much fever swallowing bits of ice is grateful to the patient.

The degree of efficacy to be attributed to local treatment, and the particular applications to be preferred, are to be settled by further clinical experience. Physicians differ as to the importance of topical applications and as regards preferences among those which are in use. In view of the deficiency of data for exact conclusions the opinions of individual observers will have weight in proportion to their opportunities for observation and the confidence placed in their opinions.

With reference to the general treatment, if the germ theory be adopted it is a legitimate purpose of clinical observation to discover a remedy to be taken into the system for a parasiticidal effect. Several remedies are supposed to have this effect. Some physicians attribute to the sulphites efficacy in this way, given in full doses after short intervals. Twenty or thirty grains of the sulphite of sodium may be repeated every two or three hours. The sulphocarbolate of sodium, in doses of a scruple to an adult every hour or two, in the practice of a medical friend of much experience, has seemed to him to be signally useful. This remedy, improperly prepared, is sometimes extremely offensive to the taste, but, properly prepared and given with an agreeable syrup, it is not repulsive. The benzoate of sodium, given hourly in doses aggregating in twenty-four hours between fifteen and twenty-five grammes, has been used with apparent benefit in Germany, but it has no special efficacy. It is claimed by Dr. E. W. Chapman, on the basis of clinical experience, that alcohol, given early and as largely as can be borne without symptoms of alcoholism, acts as an antidote, and that, conjoined with quinia, it is an effective curative agent. Calomel, corrosive sublimate, and other preparations of mercury, given in frequently-repeated doses, are recommended for their germicide effects, and have apparently proved useful. Pilocarpine has been administered internally with the idea that by inducing abundant secretion it aids in the detachment of the false membrane, but the favorable results at first reported as following the use of this remedy have not been confirmed by subsequent experience. We may hope that for this disease and for other infectious diseases remedies may be discovered which will have a controlling influence, like that of quinia in the malarial fevers; but at present we know of no drug which exerts a specific curative influence over diphtheria.

Aside from remedies for a special controlling influence, in view of the fact that if the larynx be not affected the danger is chiefly from asthenia, the great object of treatment is support. Much importance belongs to alcohol as the most efficient factor in the supporting treatment. The principles which should govern its employment are the same as in the essential fevers and in other diseases tending to destroy life by asthenia. These principles need not here be repeated. In this disease, as in the essential fevers, there is sometimes a notably increased tolerance of alcoholics. Jenner cites a case in which two drachms of brandy were given hourly with advantage to a child three years of age. In a case of great severity which came under my observation half an ounce of brandy was given hourly, the patient being thirteen years of age, without any excitant effect, the case ending in recovery. In the use of alcoholics the physician is to be guided by the indications and by carefully watching the immediate effects. Alimentation is an essential part of the treatment. The diet should be concentrated and highly nutritious, and it should embrace the necessary variety of alimentary principles. Milk meets pre-eminently these requirements. A serious difficulty in the treatment often arises from the invincible repugnance to nutriment, and sometimes from the persistence of vomiting. Owing to the difficulty of alimentation in some cases, and sometimes a want of appreciation of its importance, death takes place from innutrition. Of tonic remedies, the sulphate of quinia and the tincture of the chloride of iron are very useful.

The measures heretofore known as antiphlogistics—namely, bloodletting, general or local, purgation, emetics, and nauseants—are rationally contraindicated by the simple fact that the danger is not from the inflammatory manifestations of the disease, but from the general condition which tends to destroy life by asthenia. Clinical experience has abundantly shown these measures to be injurious. Blisters and other counter-irritant measures are not to be employed, occasioning, as they are likely to do, new foci of diphtheritic inflammation.

Symptoms incidental to the disease claim palliative treatment. Vomiting is to be relieved by bismuth, creasote, hydrocyanic acid, etc. Diarrhœa calls for opium and astringents. Anodynes—that is, opium or its succedanea, the bromides, or the chloral hydrate—are often indicated by restlessness and vigilance. Hemorrhage indicates the use of hæmostatics topically and internally.

The treatment of cases in which diphtheritic laryngitis becomes developed involves the same general principles as that of cases without the laryngeal affection; but, in addition, measures are indicated to hasten the separation of the false membrane within the larynx, precisely as in cases of laryngitis with false membrane disconnected from diphtheria; that is, in cases of sporadic or primary so-called true croup. The most efficient of the measures for this end is the inhalation of warm vapor or steam. With reference to this measure and other points relating to the treatment directed to the laryngeal affection the reader is referred to the article on Laryngitis with Fibrinous Exudation.[1]

The rules with respect to tracheotomy when the danger from laryngeal obstruction is imminent are the same as in cases of primary croup. There is undoubtedly less hope of success from surgical interference than in the latter affection. With reference to the propriety or importance of the operation, however, the simple question is, Are lives ever saved by it? This question is undoubtedly to be answered in the affirmative. The question, How many lives are saved? is of less importance in its practical bearing. If lives be ever saved by tracheotomy in diphtheria, a practitioner is reprehensible if he allow a patient to die from laryngeal obstruction without opening the trachea. The patient is entitled to the chance of being saved by the operation, however small that chance may be.

The method of treating laryngeal stenosis by passing a tube into the larynx (intubation) has been practised with considerable success by Dr. O'Dwyer of New York, and has been recently advocated by him. Other practitioners have in general had less favorable results in employing this method than those obtained by Dr. O'Dwyer. Experience is not yet sufficient to determine the value of intubation of the larynx in cases of diphtheria and croup.

The propriety of precautions against contagion has been already referred to. Patients affected with the disease should be at once isolated. Whenever practicable, the removal, especially of children between three and twelve years of age, from an infected house or district is advisable. Thorough disinfection of the dejections, of all articles which have been in contact with patients, and of the apartments which they have occupied is to be enjoined. Auxiliary causes are to be sought after in defective sewerage or drainage, overcrowding, want of ventilation, etc., and these removed if discovered.

The diphtheritic paralyses may be treated by massage, by the employment of the galvanic current, and by hypodermic injections of small doses of strychnine.

Milk Sickness.

During the early decades of this century the disease known in the lower animals as the *trembles*, and in human beings as the *milk sickness*, prevailed extensively in many of the frontier settlements of what were then the Western and South-western States of this country. The disease has gradually receded before the advances of improved agriculture, but it still retains a foothold in certain districts where it has been endemic. The States in which the disease has most frequently been observed are Indiana, Illinois, Ohio, Kentucky, Tennessee, Missouri, and Michigan; but cases have occurred in Western Pennsylvania, Virginia, Alabama, Georgia, and North and South Carolina. The disease is not known to have occurred east of the Alleghany Mountains, west of the Great American Desert, or in the countries of the Old World. The descriptions of the disease, coming as they do in great part from recently-settled regions where medical science has not reached a high development, are very imperfect. Notwithstanding the doubt which has been cast upon the very existence of the disease as a specific affection by some authors, and the little attention which it has received in medical literature, there is sufficient evidence for the recognition of a peculiar and specific disease which has received the names already mentioned.

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[1] Vide p. 291.

The name milk sickness is based upon the belief that the disease is communicated to man by the use of the milk of cows affected with the trembles. This name is objectionable, on the ground that there is evidence of communicability also by the use of the flesh of animals thus affected.

The disease affects primarily herbivorous animals, especially cattle, and less frequently horses, sheep, goats, and wild animals. Unweaned calves, lambs, and colts are especially liable to the disease. Animals giving milk do not manifest the characteristic symptoms so soon as those which are not giving milk.

The leading symptoms of *trembles* occurring in cattle are at first languor, indisposition to exercise, and abstinence from food, and then, when the disease is fully developed, weakness, so that the animal is unable to stand upright; trembling, thirst, obstinate constipation, a lustreless and congested condition of the eyes, coolness of the surface of the body, and labored respiration. Convulsions may occur. Most writers mention a peculiarly offensive odor of the breath. Mild cases occur, but generally, if the symptoms be well marked, death takes place within a few hours or a few days after the first manifestations of the disease. A predisposing cause of an attack often is fatigue. Exercise is sometimes employed to determine whether the disease exist in a latent or prodromic stage. Thus, cattle-dealers in milk-sick districts often chase the animals for a while in order to test their freedom from trembles.

The disease is most common in late summer and autumn, but it may occur in the winter. It is more common in warm, dry seasons than in wet seasons. It occurs only in certain localities, which are often quite narrowly circumscribed. The special cause may exist in a certain tract of land on a farm, and be absent from the land immediately adjoining. Wild and unimproved lands, especially those which are densely timbered, seem to be the favorite situations for the special poison, while open prairies and fields which are freely exposed to the sun are not favorable for its development. Cultivation of the soil causes the disease to disappear. The disease has not been observed to originate in regions where it did not previously exist, whereas, on the other hand, it has disappeared from the endemic regions after their subjection to cultivation. It is the general opinion of the farmers in the infected districts that the animals do not contract the disease during the daytime, but only when they are allowed to remain out during the night. So far as is known, no special quality of the soil characterizes the infected regions.

It is not disputed that the trembles is due to some special poison which is present in the ground, but we have no knowledge as to the nature of this poison. The idea that the poison is a mineral substance has received no support in facts, and is now generally abandoned. The prevalent view is that the disease is caused by the ingestion of some poisonous vegetable. A number of vegetables have been accused, such as certain poisonous Umbelliferæ, the Eupatorium ageratoides, the Rhus toxicodendron, and certain fungi. Although there is no evidence that any of these plants causes the disease, the theory of a vegetable poison is not without considerable probability. There are also, especially of late years, advocates of the theory that the trembles is caused by a special micro-organism, and Phillips claims to have found a spirillum in the blood. Graff found that the flesh of a dog which had been poisoned by feeding him with meat from cows affected with the trembles was no less poisonous than the cow's flesh. This experiment is an argument in favor of the multiplication of the poison within the body. It is, however, desirable that these experiments should be repeated before any conclusions are drawn from them.

In all regions where the trembles prevails among cattle an apparently identical disease, called the *milk sickness*, occurs in human beings. It is generally admitted that milk sickness is caused by ingesting either the milk or the flesh of animals affected with the trembles. As has already been mentioned, the milk may contain the special poison even when the cow presents no manifestations of the disease, these being warded off, apparently, by the excretion of the poison through the mammary gland. Butter and cheese made from the infected milk are also poisonous, and hence the opinion of physicians in milk-sick districts is probably correct that cases of this disease must occur in cities, but that its true nature is not recognized by the medical attendants. Young children are said to be less liable to the disease than adults. It has not been proven whether or not the special poison be destroyed by boiling the infected milk or by cooking the meat.

The period of incubation is variable, being sometimes one or two days, sometimes several days, and, according to some writers, occasionally several weeks. The first manifestations of the disease often follow some special exertion. The premonitory symptoms, which may be of short duration or may last for several days, are malaise, lassitude, fatigue after slight exertion, pain in the head and limbs, anorexia, and slight nausea. As these symptoms become more pronounced, extreme nausea and vomiting supervene, accompanied by intense thirst, obstinate constipation, and a burning pain in the epigastrium. The temperature of the surface, particularly of the extremities, is below the normal, and the internal temperature is not usually much elevated. The skin is dry, and the abdomen is retracted, with little if any tenderness on pressure. The pulsations of the abdominal aorta are prominent. Intestinal peristole seems to be wholly suspended. The tongue is swollen and flabby, presenting the impressions of the teeth, and when protruded is tremulous. At first moist and thickly coated, the tongue may later become red, dry, and fissured. The breath is offensive, and it emits an odor which nearly all writers speak of as indescribable but characteristic, so that by means of this peculiar fetor the disease can be recognized. The urine is diminished in quantity, and sometimes its secretion is even suspended. The pulse varies in frequency, but it is always weak and compressible. The action of the heart is often labored, without any corresponding change apparent in the pulse at the wrist. The eyes are often suffused with blood. The mental state is at first one of restlessness and irritability, and later one of hebetude and indifference, passing toward a fatal termination into somnolence and coma. Convulsions sometimes occur. Vomiting continues until from extreme exhaustion it is replaced by only a feeble retching. The vomited material, consisting at first of the ordinary contents of the stomach, later contains mucus, sometimes tinged with blood. It may be frothy, but it rarely contains much bile. In the later stages the color of the vomit is said to sometimes resemble water colored with indigo-blue. Black vomit, due to hemorrhage, is a rare occurrence. The patient may sink into a typhoid state with low, muttering delirium. Death, if it occur, is from asthenia.

The duration of the disease is variable. There are mild cases which hardly pass beyond the premonitory stage. On the other hand, there are cases in which the symptoms develop rapidly and with great intensity, a fatal termination occurring within two or three days of the onset. More frequently the duration of fatal cases is between five and fourteen days. Chronic cases lasting for several weeks have been described.

It is difficult to form an estimate of the prognosis from the existing data. The writers of the early part of this century state that the majority of those affected die, whereas more recent authors give a comparatively favorable prognosis. Perhaps this more favorable result is due to improved methods of treatment. Recovery is slow.

Nothing is positively known concerning the pathology of milk sickness. The symptoms suggest an intense gastritis, but in the few recorded autopsies mention is made only of redness of the mucous membrane and contraction of the stomach. The intestinal canal has been found markedly contracted. With the exception of these alterations, the results of post-mortem examination have generally been negative.

The objects to be sought in the prophylaxis of milk sickness are apparent from the account which has been given of its etiology. The former treatment of the disease consisted in bleeding, drastic cathartics, and blisters. The modern treatment has substituted, and with beneficial results, the use of stimulants and restoratives. Notwithstanding some contradictory testimony, it is probable, in view of the extreme irritability of the stomach, that opium judiciously administered is useful. Quinine is highly recommended by some writers. Alcoholic stimulants are now freely employed in this disease. Counter-irritation to the epigastric region, as by a mustard-plaster, may be tried. While the older practitioners thought that the leading indication was to open the bowels, there are now some physicians who are opposed to the administration of cathartics, on the ground that they increase the irritability of the stomach and do not accomplish their purpose. Enemata may be employed as the least disturbing of the cathartic remedies. Agents to allay vomiting, such as bits of ice, carbonic-acid water, and creasote, should be given. The diet should be chiefly liquid and easily digestible.[1]

CHAPTER X.

ACUTE, SUBACUTE, AND CHRONIC ARTICULAR RHEUMATISM.

Acute Articular Rheumatism: Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Subacute and Chronic Articular Rheumatism.

THE term *rheumatism* is used to denote a constitutional disease characterized by certain local manifestations seated in the articulations and the fibrous tissues in other parts. The term has also been applied to neuralgic and other affections supposed to arise from the constitutional morbid condition existing in the ordinary form of the disease. I shall consider *first*, *acute articular rheumatism* or *rheumatic fever;* and *second*, *subacute* and *chronic articular rheumatism.* The affection known as *muscular rheumatism* has been considered in connection with diseases of the nervous system under the name Myalgia. The disease called *gout* is in certain respects analogous to rheumatism, although presenting essential points of difference. After having considered the forms of rheumatism just named, I shall consider, in the next chapter, gout and rheumatoid or deforming arthritis, often called rheumatic gout.

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[1] The following are among the articles which describe this disease: Hirsch, *Handb. d. Historisch-Geog. Pathologie*, Bd. 2; Drake, *A Memoir on the Disease called by the People the Trembles and the Sick Stomach or Milk Sickness*, Louisville, 1841; Graff, "On the Milk Sickness of the West," *American Journ. of the Med. Sciences*, April, 1841; "Report on Milk Sickness," *Trans. of the Kentucky State Medical Society*, 1868; Beach, "Milk Sickness," *The Journal of the American Med. Assoc.*, July 28, 1883; Fulton, *Philadelphia Med. and Surg. Reporter*, Apr. 12, 1884. A thorough monographic treatment of the subject is much to be desired.

Acute Articular Rheumatism—Rheumatic Fever.

Acute articular rheumatism, as the name imports, involves an affection of the articulations or joints. The affected joints present the phenomena of inflammation, and the affection is also called acute rheumatic arthritis or polyarthritis. Opportunity is rarely offered to study the morbid appearances within the joints in cases of acute articular rheumatism. Often when symptoms referable to the joints have been well marked during life little change is to be found in these structures after death. It may be supposed that in these cases the condition is one chiefly of hyperæmia and œdema. In many cases, however, the articular cavity contains an increased amount of fluid, which is more or less turbid from admixture with pus-cells. The synovial membrane may be reddened from hyperæmia and thickened from serous infiltration. In some cases flocculi of fibrin have been found within the articular cavity. Much of the swelling about the joints during life is due to inflammatory œdema of the tissues surrounding the affected parts. The quantity of fibrin or of fibrin-factors in the blood is notably increased. In the course of the disease certain internal organs are liable to become inflamed. The inflammation is generally seated in the fibro-serous tissues, and especially in the endocardium and pericardium. In a pathological view the endocarditis, pericarditis, and other internal inflammations which occur in cases of acute articular rheumatism are local manifestations of the disease, like the arthritic affections, but it is most convenient to consider them in the light of complications.

Clinical History.—Acute articular rheumatism, in the majority of cases, begins with a sudden attack, and in nearly one-half of the cases in which the attack is sudden it takes place at night. In a small proportion of cases pyrexia precedes the local manifestations for a period varying between a few hours and one or two days; hence one reason for the name *rheumatic fever* instead of acute rheumatism. In most cases, however, the fever and the local manifestations are simultaneous in their occurrence. Sometimes pain and soreness of the joints, together with indefinite ailments, precede the development of the disease for a variable period.

The development of the disease is denoted by an affection of one or more of the articulations. The larger joints (knee, shoulder, or elbow) are first affected in the great majority of cases. The local phenomena are pain, tenderness, increased heat, swelling, and redness of the skin. The pain differs in intensity in different cases, but, as a rule, it is not intense so long as the parts are perfectly quiet. Pain is especially excited by movements of the affected joints. In severe cases the slightest motion is insupportable, and even jarring the bed or room occasions suffering. Pressure over the joints is painful. Owing to the pain occasioned by movements, the patient maintains as long as possible a fixed position. Changes of the decubitus often cause extreme suffering. Swelling is most apparent in joints not covered with muscle—namely, the knee, wrist, elbow, ankle, and the smaller joints of the hands and feet. It is less apparent in the hip and shoulder. The swelling is due, in part, to a morbid increase of liquid within the synovial cavity, and in part to serous effusion into the surrounding structures. The redness is due to an erythema in the form of patches or zones which do not present well-defined borders. The redness, like the swelling, is not apparent over the hip and shoulder, but only over joints that are comparatively superficial.

The swelling and the redness are, in general, proportionate to the acuteness of the rheumatism.

In some cases several joints are affected either simultaneously or in quick succession (polyarthritis); but in other cases the affection is limited to a single joint for a greater or less period (monarthritis). In the course of the disease, in most cases, various joints are successively affected, and frequently a greater or less number of joints are affected at the same time. It is a peculiarity of the rheumatic inflammation to leave one or more joints abruptly, the local phenomena sometimes disappearing within a few hours, and to attack as abruptly other joints. Cases differ as regards the number of joints affected in the course of the disease. In some cases few or no joints escape, and in other cases the rheumatic inflammation remains limited to a few joints or even to a single joint. The fibrous tissues elsewhere than within and around the joints, and exclusive of the visceral organs, are liable to be attacked. Thus, those of the dorsum of the hand and of the instep, the ligaments of the neck and back, and the sclerotic tunic of the eye are sometimes involved. The sheaths of the tendons and the bursæ are liable to rheumatic inflammation.

The disease is extremely variable as regards the number of joints affected, either simultaneously or successively, the order in which they are attacked, etc., yet the local manifestations are governed by an important pathological law—namely, the law of parallelism. Corresponding joints are often affected together, and when this is not the case the different affected joints are either on one side of the body, or joints on the two sides which are analogous—namely, the knee and elbow, wrist and ankle, hip and shoulder—are affected in combination. In an analysis of 21 cases with reference to this point, out of 88 instances in which either a joint was affected singly or more or less joints affected in combination, there was but a single violation of the law of parallelism.[1] This disease, therefore, is eminently one of the bilateral or symmetrical diseases. As respects the relative liability of the different joints to become affected, the analysis just referred to showed the order to be as follows: The knee, ankle, wrist, shoulder, hip, elbow, phalangeal joints, first of the hands and second of the feet, and the maxillary joint.

Acute articular rheumatism is always accompanied by more or less pyrexia. In addition to the fact that the pyrexia sometimes precedes the local manifestation, a reason for the propriety of regarding the disease as an essential fever is to be found in the fact that the pyrexia is not in proportion to the number of joints affected or to the intensity of the rheumatic inflammation, as denoted by pain, soreness, heat, etc. The pulse rarely exceeds 100 per minute. The axillary temperature in different cases varies between 102° and 110° F. Sweating is generally a symptom more or less prominent, occurring especially at night. The sweat emits a notably sour odor. In connection with profuse sweating, sudamina or miliary vesicles not infrequently appear on the neck and trunk. The appetite is either lost or greatly impaired. Thirst is more or less urgent. The tongue is frosted or coated. The saliva becomes acid. Generally the bowels are constipated, but exceptionally there is diarrhœa. The urine is diminished in quantity; its specific gravity is high, owing chiefly to a decrease of water; the coloring matter is increased, and on cooling the urates are deposited in abundance. Owing to the increase of the pigment of the urine the deposit is usually of a reddish color. The deposit of this lateritious sediment does not show an increase of the amount of uric acid in the urine, but only the diminution of water. Quantitative analyses of the urine have shown the quantity of uric acid not to be greater than in health. The chlorides are sometimes deficient, and occasionally the urine is slightly albuminous. Pain in the head is not common. The intellect, as a rule, is not disturbed, but mild delirium is sometimes observed. In general the strength is well preserved. Sleeplessness is usually a source of discomfort and more or less prostration, sleep being prevented by pain on change of position and by the perspirations.

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[1] "Analysis of Twenty-one Cases of Articular Rheumatism," by the author, *Buffalo Medical Journal*, March, 1854.

An important feature of the disease is its tendency to invade certain of the structures of the heart. Endocarditis occurs in a considerable proportion of cases. It is, however, less frequent than has heretofore been supposed, as already explained in treating of that affection. The inflammation, as a rule, is limited to the membrane lining the left cavities of the heart, and it affects especially the portion of this membrane situated upon the mitral valve. In a large proportion of the cases of valvular lesions of the heart, especially mitral lesions, their point of departure is rheumatic endocarditis. As a rule, the endocarditis is simple and devoid of immediate danger; but in a small proportion of cases the endocarditis is of the fatal variety distinguished as ulcerative or malignant. Pericarditis occurs less frequently. As a rule, when pericarditis becomes developed endocarditis coexists. For a consideration of these affections, as developed in rheumatism, the reader is referred to the section devoted to diseases affecting the circulatory system. It may be repeated here that pericarditis, and probably endocarditis, sometimes precede the local manifestations elsewhere. It is convenient to speak of these affections as complications, but, properly speaking, they are to be reckoned among the local manifestations of the disease; that is, they are dependent on the same determining cause to which the affection of the joints is to be referred. The structures of the heart are liable to become involved in proportion to the acuteness of the rheumatism. They may become involved at any time in the course of the disease; but the liability is greatest in the early and middle periods. They are more likely to occur the younger the patient. They occur rarely in patients more than forty years of age. Acute dilatation of the heart may occur as in other febrile diseases attended with marked debility. A murmur of temporary duration, referable to acute dilatation of the right ventricle, may occur, as has been pointed out by Sibson. Examination of the blood by means of the hæmacytometer has shown in some cases a notable diminution of red blood-corpuscles.

In a small number of cases of acute articular rheumatism important symptoms develop which are described under the names of *cerebral rheumatism* and *rheumatic hyperpyrexia*. Under the name cerebral rheumatism cases have been described which present symptoms referable to different pathological conditions, some without and some with demonstrable lesions of the brain. The most important group of cerebral symptoms, and the one commonly understood as cerebral rheumatism, is that which is generally associated with very high temperature. It is by no means certain that the high temperature itself is the cause of these grave symptoms. It is at least not improbable that the same cause which, acting upon the thermic centre in the brain, produces hyperpyrexia, acts also upon other parts of the brain, producing various disturbances of the cerebral functions. The nervous symptoms may appear at the onset of the disease, but more frequently they develop at some later period. They are ushered in by rise of temperature, restlessness, disturbed sleep, and headache. Delirium, often violent, but sometimes of a low, muttering character, makes its appearance. Muscular twitchings, and even tonic or clonic spasms, are not infrequent. The pulse becomes feeble and very frequent; a state of semi-consciousness or stupor with great prostration ensues; the face becomes cyanosed; and the temperature rises higher and higher, reaching sometimes an elevation of 110° or even higher. In the majority of cases death takes place during coma. In some cases recovery sets in even after the appearance of very grave symptoms. In fatal cases no lesions of the brain or of the meninges can usually be found. The symptoms seem to be referable to some profound infection or intoxication which acts upon the thermic and other nervous centres.

Purulent meningitis may occur as a complication of rheumatism, but this is very rare. More frequent, however, is the occurrence of cerebral embolism resulting from endocarditis. Uræmia with its attendant nervous phenomena is a rare complication of rheumatism.

A pathological relationship is supposed to exist between chorea and rheumatism. These two affections, however, are not often associated. There is little ground for the assumption that they involve in their causation the same specific causative agent.

Insanity, especially melancholia, has been observed as a rare complication or sequel of acute rheumatism. The prognosis in these cases is generally favorable. Sometimes marked hysterical symptoms develop during an attack of rheumatism.

In addition to sudamina or miliaria already mentioned, urticaria, erythema nodosum, especially on the lower extremities, and herpes labialis, occasionally occur in the course of the disease. The skin is sometimes the seat of purpura and more or less diffuse hemorrhagic extravasation. In the centre of urticarial papules in some instances there are petechial spots (urticaria hæmorrhagica). The prognosis is grave in the rare cases of a general hemorrhagic diathesis (hemorrhage from mucous surfaces, etc.) occurring in acute rheumatism. In these cases there are usually severe constitutional symptoms and high fever.

The development of small subcutaneous, firm, movable nodosities during or after an attack of acute rheumatism has been noted as an exceptional occurrence by several observers.[1] These nodosities, which, save some tenderness on pressure, are generally painless, are attached to the tendons, fasciæ, periosteum, etc. They persist from a few hours, or usually a few days, to several months. They are unattended by marked local or constitutional symptoms. They are composed of granulation-tissue.

Of complications of acute rheumatism other than those which have received consideration, the following may be mentioned, all of which are infrequent: pharyngitis, bronchitis, pneumonitis, peritonitis, nephritis, myocarditis, embolism of other than cerebral arteries, phlebitis, suppurative arthritis, erysipelas, pyæmia, and paralysis and atrophy of muscles in the neighborhood of the affected joints.

The *duration* of acute articular rheumatism varies greatly. The disease ends by self-limitation, but the minimum and the maximum of duration are widely apart. These statements have reference to the course of the disease when left to itself; that is, no active measures of treatment being employed. In 1862, I observed at Bellevue Hospital 13 cases which were allowed to pursue their course uninfluenced by therapeutical interference, some palliative measures being alone employed. I am not aware that prior to this date any one had undertaken to study in this way the natural history of the disease. I reported the 13 cases in the *American Journal of the Medical Sciences* for July, 1863, entitling my article "A Contribution toward the Natural History of Articular Rheumatism." In making an analysis, in 1854, of 24 cases which I had recorded, I was impressed with the importance of obtaining a knowledge of the natural history of the disease as the point of departure for the study of therapeutical measures. With reference to this point in my report of the analysis I used the following words: "At the present moment we cannot answer the question, What are the intrinsic tendencies of articular rheumatism as respects its continuance, its complications, and its remote consequences in the organism? Were we able to answer this question by an appeal to facts, we should then have a criterion by which to estimate the favorable or unfavorable influences of different methods of treatment pursued in a series of cases; as it is, in bringing statistical information to bear on the therapeutics of the disease we can only study the immediate effects of different remedies and institute comparisons in this point of view, and also with reference to the duration of the disease, etc. in different series of cases treated by different methods."[1] In my "Contribution to the Natural History of Articular Rheumatism," published in 1863, I premised a report in full of all the cases (13) by the following account of my plan of study: "On entering on duty at Bellevue Hospital in August, 1862, I resolved to treat with only palliative measures the cases of articular rheumatism which should be received into my wards, so long as circumstances might lead me to conclude that by continuing this plan no injustice was done to the patients, whose relief was of course paramount to any other object. The cases thus treated progressed so satisfactorily that I found no ground for a discontinuance of the plan. The last of the cases, in fact, was the only one in which any important complication occurred. Some of the cases were recorded by myself, and the remainder by my clinical assistant, Dr. Shiverick." . . . . "The reported cases were treated throughout the whole course of the disease with only palliative measures. These measures consisted of opium in some form given in small or moderate doses, the application generally of dry flannel to the affected joints, and the external use of either the soap-and-opium liniment or the tincture of aconite. But to secure the moral effect of a remedy given specially for the disease a 'placebo' was given, consisting, in nearly all the cases, of the tincture of quassia very largely diluted. This was given regularly, and became well known in my wards as the 'placeboic remedy' for rheumatism. The favorable progress of the cases was such as to secure for the remedy generally the entire confidence of the patients. I may add that all the cases were brought before the medical class in attendance during the winter."

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[1] Barlow and Warner, *Trans. International Med. Congr.*, London, vol. iv., 1881; Troisier, "Les Nodosités rhumatismales sous-cutanées," *Union médic.*, 1884.

Of these 13 cases an endocardial murmur existed in 11, but the murmur was limited to the base of the heart in all but 3 cases. Moreover, all the patients but 2 were women; and in women affected with articular rheumatism a systolic basic murmur is the rule with few exceptions, the murmur being generally of hæmic origin. In only one case was pericarditis developed. In this case the pericarditis occurred immediately after the patient's admission. Pneumonia occurred subsequently. The patient, however, recovered.[2]

The duration in these cases respectively, from the date of attack to convalescence, excluding the case in which pericarditis and pneumonitis occurred, was as follows: In 3 cases, less than fifteen days; in 1 case, between fifteen and twenty days; in 3 cases, between twenty and twenty-five days; in 3 cases, between twenty-five and thirty days; and in the remaining 2 cases, forty-five and fifty-six days. The mean duration was a fraction less than twenty-six days.

These cases establish the fact that acute articular rheumatism belongs among the self-limited diseases. Two years after the publication of my report in 1863—namely, in 1865—a communication by Dr. Henry G. Sutton, embracing a tabulated report of 41 cases treated chiefly with mint-water, appeared in *Guy's Hospital Reports.* 22 were males, and 19 were females. The average duration from admission to the cessation of pain, the skin being cool and no relapse taking place, was in the male cases 13.5 days, and in the female cases 15.5 days. The average number of days in hospital was for males 27.6 days, and for females 26.3 days. The duration prior to admission into hospital in many of the cases was considerable, the longest being six weeks. Pericarditis became developed in 4 cases, and pleurisy in 5 cases. In a second report by Dr. Sutton of cases treated in the same manner, in a subsequent volume of *Guy's Hospital Reports*, the average duration to convalescence was nine days; and in a third report of 25 cases in the *Trans. Royal Med.-Chirurg. Society*, 1869, the average duration was ten days.[1]

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[1] Vide *Buffalo Medical Journal*, March, 1854, vol. ix. p. 557.

[2] Vide "A Contribution toward the Natural History of Articular Rheumatism," by the author, *American Journal of the Medical Sciences*, July, 1863.

The illness of patients affected with rheumatism may be indefinitely prolonged by pericarditis and pleuritis; but of the cases which I analyzed in 1854, embracing 5 cases of endocarditis and 1 case of endo-pericarditis, different methods of treatment being employed, the minimum duration was seven days, the maximum twenty-six days, and the average duration a fraction more than sixteen days.

A peculiarity of rheumatism is that it does not pursue a steady, continuous march from the beginning to the end, but its course is often marked by notable fluctuations as regards the general and local symptoms. Not infrequently, after a few days the affected joints are nearly free from pain, soreness, etc., the febrile movement subsides, and convalescence appears to be near at hand, when suddenly the local manifestations and the pyrexia are renewed with perhaps as much or even more intensity than before. This may occur repeatedly during the course of the disease.

PATHOLOGICAL CHARACTER.—There have been various theories as to the pathological character of rheumatism, none of which has gained general acceptance. That the disease involves the entrance of some morbid principle into the blood is generally admitted, and is a reasonable supposition in view of the clinical and anatomical characteristics of acute rheumatism. The opinion has been prevalent that this morbid principle is uric acid or lactic acid; but examinations of the blood have failed to reveal any excess of these acids, and their agency in the causation of rheumatism is now generally discredited. The theory advocated by J. K. Mitchell, that acute articular rheumatism is dependent upon some affection of the nervous system, has not met with favor. There is a growing belief that acute rheumatism belongs to the class of infectious diseases, and is caused by the presence of a specific micro-organism. The course and the symptoms of the disease can be explained better upon this supposition than upon any other which has been suggested. The occasional endemic and epidemic occurrence of acute rheumatism is in favor also of its dependence upon some specific infectious agent.

CAUSATION.—Rheumatism is one of the diseases supposed to be frequently, if not generally, caused by suppression of the functions of the skin, and attributed especially to the action of cold upon the surface of the body. This view of the causation has but little foundation. It is true that patients are likely to refer the disease to cold, but they often fix upon some particular exposure under the belief that this agency is of course involved, for it is a popular notion that rheumatism generally proceeds from cold. A careful interrogation will show in many, if not most, cases that the statement has little to support it; and how often is exposure to cold not followed by rheumatism! That the causation may involve the agency of cold is not to be denied *in toto;*

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[1] In Dr. Sutton's communications there is no reference to my report in the *Amer. Journ. of the Med. Sciences*, 1863.

but the considerations which are to follow render it probable that cold acts only as an exciting or accessory cause.

A special predisposition is requisite; in other words, the disease involves a rheumatic diathesis. This predisposition or diathesis may be congenital and inherited or it may be acquired. There are persons congenitally liable to the disease. This is shown by the occurrence of the disease in early life, and by its recurrence more or less frequently in the majority of the persons who are once attacked by it. Statistics establish conclusively the hereditary transmission of this predisposition. It is a matter of common observation that the disease prevails among the different members of certain families.

Age has a decided influence in the causation. It occurs very rarely at less than five years of age. It may, however, occur in infancy. I have met with a case in which it occurred at one year of age, aortic lesions and hypertrophy of the heart existing at the age of two years. In the majority of the cases of a primary attack patients are between fifteen and thirty years of age. If a person have not experienced one or more attacks before the age last named, the liability to the disease afterward diminishes and becomes quite small after the age of fifty. The number of males attacked by this disease exceeds the number of females, but the difference is not great. External influences pertaining to season play a small part in the causation. The disease prevails most in humid, changeable climates. It is a disease of frequent occurrence in all climates within the temperate zone, but is comparatively rare in tropical and frigid latitudes.

Accepting the parasitic doctrine, an essential causative factor, acting in conjunction with a special predisposition, is a specific micro-organism. This is a logical conclusion, but the demonstrative proof has not as yet been obtained.

The predisposition or susceptibility is not destroyed after the disease has once occurred, as in cases of certain other infectious diseases. In a considerable proportion of cases recurrences take place.

Acute articular rheumatism may occur as a complication of various diseases; but it is not probable that the articular affections which occur sometimes as complications or sequels of scarlatina, gonorrhœa, syphilis, diphtheria, puerperal fever, dysentery, purpura, and some other diseases are in most cases etiologically identical with primary acute articular rheumatism.

DIAGNOSIS.—The disease is to be discriminated from gout. For the differential points the reader is referred to the chapter treating of the latter disease. In general, the diagnostic features of acute articular rheumatism are so obvious that the disease is at once recognized. The chief difficulty in diagnosis, exclusive of the discrimination from gout, relates to cases in which the rheumatic affection is confined to a single joint. The problem in these cases is to distinguish rheumatism from a purely local affection of the joint; that is, *synovitis* or *simple acute arthritis*. The following are the points in this differential diagnosis: The fixedness of the inflammation in a single joint from the first in the latter, whereas in articular rheumatism it is rare that the mobility of the disease is not to some extent manifested; the existence of pyrexia in some cases of articular rheumatism prior to the affection of the joint; the want of correspondence between the degree of pyrexia and the local phenomena in rheumatic cases; the long duration of the local affection in cases not rheumatic, and in certain cases the swelling and redness being greater than in rheumatic cases; the rapid disappearance of the local affection often in cases of rheumatism, and the speedy recovery of the use of the affected joint, the disappearance and recovery being more slow in cases not rheumatic; the occurrence of endocarditis or pericarditis in a certain proportion of rheumatic cases. The age of the patient and the previous occurrence of rheumatism are points to be taken into account.

Cases of pyæmia in which a purulent effusion takes place into certain joints are sometimes mistaken for cases of rheumatism. Attention to the antecedent and concomitant circumstances should prevent this error. Purulent collections within the joints in cases of puerperal fever are embraced in this category. The so-called gonorrhœal rheumatism is generally monarthritic, and the association with gonorrhœa or gleet points to the character of the affection.

PROGNOSIS.—This disease is rarely fatal, and the immediate danger to life is small. When death takes place during the career of the disease it is due to complications, including under this head pericarditis; but the majority of cases in which pericarditis occurs do not end fatally. Active delirium, convulsions, and coma denote imminent danger, but it has been seen that these are extremely infrequent events. Pulmonary complications may prove serious. A source of danger in some instances is intense hyperpyrexia. The development of the hemorrhagic diathesis, with hemorrhages into the skin and from mucous membranes, is a grave complication.

The disease, aside from the complications just referred to and the suffering attending it, is serious in view of the liability to endocarditis which may lay the foundation for incurable and grave lesions of the heart. The immediate danger from simple rheumatic endocarditis is slight; and the lesions resulting from this affection rarely occasion inconvenience until after the lapse of several years. Grave cardiac lesions do not invariably follow rheumatic endocarditis, and in some cases there remains no evidence of any permanent injury from that disease. An endocardial murmur sometimes disappears. Ulcerative endocarditis is an occasional event, giving rise to fatal pyæmia.

The liability to embolism is to be mentioned. Emboli may be derived from the left side of the heart, consisting of fibrinous masses formed in connection with endocarditis, or a clot may form in the right side of the heart and cause plugging of the pulmonary artery. Sudden death during the course of acute rheumatism is sometimes caused by pulmonary embolism. An example has fallen under my observation.

Acute articular rheumatism seldom ends in the chronic form of the affection.

TREATMENT.—There are few diseases in the nosology the treatment of which, during the last half century, has undergone such mutations as that of acute articular rheumatism. Among the measures more or less in vogue during the period just named are the following: Bleeding, general and local; mercurialization; colchicum carried to the extent of producing vomiting and purging; nitrate of potassa, an ounce or more given daily; opium in large doses; large doses of quinia; and the use of the veratrum viride. Cases in which reliance has been chiefly placed on each of these measures have pursued a favorable course and the treatment has seemed to be successful. Want of knowledge of the natural history of the disease—that is, the history based on the observation of cases in which the disease has been allowed to pursue its course under favorable hygienic circumstances uninfluenced by therapeutical interference—has heretofore rendered it difficult to judge of the effect of different methods of treatment. My report of 13 cases, and the report by Dr. Sutton of a larger number, treated with only palliative remedies, show an intrinsic tendency in the disease to end within a shorter period and with a less degree of liability to complications than have been hitherto generally supposed. Hence it may be reasonably concluded that the influence of different methods of treatment over the duration of the disease and the occurrence of complications has been much over-estimated. Each of the measures above enumerated probably is, to a certain extent, useful in certain cases; but a special controlling influence over the disease is exerted by none, and clinical observation has shown the development of endocarditis, pericarditis, and other complications under treatment by each of them. It is not worth while, therefore, to discuss their merits and demerits severally, inasmuch as few if any practitioners now rely upon any one of them.

Before the introduction of the salicylate treatment the practitioners of this country generally relied upon the alkaline treatment, first systematically employed by Wright in 1847, and afterward, on a larger scale, by Garrod and Fuller. The bicarbonate of potassa, the bicarbonate of soda, the tartrate of potassa and soda, and the citrate of potassa are the remedies usually employed. Garrod's report of 50 cases treated with alkalies, largely given, showed an average duration of six or seven days; and in no case after the patient had been under the influence of this treatment for forty-eight hours was any affection of the heart developed. It would, however, be an erroneous inference that cardiac complications never occur after the patient is fairly under the influence of this treatment. I have notes of several cases in which pericarditis was developed when alkalies had been freely given, the urine having been rendered alkaline; but that the liability to these complications is lessened by this treatment must be admitted. Moreover, this treatment seems to exert some influence in abridging the duration of the disease. Fuller claimed for the alkaline treatment, efficiently pursued, the power of lessening the febrile movement, relieving the arthritic inflammation, abridging the duration of the disease, and preventing endocarditis and pericarditis. With reference to the latter, which is manifestly the great object of treatment, he stated in 1862 that his experience embraced, up to that time, 194 cases, and that "in no single instance has any heart affection occurred after the patient has been under treatment for twenty-four hours." The analysis by Dickinson of cases treated with alkalies and of cases treated by different remedies affords conclusive evidence of the usefulness of the alkaline treatment as regards diminishing the liability to cardiac complications. His statistics show that a "partial alkaline treatment" is useless. To be effectual the alkalies must be given sufficiently to render the urine alkaline.[1]

To secure as quickly as possible protection against cardiac affections the alkali selected should be given at once in large doses and repeated at short intervals until the urine gives an alkaline reaction. One or two drachms of the bicarbonate of either potassa or soda may be given every three or four hours. To render it more grateful to the palate and stomach, citric acid or lemon-juice may be added to each dose, which is to be taken during effervescence. This quantity is generally well borne, and the urine, as a rule, is rendered alkaline in twenty-four hours. I have given, without inconvenience to the patient, the dose just stated every two hours, and produced alkalinity of the urine in twelve hours. The quantity of the alkaline remedy should be much diminished as soon as the urine is alkaline, the doses afterward being repeated only once or twice in the twenty-four hours. To secure more diuretic effect than is produced by the alkaline remedy alone, the acetate, nitrate, or bitartrate of potassa, the acetate of ammonia, or small doses of colchicum may be added. The tartrate of potassa and soda, which is much used as an alkaline remedy in this disease, is inferior to the bicarbonate of potassa or soda, because it must be given for a longer period before alkalinity of the urine is produced. It is well adapted to maintain an alkaline condition of the urine after this condition has been produced by the bicarbonate of potassa or soda.

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[1] Vide article by Dr. Wm. H. Dickinson in *Trans. Med. and Chirurg. Society*, 1862.

The remedy which is now chiefly relied upon in the treatment of acute rheumatism is salicylic acid or the salicylate of sodium. This drug, which was first introduced as a remedy for rheumatism in 1875, exerts over this disease a specific controlling influence analogous, in some but not in all features, to that of quinia over malarial fever. Salicylic acid may be given in the form of the powder enclosed in capsules in doses of 8 grains repeated every hour until ten or fifteen doses have been taken, or until ringing in the ears, the first symptom of salicyl intoxication, has been produced. Salicylate of sodium may be given in 10–20-grain doses repeated every hour until eight to twelve doses have been administered or ringing in the ears is produced. Another method is to administer at the beginning a single large dose of 60 to 80 grains of salicylate of sodium. This dose need be repeated only once or twice during the day. In general, the quantity of salicylate of sodium should not exceed 1½ to 2 drachms during the twenty-four hours.

The employment of salicylic acid or of salicylate of sodium in large doses is liable to lead to the symptoms of salicyl-poisoning. These symptoms are tinnitus aurium, nausea, vomiting, partial deafness, disturbed vision, vertigo, headache, shivering, rapid deep respiration, and delirium. The intensity of the toxic effects is generally in proportion to the amount of the drug which has been taken, but some persons have a special susceptibility to the action of this remedy and develop toxic symptoms after only moderate doses. As a rule, the unpleasant symptoms subside rapidly after the discontinuance of the remedy. A very few instances have been reported in which the fatal termination has been attributed to the action of salicylic acid, particularly upon the heart, but it is not certain that this has been the correct explanation.

In consequence of the unpleasant symptoms sometimes attending the use of salicylic acid or its sodium salt, it has been proposed to substitute for these salicin and the oil of wintergreen. Salicin is undoubtedly a beneficial remedy in acute rheumatism, but it has not been proven that it exerts so powerful an influence over the disease as salicylic acid. Maclagan, who introduced the salicin treatment of rheumatism, administers 20 to 40 grains, at first hourly, and then every two hours as the symptoms begin to improve. He continues the drug in scruple doses three to four times daily for a week or more. The treatment with large doses is to be resumed whenever pyrexia and acute arthritic symptoms return.

The oil of wintergreen, which is essentially a methyl-salicylate, has been recommended by Dr. F. P. Kinnicutt as an efficient substitute for salicylic acid and as devoid of its toxic properties.[1] Kinnicutt gives 10 to 15 minims of the oil every two hours until eight doses have been taken. The oil may be taken in milk or in capsules.

The efficacy of the salicylate treatment of rheumatism is most apparent in acute cases with considerable fever and severe pain. In such cases, within twelve hours after the beginning of the treatment the fever and the acute arthritic symptoms often subside. The cases of acute rheumatism are exceptional in which some relief is not obtained by the employment of the salicyl compounds, but there are cases in which this treatment proves ineffectual, and recourse must be had to other measures.

In two respects the expectations held of the advantages of this treatment have met with disappointment. The salicylate treatment does not prevent relapses. Even the continuance of moderate doses of salicylic acid or its substitutes after the subsidence of an acute attack does not prevent the occurrence of other attacks. For this reason, while most writers recommend the continuance of moderate doses after the cessation of the acute symptoms, there are some who withhold the drug until the first symptoms of a renewed attack make their appearance. By the latter method, it is claimed, the patient does not acquire repugnance for the drug, unpleasant symptoms are avoided, and the results obtained are equally favorable with those of the continued administration of the remedy.

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[1] *New York Medical Record*, Nov. 4, 1882.

The salicylate treatment does not prevent the heart complications of rheumatism, except in so far as it shortens the duration of the disease. For this reason this treatment should not supersede the full alkaline treatment. In order to prevent these complications, alkalies are to be given not less promptly and freely than they were before the introduction of the salicylate treatment. The best results are obtained by the combination of the salicylate and the alkaline treatment.

Antipyrine is an efficacious remedy in many cases of acute rheumatism. By the administration of 15-grain doses hourly the pyrexia and acute arthritic symptoms have been observed to subside in a manner similar to that after the use of salicylic acid. Not more than two drachms should be given during twenty-four hours. Neither heart complications nor relapses are prevented by antipyrine.

Intense hyperpyrexia claims more prompt and efficient treatment than by salicin or salicylic acid. The antipyretic measures employed in other fevers are indicated—namely, large doses of quinia or of antipyrine, and cold sponging, the cold bath, or the wet sheet. The arthritic affections do not contraindicate these measures. The following case is cited in illustration of their efficacy: A young woman was attacked with acute rheumatism in the seventh month of pregnancy. A mitral systolic murmur denoted endocarditis. The axillary temperature rose to 109° F. She miscarried, the labor being brief and easy. She was treated with quinia in large doses, alkalies, and sponging the whole body with cold water. The latter was employed constantly during the hyperpyrexia. These measures proved efficient and the patient recovered.

The palliative treatment embraces opiates or other anodynes for the relief of pain, restlessness, and inability to sleep. Thirst may be relieved by the free use of water or lemonade. Lemon-juice given in large quantity is one of the remedies for this disease in vogue within late years. It is, in fact, an alkaline remedy, but taken in sufficient quantity to render the urine alkaline the stomach often rebels against it, and the alkaline carbonates are to be preferred.

The affected joints claim local measures of treatment. The application of cloths saturated with an alkaline and opiate solution, as recommended by Fuller, is useful. Fuller's solution consists of from 4 to 6 drachms of the carbonate of soda or potassa, dissolved in a mixture of an ounce of Battley's solution (for which laudanum may be substituted), 2 ounces of glycerin, and 9 ounces of rose-water. The soap-and-opium liniment is a good local application. A liniment containing chloroform has been found useful. I have found the tincture of aconite applied to the parts to afford marked relief. Some practitioners prefer to envelop the joints with dry flannel or cotton. In the choice of local applications the sensations of the patient may serve as a guide. Extension of the limbs by means of an apparatus for that purpose, or simply a cord, pulley, and weight, in order to diminish the pressure of the articulated surfaces upon each other, affords marked relief. The application of pasteboard splints lined with cotton and confined by a firm bandage has been found not only to relieve pain, but to promote the disappearance of the inflammation. Any measure which has the latter effect lessens the pyrexia, for, assuming the disease to be an essential fever, the pyrexia is in part symptomatic of the arthritis. Cold applications to the affected joints, and even the ice-bag, are recommended by German authors. Much relief is afforded in some cases by what may be termed methodic friction. Using some lubricating embrocation, the friction at first over the affected joints, if they be extremely tender, must be as light as possible to avoid giving pain. The force of the friction, however, may be gradually increased, until at length the patient is sometimes able to bear without suffering as much pressure as can conveniently be made. Methodic friction, gradually increased up to as much pressure with the hands as can be borne without pain, leaves the affected joints more comfortable for several hours, and the operation may be repeated once or twice daily.

It is important, in order to avoid relapses, that the patient should not leave the bed and should not make unnecessary exertion until some time—if possible a week—has elapsed after all acute symptoms have subsided.

The treatment of rheumatic endocarditis and pericarditis has been already considered. Pleuritis and pneumonitis are to be treated according to the indications in individual cases, as when they occur in other pathological connections. The indications pertaining to active delirium, convulsions, and coma are derived from the pathological conditions with which these events may be associated—namely, hyperpyrexia, cerebral meningitis, or uræmia.

DaCosta has reported 30 cases treated with the bromide of ammonium in doses of 15 or 20 grains every three hours. Of these cases, in 23 there was no cardiac complication when this treatment was begun, and in none of the 23 cases was any cardiac complication subsequently developed. The mean duration of the disease was 22.5 days.[1] Senator has made some observations on the treatment with benzoic acid. Giving 2½ to 3 drachms of the acid, or from 3 to nearly 4 drachms of the benzoate of soda, during the twenty-four hours, the disease was apparently shortened. This result I have observed in a few cases. Its efficacy is vastly inferior to that of salicin and salicylic acid. Trimethylamine, employed largely in this disease by Arvenarius of St. Petersburg a quarter of a century ago, has more recently been employed with success by Lagrange and Dujardin-Beaumetz in France.[2]

Subacute and Chronic Articular Rheumatism.

The terms subacute and chronic rheumatism are applied not alone to cases having the distinctive features of the disease just considered, save acuteness, but to affections resembling articular rheumatism only in their situations, and also to persistent pains in different parts of the body supposed to involve a rheumatic diathesis. I shall notice under this heading true articular rheumatism in so mild a form as to be distinguished as subacute, and the chronic form, involving, as there is reason to suppose, a constitutional condition, if not identical with, allied to, that in the acute disease. The affection called rheumatoid arthritis will be noticed after gout has been considered.

Subacute rheumatism may have all the characters of the acute disease, minus intensity. It is then the same disease in a mild form. There is a liability to the cardiac complications, although it is much less than when the disease is acute. The treatment is the same, with modifications which are suggested by the want of acuteness of the symptoms. The alkaline remedies are indicated as protective against cardiac complications.

Both acute and subacute articular rheumatism may end in a chronic arthritic disease. The essential constitutional affection in these cases may not continue, but there remains an affection of one or more of the joints previously involved. The arthritic affection is perhaps a sequel of the rheumatic disease, rather than the latter persisting in a chronic form.

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[1] Vide *Pennsylvania Hospital Reports*, 1869, vol. ii.

[2] Vide *National Dispensatory*, 1879.

In most cases of *chronic rheumatism* the disease is subacute from the first. It depends doubtless upon an internal determining cause. If it cannot be affirmed that this cause is identical with that in acute articular rheumatism, a relationship between the two diseases may fairly be inferred.

Cases of chronic rheumatism are frequently met with in medical practice. The patients are generally in middle or advanced life. A greater or less number of joints may be affected. The affection remains fixed in certain joints; that is, it does not shift from joint to joint, as in cases of acute rheumatism. The affected joints are tender, painful to the touch, sometimes swollen from intra-articular effusion; their temperature is more or less raised, and the surface may be reddened. Motion is painful, especially at first after periods of rest. A grating sensation and sound often accompany the first movements after these periods. The local symptoms vary much in degree at different times, and patients often observe a connection between an increase of these symptoms and certain atmospherical conditions. They often say that their sensations enable them not only to judge of existing conditions, especially as regards moisture, but to foretell changes which are soon to take place.

Pyrexia is wanting except when there is an exacerbation as regards the local symptoms. The digestive and nutritive functions may be but little or not at all affected. There is no liability to cardiac complications or to disease of internal organs other than the heart.

The DIAGNOSIS involves discrimination from rheumatoid arthritis, which will be considered in the next chapter. In the latter occur anatomical changes, dislocations, and distortions which do not belong to the history of chronic rheumatism. Subacute or chronic synovitis, not rheumatic, is to be excluded. The points to be considered in differentiating the latter from rheumatoid arthritis are those which have been stated in connection with the diagnosis of acute articular rheumatism. Whenever chronic rheumatism has been preceded by the acute disease, this fact has of course an important bearing on the diagnosis.

Chronic rheumatism is devoid of immediate danger, but it persists in many cases indefinitely, and in not a few instances during life. Ankylosis of the affected joints sometimes takes place. Notable atrophy of muscles may be a result of their inaction when the disease interferes greatly with muscular movements, or possibly the result of some nervous disorder.

In cases of chronic rheumatism the TREATMENT so important in the acute disease is less effective. Alkalies are of comparatively little service. Salicin or salicylic acid has little controlling influence. A great variety of remedies with reference to a constitutional effect have had advocates. Observing proper care not to do harm by medication, trial should be made of different remedies. Of these may be mentioned the preparations of guaiacum—namely, the simple tincture, the ammoniated tincture, the mixture or the compound decoction of sarsaparilla into which guaiacum enters—the bichloride of mercury, iodide of potassium in large doses, colchicum in small or moderate doses, the hydrochlorate of ammonia, sulphur, and arsenic.

The local treatment is important. Benefit may be obtained from flying blisters or the application of the tincture of iodine. Methodic friction or shampooing, as described in connection with the acute form of the disease, and stimulating embrocations are often of much utility. Counter-irritation by "firing" has been recommended. Palliation of pain may frequently be procured by the application of the tincture of aconite or the veratria ointment. The treatment has reference to two objects—namely, the removal of, *first*, the inflammatory condition, and *second*, the effects of the inflammation. In order to prevent stiffness and ankylosis passive motion is advisable so soon as the inflammatory condition subsides.

Rendering the affected joints immovable by means of pasteboard splints and bandaging should be tried. The immobility, however, should be continued for only a few days at a time, and in the intervals passive movements may be important to prevent ankylosis. Local douches with either cold or warm water, or with each in alternation, may prove of service. Electricity, especially the constant current, has been found serviceable.

External measures acting upon the entire surface are perhaps, in certain cases, not less useful than internal remedies. Reference is had to the simple tepid bath, the alkaline bath, the Turkish bath, the sulphur vapor-bath, and hydropathic packing. The resources of treatment should not be considered as exhausted without a fair trial of these measures.

Measures having reference to improvement of the general health and the invigoration of the system form an essential part of the treatment, embracing tonic remedies, with attention to diet, clothing, exercise, etc. It is unnecessary to go into details which are essentially the same in the treatment of many chronic affections. Change from a cold, changeable, and humid climate to one mild, uniform, and dry often proves the most effective measure which can be adopted. The mineral springs are frequently resorted to with advantage by persons suffering from chronic rheumatism. Of the different waters, the alkaline, sulphurous, saline, and chalybeate are each useful in certain cases. Doubtless, however, much of the benefit derived from watering-places is due to the various incidental hygienic influences.

CHAPTER XI.

GOUT.—RHEUMATOID ARTHRITIS.

Gout: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Treatment.—Rheumatoid Arthritis.

Gout.

THE disease called gout has been thought to be allied to articular rheumatism, and the two affections have even been considered to be identical. As will be seen, however, they are shown to be essentially distinct forms of disease by points of contrast pertaining to morbid anatomy, clinical history, pathological character, and causation. The name gout or its synonym in different languages is universally applied to the disease to be now considered. The name is derived from *gutta*, a drop, and is supposed to refer to the dropping of some morbid fluid into the joints.

Gout occurs as an acute, a subacute, and a chronic affection. Writers have made other varieties, such as irregular and misplaced gout. As the subacute form is not always chronic, it is convenient to consider this form when of brief duration and the acute form under one head. The term *transient gout* will embrace the acute and subacute form, the latter ending too soon to be considered as chronic. *Persistent* or *chronic gout* will embrace all cases in which the disease is protracted.

Anatomical Characters.—The most distinctive anatomical character of gout consists in the deposit within the tissues of crystalline urates, chiefly urate of sodium. Mingled with the urate of sodium have been found in small quantity other salts, as urate of calcium and of magnesium and carbonate and phosphate of calcium. The urate of sodium is in the form of acicular crystals, with which may be mingled amorphous granular matter. With few exceptions the urates are deposited first in the joints, and in the majority of cases first in the metatarso-phalangeal articulation of one of the great toes. In the course of the disease other joints become affected. At first the disease manifests a preference for the small joints of the foot and hand, but subsequently (and in rare cases primarily) larger joints, as the knee and elbow, may become involved. The urate deposit seems to form a white, chalk-like incrustation upon the free surfaces of the articular cartilages. Careful examination, however, shows that the deposit is in the substance of the cartilage. Usually only the superficial layers of the cartilage contain the deposit. Upon microscopical examination are found acicular crystals arranged in little clusters which radiate from a centre. The centre of a cluster is often a cartilage-cell, but the crystals are deposited in the basement-substance as well. The deposition of urates in the joints may occur either with or without paroxysms of articular gout. Garrod has shown that a single paroxysm of gout suffices to cause a certain amount of the urate deposit in the affected joint.

Urate concretions are often found in the synovial membranes and the articular ligaments. These gouty concretions are called *tophi* or *chalk-stones*. Tophi consist of needle-shaped crystals of urate of sodium, mingled with granular matter and animal substance. Their consistence is usually compared to that of mortar.

Although the urate deposit is often associated with acute transitory inflammation in and about the joints, it is sometimes though less frequently combined with destructive inflammatory and degenerative changes in the joints. The stiffness of the joints observed during life is due more to the affection of the ligaments than to the alterations of the cartilage. Fibrous ankylosis may occur. There may be erosion of the articular cartilages. These changes, combined with tophaceous nodules, and sometimes subluxation of the joints, may give rise in chronic gout to striking deformities, which are observed most frequently in the hands.

The deposits of urates may be found not only in the ligaments and cartilages, but also in various other situations, of which the most frequently affected are the perichondrium, the periosteum, the periarticular connective tissue, the tendons and their sheaths, the bursal sacs, the kidneys, the skin, and the cartilages of the external ear. Tophaceous masses occurring in the external ear, varying in size from that of a pin's head to that of a split pea, are often of great assistance in diagnosis. Among the rare situations for gouty deposits may be mentioned the spongy texture of bone, the small laryngeal cartilages, the tarsal cartilage, and the sclerotic coat of the eye, the outer sheaths of vessels and of nerves, the endocardium, and the dura mater. In general, those tissues are most liable to be affected which are possessed of little vascularity, but the gouty deposit has been found in such vascular structures as the skin and the muscles. Tophi, especially those seated subcutaneously, may lead to the formation of abscesses, which burst and discharge pus mingled with chalky material. In this way or by simple necrosis of the skin over the tophi sluggish ulcers are formed which have little tendency to repair.

The interesting discovery has been made by Ebstein that the tissues which are the seat of urate deposits in gout are in the condition of coagulation necrosis. According to Ebstein, this coagulation necrosis precedes the deposition of urates, and is caused by the abnormal chemical constitution of the blood. The fixation of urates in the tissues, according to this view, is due to the acidity of the necrotic tissues, leading to the formation of the insoluble acid urates out of the neutral urates, which are in excess in the circulating blood. An area of reactive inflammation exists around the foci of coagulation necrosis.

Todd and Garrod have established the fact that gout is one of the causes of the small granular kidney. The diseased kidney presents the gross and the microscopical changes which have already been described as characteristic of this form of chronic Bright's disease (p. 878). The gouty kidney is the seat of urate deposits, chiefly in the pyramids, where they appear as whitish streaks and points. The morbid deposit is found both within and between the tubes. The gouty kidney may develop without the occurrence at any time of articular symptoms, although at the autopsy urates are found in the articular cartilages. Gout is an occasional cause of waxy degeneration. Concretions of urate of sodium have been found in the renal calices and pelvis.

It has been shown by Garrod that the blood in gout contains an excess of uric acid, which is present in the form of urate of sodium. According to this author, no appreciable excess of uric acid is present in the intervals between the early attacks of gout, but in chronic gout the blood is continuously rich in uric acid. The accumulation of uric acid in the blood constitutes the condition called *uricæmia* or *lithæmia*, which has been considered in Part I. of this work.

The gouty dyscrasia is frequently associated with various anatomical changes other than those which have been described. These lesions, however, are generally without special peculiarities by which they can be recognized as gouty in origin.

Patients with gout are especially liable to chronic inflammations, of which the most common are chronic gastritis, chronic enteritis, chronic bronchitis, and chronic endarteritis (atheroma). Fatty liver and, according to some authors, cirrhosis of the liver may be an effect of gout. Various cutaneous diseases, especially eczema, are ascribed to gout. Cardiac lesions are common in chronic gout. They are most frequently secondary to chronic nephritis, but fatty heart and chronic endocarditis and myocarditis may occur in gout without this association. Gouty patients have a tendency to the formation of renal calculus and to chronic inflammations of the bladder, urethra, and prostate gland. Gout and diabetes mellitus are sometimes combined, or one may be a sequel of the other.

CLINICAL HISTORY.—Transient gout and persistent gout require separate consideration as regards their clinical history. Having considered these forms of the disease, I shall notice irregular and misplaced gout.

Transient Gout.—Under this head are to be embraced all cases in which the disease is of short duration; that is, the duration not sufficiently prolonged for the disease to be regarded as chronic. In this form of the disease the patient is said to have an attack or fit of gout. The disease is generally acute, but sometimes it is subacute.

The seizure is generally sudden, occurring, as a rule, during the night, and in the majority of cases after midnight. In a certain proportion of cases the seizure occurs without premonitions; and the patient may have retired to bed feeling as well as usual. In some cases, however, there are prodromic phenomena consisting of cardialgia, gastric flatulency, eructations, and other dyspeptic ailments, together with inability to exert the mental faculties, irritability of mind, and depression of spirits. These phenomena are not sufficiently significant to lead to the expectation of an attack of gout if one or more attacks have not been already experienced, but in persons who are subjects of the disease they may denote an impending seizure. Observations have shown that for several days preceding a gouty paroxysm there is a notable diminution of uric acid in the urine.

The seizure is marked by pain, which in the great majority of cases is seated in the metatarso-phalangeal joint of one of the great toes. The pain soon becomes more or less intense, and frequently it is excruciating, being compared by patients to pain caused by the gnawing of an animal, a dislocation of the bone, a nail driven into the joint, tearing of the parts with pincers, etc. Such comparisons express only intensity of suffering, for it is not to be supposed that patients have actually experienced the pain produced by the various causes mentioned. A sense of throbbing in the part accompanies the pain. More or less pyrexia is developed, preceded by shivering. These symptoms continue for several hours, and then the pain and fever subside. The patient is comparatively comfortable and obtains some sleep. The relief is usually accompanied by slight perspiration. In the mean time, the affected joint becomes swollen, the skin is reddened and shining, the subcutaneous veins are distended, the heat is raised, and there is exquisite tenderness to the touch. These appearances are observed after the severe pain has abated.

In some cases only a single paroxysm of severe pain is experienced, but much oftener the seizure is renewed on the following night, and on successive nights for a variable period. The local affection may remain in the joint affected at the first seizure, or the affection may be transferred from this joint to the corresponding joint of the opposite foot. In some cases the inner side of the foot, the instep, or the heel becomes affected, and the affection may extend to the larger joints and to the small joints of the upper extremities. This extension of the local manifestations of the disease rarely occurs until after several attacks, and it occurs especially in cases of chronic gout. After recovery from transient gout there is often a feeling of better general health than before its occurrence, the patient being relieved of indefinite ailments which previously existed.

During the continuance of transient gout the symptoms other than those referable to the part or parts affected vary considerably in different cases. Pyrexia is more or less marked in proportion to the acuteness of the local manifestations. The fever appears to be symptomatic, and, as a rule, it is less than in cases of acute articular rheumatism. The appetite is frequently impaired or lost, but in some cases it is preserved. Dyspeptic ailments are sometimes present and sometimes absent. The urine is generally scanty, high-colored, and deposits a lateritious sediment on cooling. The excretion of uric acid is lessened. The bowels are generally constipated.

Cases also differ much as regards the local symptoms. The affection may be called acute when the pain is severe, the swelling, redness, etc. are marked, these local symptoms being accompanied by a corresponding amount of fever. On the other hand, the affection is subacute when the pain is notably less severe, the other local symptoms are less marked, and fever is either slight or wanting. As regards the seizures of pain, which occur usually at night, they are in some cases paroxysmal, the pain passing off in the day, but in other cases more or less severity of pain continues during the day as well as at night; in other words, remissions occur instead of intermissions. The local symptoms gradually decline as the fit or attack passes off. Œdema of the part or parts is more or less marked at the decline of the local affection, with frequently pruritus, and in the majority of cases furfuraceous desquamation of the epidermis. The local symptoms just named are diagnostic of gout as contrasted with rheumatism. Cramp affecting the muscles of the leg, the thigh, and other parts of the body is a symptom more or less marked in a considerable number of cases.

The duration of transient gout varies between a few days and several weeks. In the majority of cases the paroxysms recur nightly for at least a week. Generally, the paroxysms or exacerbations are less and less severe in proportion to the duration of the attack. If the disease continue for more than three or four weeks it is to be considered as persistent or chronic. An attack of acute or subacute gout, especially the latter, may eventuate in the persistent or chronic form of the disease, or, having continued for a variable period, the disease for the time ceases, recurring at longer or shorter intervals in the vast majority of cases.

Persistent or Chronic Gout.—Gout, beginning as either an acute or subacute affection, in a certain proportion of cases continues as a chronic affection for many weeks, months, and even years. Generally, this persisting or chronic form of the disease is preceded by the occurrence of many transient attacks recurring after intervals which become shorter and shorter, until at length, as it were, they run together. When the disease is persistent it has not continuously a uniform severity, but remissions occur from time to time, and not infrequently intercurrent acute attacks take place.

In the persistent or chronic form the local symptoms which characterize the acute affection—namely, intense pain, heat, and redness—are either slight or wanting. There is little or no fever. Disorders of the digestive system are often present and the general health is more or less impaired. In this form the joints are liable to become stiffened, ankylosed, and deformed from the abundance of the gouty deposit. Chalk-like concretions may occur within and around the small joints of the hands. In some cases collections of semi-solid matter are felt near the surface, and if they be opened the matter may be pressed out. Sometimes collections of considerable size open spontaneously. The solid concretions, or chalk-stones, sometimes make their appearance through the skin. The gouty matter may be deposited in the bursæ mucosæ in the neighborhood of joints. In a certain proportion of cases—the number, happily, not large—the hands and feet become permanently distorted and crippled, the fingers sometimes presenting an appearance compared by Sydenham to that of a bunch of parsnips. Nodules of the deposit are frequently seen on the helix of the ear, sometimes also on the eyelids, and occasionally on the face.

In chronic as in acute gout the predilection of the disease, as regards the seat of the local manifestation, is for the joints of the great toe. An affection of other joints occurs primarily in a small proportion of cases. After the great toes, the order in which parts are liable to become implicated, according to Garrod, is as follows: The heels and ankles, the knees, the smaller articulations of the hands, the elbows, and lastly the shoulders and hips. There are, however, many exceptions to this order of sequence. A blow, sprain, or other injury may determine the seat of the local affection. A point of difference in different cases pertains to the number of joints which become affected.

In some cases the local manifestations of the disease, however long it may persist or frequently recur, remain concentrated on a few joints; in other cases a large number of joints are implicated; and, again, in some cases different joints are affected in successive attacks. If the same and a few joints be always affected, and if the disease either persist or recur frequently, permanent changes in the affected joints are more likely to occur. Another point of difference in different cases relates to the palpable changes in the affected joints. Some gouty patients escape ankylosis and nodosities, although they suffer from the disease more or less throughout their lives, whereas in other cases the hands and feet become deformed and crippled after a comparatively short duration of the disease.

Irregular and Misplaced Gout.—It is an old doctrine that local manifestations of gout are liable to be seated in the different visceral organs—namely, the stomach, intestines, lungs, heart, liver, kidneys, and brain. Disorders referable to these viscera preceding the ordinary manifestations of gout have been considered as gouty, and it has been supposed that gouty disorders of viscera may occur without any affections of the joints either accompanying or ensuing. In such cases gout is said to be *irregular* and *misplaced*. *Anomalous* and *latent gout* are other terms applied to these cases. Sometimes during an attack of gout the affection of the joint or joints suddenly disappears, and symptoms denoting an affection of some internal organ supervene. In these cases a transference or metastasis of the gouty manifestations is supposed to take place, and the name *retrocedent gout* is employed to express such an occurrence. These notions are probably, in a measure, well founded, but it is difficult with our present knowledge to say how far they are to be accepted as correct. The difficulty consists in the inability to determine whether antecedent, coexistent, or consecutive affections in certain cases of gout are not associated merely by coincidence. It has doubtless been too much the custom to consider all affections occurring in persons subject to gout as of a gouty character, and the gouty diathesis is often considered to exist on insufficient data. Making, however, due allowance for error, it can scarcely be doubted that the constitutional morbid condition which constitutes the disease in cases of gout may determine disorders, sometimes of a grave character, in organs more important to life than those in which the local manifestations are usually seated. Evidence of a pathological connection of these disorders with gout is afforded by complete relief following the development of a gouty affection of the joints, by their occurrence immediately after a gouty affection of the joints has been apparently suppressed, by the absence of any other appreciable causes explaining their occurrence, and by the indefinite or anomalous character of the disorders.

Disorders of the stomach and intestine attributed to gout are neuralgic pain or spasm, flatulent dyspepsia, and persistent vomiting, accompanied with more or less prostration, diarrhœa, and in some cases symptoms denoting enteritis.

Disturbed action of the heart is not uncommon in gouty patients. It sometimes occurs in an alarming degree, but it may be doubted if a purely functional disturbance occurring in this connection ever prove fatal. Valvular lesions and enlargement of the heart often exist in persons affected with chronic gout, being usually associated with chronic Bright's disease. The want of a tendency to acute endocarditis and pericarditis is one of the points distinguishing gout from rheumatism.

Cough, asthmatic dyspnœa, and bronchitis are attributed to gout. Spasmodic affections of muscles or cramp in various situations, neuralgia affecting the branches of the fifth pair, the sciatic nerve, and other nervous trunks, paroxysmal headache and hysteria, are to be added to the list.

Delirium and fatal coma are among the disorders imputed to gout. These symptoms may be due to uræmia or to meningitis, which may or may not be dependent on gout.

The deposit of the urate of sodium in the uriniferous tubes in cases of gout has been stated under the head of the morbid anatomy. It is not difficult to understand that this deposit may accumulate and form renal calculi. Clinical observation shows that gouty persons are liable to the formation of renal calculi and to paroxysms of pain caused by the passage of the stones along the ureter, or renal colic. It has been established that the contracted kidney may be due to gout. The coexistence of renal disease in certain cases may account for the occurrence of some events which have been heretofore considered as being specially connected with gout—namely, vomiting and purging, headache, neuralgia in different situations, delirium, convulsions, and coma. The existence of renal disease is to be ascertained by means of the urinary changes which have been already considered. The urine presents the characters distinctive of the small granular kidneys. It usually is clear and abundant, and it deposits but little sediment. The amount of albumen in the urine may be slight, but in some cases it is considerable. Casts are usually not abundant, and they may be absent. Slight albuminuria in the course of gout is not necessarily indicative of Bright's disease.

Cystitis and urethritis have been attributed to gout, but the existence of any special pathological connection may be doubted. Certain affections of the skin, particularly eczema, are common in persons subject to gout. Graves called attention to the habit of grinding the teeth as peculiar to cases of gout. The habit proceeds from an uneasy sensation in the teeth, which is in this way momentarily relieved.

A small amount of sugar may be found occasionally in the urine of gouty patients. The association of gout and of diabetes mellitus in some cases has been already referred to. This association has led to the idea that the vices of nutrition which are the causes of these diseases are in a measure allied, but we are wholly in the dark as to the fundamental disorders of nutrition in both of these diseases.

PATHOLOGICAL CHARACTER.—The researches of Garrod show that the pathology of this disease involves a particular morbid condition of the blood—namely, an abnormal accumulation of uric acid, *uricæmia* (*lithæmia*).[1] The blood in health contains only a trace of uric acid. In 47 cases of gout an analysis of the blood-serum by Garrod showed a notable increase of this constituent. Garrod has also shown that during an attack of acute gout the uric acid in the urine is notably diminished. It is apparently increased, since a deposit of the urates in more or less abundance when the urine cools is common; but the increase is only apparent, the deposit being due to the scanty quantity of urine. In chronic gout the quantity of uric acid excreted by the kidneys is habitually below the average in health. Garrod has found the uric acid in excess in the blood in cases of lead-poisoning.

From the facts just stated, taken in connection with the deposit of the urate of sodium within or around the joints, it is rationally concluded that the local manifestations of gout are effects of the uricæmia; but it is evident that the uricæmia is itself an effect, and the primary perversions underlie the excess of uric acid in the blood. Either this principle is produced in too great abundance, or, without being unduly produced, it accumulates as a result of insufficient excretion by the kidneys. Probably both explanations are involved. Direct observation, at all events, shows deficient excretion, since the amount in the urine is less than in health. Garrod entertains the opinion that the deposit of uric acid in the form of the urates takes place within and around the joints in consequence of diminished alkalinity of the blood; whereas Ebstein considers the primary effect of the abnormal condition of the blood to be the production of coagulation necrosis, which is followed by the deposit of urates.

In treating of rheumatism it has been stated that an examination of the blood in that disease with reference to the amount of uric acid shows this principle not to be in excess. Here is a capital point of difference between gout and rheumatism as regards pathological character. The clinical history of gout has afforded several striking points of contrast between the two diseases. These points will be reproduced under the head of Diagnosis. Other points of contrast will be presented under the head of Causation. The individuality of each disease is therefore sufficiently established.

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[1] Vide Part I. p. 75.

CAUSATION.—Gout is a diathetic disease, and the diathesis in a certain proportion of cases is congenital and inherited. It is a matter of common observation that certain families are predisposed to this disease, and it is sometimes transmitted successively through a series of generations. Scudamore's statistics showed that of 523 patients affected with gout, in 309 the disease had existed in either the parents or grandparents. Of 80 cases reported to a commission of the French Academy, in 34 the disease had been transmitted, and Garrod states that in the cases which he has observed an hereditary predisposition existed in 50 per cent. The diathesis, however, is by no means invariably inherited; in a certain proportion of cases it is acquired; and on the other hand the disease is not always transmitted to offspring. There are causes, then, which may produce it in those in whom there is no reason to suppose a congenital predisposition, and in those predisposed by inheritance other causes may be required for its development.

A causative influence relates to age. It occurs very rarely before the age of puberty. Of 515 cases analyzed by Scudamore with reference to age, in only 5 did the disease occur in persons less than eighteen; in 142 cases the ages at the time of the development or first occurrence of the disease were between twenty and thirty years; in 194 cases, between thirty and forty; in 118 cases, between forty and fifty; in 38, between fifty and sixty; and in only 10 cases between sixty and sixty-six years. The liability thus diminishes progressively after fifty, and the development of the disease is as rare, if not more so, after seventy as before puberty. In the cases in which it appears before the age of twenty-five it generally is inherited. In the causative influence relating to age there is a point of contrast to rheumatism, the latter disease affecting by preference young subjects.

A very marked difference exists between the two sexes in the liability to gout. Gout—at least articular gout—is much more common in men than in women. Here is another point of difference from rheumatism, the latter occurring scarcely less frequently in females than in males.

Dietetic and regiminal habits lead to the acquirement of the diathesis, and co-operate with a congenital predisposition in producing the disease. To the habitual use of wine or malt liquors the disease is in many cases chiefly attributable, and, on the other hand, the use of distilled spirits seems to exert no influence in its production. Gout prevails in countries in which wine and malt liquors are largely used, whereas in countries in which the use of spirits predominates the disease is relatively rare. It is comparatively rare in Scotland, Russia, Poland, Denmark, and in this country, probably in a great measure for the reason just stated. It is proverbially prevalent among those of the opulent class of society who are addicted to luxurious habits; but doubtless the habitual indulgence in the pleasures of the table, exclusive of the use of fermented liquors, contributes largely in this class to the production of the disease. Physical indolence also enters into the causation. The agency of malt liquors alone is strikingly shown by the frequent occurrence of the disease among a class of laborers in London employed to raise ballast from the bottom of the Thames, who are accustomed to drink two or three gallons of porter daily. According to Budd, gout is quite common among this class. The lighter wines are less likely to produce the disease than the stronger varieties—namely, sherry, madeira, and port—and this is true of malt liquors. There is little reason to suppose that cider tends to produce it. It is evident from the facts just stated that the causative agent in fermented liquors is not exclusively alcohol. Ascribing to the use of wine and malt liquors, to high living in other respects, and to indulgence in luxurious ease a due causative influence, the disease is sometimes developed irrespective of these causes. Examples are occasionally met with among laborers accustomed to a plain diet and neither wine-drinkers nor beer-drinkers. My clinical records contain notes of several such cases. Gout developed in individuals of the poorer classes often pursues an atypical course, and the acute paroxysms may be absent.

Various causes may determine the occurrence of an attack of gout; that is, they may act as exciting causes. Attacks are sometimes attributable to prolonged intellectual exertion, anxiety of mind, bodily fatigue, exposure to cold, acts of excessive indulgence in eating or drinking, etc.; but in not a few instances the disease occurs without any appreciable exciting cause. The disease is rarer in tropical than in temperate climates. Attacks are more likely to occur in the spring and autumn than at other seasons of the year. Garrod adduces facts which show that impregnation of the system with lead is among the causative agencies. Of 51 male hospital patients, 16 were either painters, plumbers, or workers in lead. This connection has been noted by other observers. The fact that in cases of lead-poisoning the uric acid is found in excess in the blood has already been stated.

DIAGNOSIS.—The difficulties in the way of the diagnosis of articular gout relate to its discrimination from rheumatism and the affection called rheumatoid arthritis. The distinctive features of the latter will be presented in treating of that affection, and the differential diagnosis of gout and rheumatism only will be here considered. The points involved in this differential diagnosis embrace facts relating to anatomical characters, clinical history, pathological character, and causation which have been presented under these respective heads.

Distinctive Features relating to Anatomical Characters.—Under this head reference is had to the morbid changes within and around the affected joints. A distinctive anatomical change in gout is the deposit of a morbid product abounding in the urate of sodium, the gouty or tophaceous matter, either in collections of a semi-solid consistence or in hard concretions commonly called chalk-stones. These are never present in rheumatism and are pathognomonic of gout. The deposit, however, in a situation and in sufficient quantity to be ascertained during life occurs in only a certain proportion of cases, and chiefly in chronic gout.

Distinctive Features relating to Clinical History.—In the great majority of cases the primary local affection is seated in the metatarso-phalangeal joint of one of the great toes. The larger joints are affected subsequently, if at all. The local manifestations tend to the smaller joints of the toes and hands. In acute cases the pain is more intense than in acute rheumatism, and the tenderness is greater. Pain occurs in paroxysms or in marked exacerbations. Œdema, pruritus, desquamation of the cuticle, and enlargement of the veins are distinctive of gout. Febrile movement is less marked than in acute rheumatism, and is apparently symptomatic, whereas in rheumatism the fever is essential as well as symptomatic. Acute endocarditis and pericarditis are very rarely, if ever, developed in the course of the disease. The deposit of small collections of the urate of sodium on the helix of the ear is of frequent occurrence in cases of gout, and serves to establish the diagnosis. They should always be sought for.

Distinctive Features relating to Pathological Character.—The uric acid in the blood is morbidly increased. This may be ascertained by obtaining serum either from a small quantity of blood or by means of a small blister, and resorting to what Garrod calls the "uric-acid thread experiment." The mode of performing this experiment, as described by Garrod, is as follows: "Take one or two fluidrachms of the serum of the blood and put it into a flattened glass dish or capsule. To this add ordinary strong acetic acid in the proportion of six minims to each fluidrachm of serum, which causes the evolution of a few bubbles of gas. When the fluids are well mixed introduce one or two ultimate fibres, about an inch in length, from a piece of unwashed linen fabric, which should be depressed by means of a small rod, as a probe or the point of a pencil. The glass should then be set aside in a cool place until the serum is quite set and almost dry. Should uric acid be present in the serum in more than a small amount, it will crystallize, and during its crystallization will be attracted to the thread and assume forms not unlike those presented by sugar-candy upon a string. To observe this, the glass containing the dried serum should be placed under a linear magnifying power of about fifty or sixty, procured with an inch object-glass and low eye-piece; or a single lens of one-sixth of an inch focus answers perfectly. To ensure perfect success the glasses should be broad and flat; the acetic acid should be neither very strong nor too weak; the glass should not be disturbed during the drying of the serum; the temperature should be that of an ordinary sitting-room; and the glass should be protected from dust."[1]

For a diagnosis the thread experiment, according to Garrod, suffices. Crystals of uric acid do not form and adhere to the thread in the blood-serum in health. The uric acid of the blood is not morbidly increased in rheumatism.

Distinctive Features relating to Causation.—Gout occurs very rarely before puberty, and it occurs generally between thirty and fifty years of age. Rheumatism affects especially young subjects. Gout is oftener hereditary than rheumatism. It rarely affects women. The use of fermented liquors and habits of luxury as regards diet and exercise exert an agency in producing gout which is not exerted to the same extent in the causation of rheumatism. The poorer classes are as liable to rheumatism as are the rich, if not more so; the reverse being true of gout.

To determine the existence of the diathesis prior to the arthritic manifestations of the disease, and to recognize the irregular or misplaced manifestations of gout, are objects of diagnosis involving not a little difficulty and doubt. Certain symptoms should excite strong suspicion of the constitutional gouty condition if they occur within the periods of life most favorable for the development of the disease and in a person of a gouty family whose habits of life are such as will co-operate with an innate predisposition. Among the symptoms referred to are those which belong to dyspepsia, with a scanty secretion of urine which deposits the urates, and the liability to gravel or attacks of renal colic. If with these symptoms an examination of the blood-serum show a morbid excess of uric acid, the development of an attack of gout may be predicted with considerable confidence. The diagnosis of disorders of internal organs imputed to gout—or, to use a term which has been applied to these cases, of "visceral gout"—must rest mainly on their occurrence in proximity to attacks of gout, either preceding, accompanying, or following the sudden cessation of well-marked gouty manifestations.

PROGNOSIS.—The immediate danger to life from gout is slight. If it ever, *per se*, prove fatal, it is by its irregular or so-called misplaced manifestations; and in many, if not most, of the cases in which death is imputed to gouty affections of vital organs it is probable, as already intimated, that these affections do not strictly belong to gout, but are to be regarded as coexisting or intercurrent affections. The prognosis becomes grave if the small granular kidney be developed in the course of gout. It is an error to suppose that gout is salutary as regards the duration of life. Frequently-recurring attacks of transient gout and the persistence of the disease in a chronic form impair the constitutional powers, diminish the ability of the system to resist other diseases, and thus shorten life. It is a serious disease in certain chronic cases in which it leads to deformity and rigidity of the affected joints. It has been a favorite notion that the liability to gout lessens the liability to other diseases. There is little foundation for this notion, and, on the contrary, there is ground for the belief that if gout do not tend to produce other diseases it favors their inroads upon the system.

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[1] This account from Garrod's work has been somewhat condensed.

A tendency to recurrence is a law of the disease. It is extremely rare for a single attack only to occur. If life be not cut off by some other disease, frequent attacks are almost invariably experienced, and the disease may become chronic. The diathesis is thus rarely if ever extinguished.

TREATMENT.—Various therapeutical measures heretofore considered as having a curative influence over gout are now rarely employed. Their disuse has resulted from the clinical study of their effects and from improved pathological views. Among the measures referred to are embraced bloodletting, emetics, mercurialization, and continued or often-repeated purgation. These measures are now regarded as contraindicated in both the transient and the persistent form of gout. Other measures, long more or less in vogue, are still prized in the treatment of this disease. In the latter list are colchicum and alkaline remedies. The indications for treatment during attacks of transient gout, during the intervals between these attacks, and in cases of persistent or chronic gout respectively claim separate consideration.

Treatment during Attacks of Transient Gout.—The great Sydenham, himself for more than thirty years a sufferer from this disease, was led to conclude that an attack should not be interfered with. Regarding it as an effort of nature to get rid of a noxious material, he believed it to be undesirable to arrest or abridge it. The propriety of non-interference was also advocated by Trousseau. Few physicians, however, would feel satisfied to fold their hands and await the cessation of the disease, and still fewer patients would be content to forego measures to alleviate their sufferings. Nor is this line of conduct consistent with either reason or experience. Transient gout is a self-limited affection. It is probably rarely, if ever, arrested, and it might not be advisable to arrest it were this practicable. Perhaps its duration is not often abridged by therapeutical interference; but its severity may be lessened and the suffering connected with it may be materially mitigated by measures which, if judiciously employed, will not do harm. This is a mild expression of the resources of medicine in cases of transient gout.

Colchicum is an ancient remedy for gout, assuming the hermodactylus of the Grecian and Arabian physicians to have been this drug. It has been not less prized by the majority of physicians in modern times. Most if not all of the various secret nostrums for gout which have commended themselves to popular favor owe their efficacy chiefly to this remedy. Its special power of controlling the inflammatory phenomena of the disease is undoubted. Our knowledge of the value of this remedy is derived wholly from experience. Its special mode of operation cannot be explained. A rational object of treatment, deduced from the pathological character of gout, so far as it is at present known, is the elimination of uric acid; but the researches of Garrod show that this remedy does not increase the amount of uric acid excreted by the kidneys, nor does it in all cases increase the quantity of urine. It may be here added that other rational objects of treatment are prevention of an undue formation of uric acid or its undue accumulation in the blood, and neutralization of this acid, or keeping it in a state of solution in the blood until it is eliminated through some of the natural emunctories.

In an acute attack of gout, colchicum may be given at first in a tolerably full dose; that is, half a drachm or a drachm of the wine, or an equivalent dose of the tincture or acetic extract. It should afterward be continued in small doses. It is never judicious to carry its use to the extent of producing vomiting or purging. Carried to this extent, it is objectionable on account of its depressing influence, although relief of the gouty inflammation be procured. Its special influence is independent of its operation as an emetic or a cathartic. An emetic of any kind is not indicated unless the attack occur soon after a full meal and there be reason to suppose that the stomach is overloaded with undigested food. If constipation exist, a free evacuation by means of a saline purgative is desirable. A saline purgative is also appropriate as a depletory measure if the patient be robust or of a full habit and the fever be high. The purgation should be effected by salines rather than by the colchicum. Some physicians regard salicylic acid and salicylate of sodium as beneficial in acute attacks of gout; but the favorable influence of these drugs is far inferior in this disease to that exerted in rheumatism.

Alkaline remedies are rationally indicated with a view of promoting the solubility of uric acid. For this end the salts of potassium and of lithium are to be preferred to those of sodium, the urate of potassium and the urate of lithium being more soluble than the urate of sodium. Moreover, these salts have a diuretic action, and thus contribute to another rational object of treatment—namely, the elimination of uric acid by the kidneys. The bicarbonate of potassa may be selected, and the acetate and the bitartrate are appropriate. The alkaline remedy may be given in conjunction with the colchicum. The phosphate of ammonia was introduced as a remedy in gout some years since by Buckler of Baltimore, its efficacy being supposed to consist in its forming a soluble salt, readily eliminated, by the combination of uric acid and ammonia, the phosphoric acid combining with the soda and forming another soluble salt. Ten grains may be given three or four times daily. This remedy, however, is better suited to the chronic form of the disease or to the intervals between attacks of transient gout. This remark will also apply to the benzoic acid, which is supposed to be useful by converting the uric into hippuric acid.

The carbonate of lithia, introduced as a remedy by Garrod, has proved to be of much value.[1] The urate of lithia is highly soluble, and the remedy in small doses acts efficiently as a diuretic. It is a remedy which produces no unpleasant consequences, although continued for a considerable period. It may be given in five-grain doses three times daily. An efficient and agreeable mode of administration is to give it largely diluted in carbonated water. The citrate of lithia has the advantage of greater solubility in water than the carbonate.

During the paroxysms of pain opiates may be given moderately if they be well borne. If they occasion unpleasant effects, the succedanea of opium must be relied upon—namely, hyoscyamus, belladonna, and aconite. From its action upon the skin the Dover's powder is an eligible form of opiate. The inconveniences of opium given by the mouth may sometimes be avoided by the hypodermic method of administration.

As regards local treatment, the affected joints may be covered with cotton over which oiled silk should be applied. In addition, relief is sometimes afforded by applications containing opium, belladonna, or aconite, singly or combined. Chloroform as a local application has been recommended. Trousseau highly recommended tobacco fumigations, not during the paroxysms, but in the intermissions, with a view to diminish the suffering when the pain returns.

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[1] This remedy was suggested as a remedy for gout, on theoretical grounds, by Buckler in 1853.

The diet for a few days should be considerably restricted, animal food being excluded. Should the attack continue, however, it is important, as in other diseases, to provide against the evils of innutrition. A nutritious but unstimulating diet should be directed, embracing milk, farinaceous articles, eggs, fowl, and fish.

Treatment in the Intervals between Attacks of Transient Gout.—The ends of treatment between the attacks are—the prevention of the recurrence of the disease, the prolongation of the intervals, and the diminution of the severity of attacks. The immediate rational objects are—prevention of the undue production of uric acid, its elimination by the natural emunctories, and the promotion of its solubility in the blood. With reference to the latter objects, it is to be borne in mind that the uric acid probably has been accumulating in the blood for some time before gouty manifestations occur, and that probably the deposit of the urates within and around joints precedes, for a greater or less period, the inflammatory phenomena which belong to an attack or fit of the gout.

Removal of the known causes of gout is an essential part of the treatment. Wine and malt liquors, especially the stronger kinds, are to be interdicted. If the use of alcoholics be desirable or allowable, spirits should be substituted. Gin is to be preferred on account of its diuretic tendency. The diet should be plain, wholesome, and restricted to an amount required for nutrition. A certain amount of bodily exercise is an important part of the hygienic management. Travelling, a long sea-voyage, and a sojourn in a tropical climate are in some cases advisable. All exciting causes are, as far as practicable, to be avoided.

If dyspeptic or other symptoms render it probable that an attack is impending, remedies with a view to prophylaxis are indicated. Alkalies should be given in small doses, largely diluted. They should be continued for a short time, then intermitted, and afterward resumed if the indications continue or return. The salts of potassa are to be preferred to those of soda. The carbonate of lithia may also be given. If the near approach of an attack be strongly suspected, colchicum in small doses may be resorted to. There is reason to believe that this remedy sometimes wards off an impending attack.

These remarks have reference to the treatment directed especially to gout. Disorders of any kind not of a gouty character of course claim appropriate treatment.

Treatment of Persistent or Chronic Gout.—The removal of causes which produce or promote the gouty diathesis, and of exciting causes, ranks first in importance. This is a *sine quâ non* in the treatment. In addition, in obstinate cases, when circumstances permit, travelling, a sea-voyage, and residence in a tropical climate may be recommended as likely to effect a salutary constitutional change. Hygienic is doubtless vastly more important than medicinal treatment, but remedies are undoubtedly of much utility.

Alkalies, the carbonate of lithia, and other diuretics are indicated precisely as in the intervals between attacks of transient gout when the symptoms denote an impending attack. It is important to bear in mind the injunction to give alkaline remedies in small doses, largely diluted, and to intermit their use after they have been continued for a short time. This injunction is also applicable to the carbonate of lithia and other diuretic remedies. Exacerbations of the disease, which are liable to occur from time to time, may be treated with colchicum in small doses, its effects upon the alimentary canal and circulation being carefully watched. Other remedies, of the value of which in certain cases there is abundance of testimony, are the iodide of potassium, guaiacum, and the phosphate of ammonia. Perhaps the benzoic acid is to be added. *Fraxinus excelsior* (European ash) is said to have efficacy "administered in an infusion with thirty grains of the leaves in half a pint of water, strained and sweetened, and taken twice or thrice daily."[1]

The most important point in the treatment of the gouty diathesis is the regulation of the diet. It has been generally held that the diet of gouty individuals should consist largely of farinaceous food. It has been customary to withhold to a great extent animal food, which is rich in nitrogen. This selection of a diet chiefly vegetable is based upon the assumption that the formation of uric acid, which it is desired to restrict, is increased by highly nitrogenous food. Clinical experience, however, has shown, as has been pointed out especially by Draper,[2] that saccharine and amylaceous articles of food are in general not so well tolerated by gouty patients as is animal food. Draper, therefore, recommends in gout a diet similar to that suitable for diabetes mellitus—namely, one composed mainly of animal food, fatty substances, and vegetables which do not contain much sugar or starch. The experience, however, of many physicians is in favor of a predominantly vegetable diet.

It is important that food should not be taken in excess; and still, it should never be inadequate to nutrition, either as regards quantity, kind, or variety. The articles of food which are to be especially recommended in the treatment of gout are lean meats, fish, soups, eggs, milk, bread, and green and succulent vegetables, such as tomatoes, cauliflower, cabbage, and the different kinds of salads. If well borne, fatty food may be taken. Saccharine substances, especially pastry, starchy vegetables, particularly potatoes, and acid substances are to be avoided. Ripe fruits may be taken in moderate quantity. Alcoholics, particularly the heavy wines and malt liquors, are to be interdicted. Pure water should be taken freely, especially whenever there are gouty premonitions, in order to hold in solution in the blood the uric acid and to promote its elimination. The sparkling table-waters are likely to be taken more freely than simple water, and they are therefore useful. Active exercise in the open air is a means only second in importance to the regulation of the diet. The function of the skin should be kept active also by frequent bathing and friction.

To relieve stiffness and soreness of the joints methodic friction or shampooing is highly useful, provided undue violence be not used. Small blisters from time to time are recommended. Collections of soft gouty matter—or "gouty abscesses," as they have been called—if near the surface may be punctured and the creamy matter gently pressed out. Occasionally ulceration follows, which is troublesome, but leads to no evil results. Deformities, nodosities, and ankylosis due to chalk-stones and structural changes are effects of the disease which do not admit of removal.

Different mineral waters have been much resorted to in the treatment of gout, particularly the Vichy water. Of the utility of the latter and of other springs there is much difference of opinion among those who have given more or less attention to an investigation of their effects. The Vichy water is alkaline, its alkalinity being due to the bicarbonate of soda. The imported and the artificial water are used to a considerable extent in this country; but springs which owe whatever therapeutical value they possess to other saline ingredients, such as the sulphate of soda or of magnesia, the chloride of sodium or of calcium, the chalybeate and the sulphur springs, are severally advocated as useful in cases of gout. The Carlsbad springs are much resorted to by gouty patients. It is difficult to eliminate the beneficial influence of hygienic circumstances connected with a residence at watering-places, and thus arrive at a fair judgment of the therapeutic value of the water. For the discussion of this question the reader is referred to other works.[1]

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[1] *National Dispensatory*, 1879.

[2] Article "Gout" in *System of Practical Medicine, by American Authors*, vol. ii.

The treatment of irregular and misplaced gout involves indications derived from the character and seat of the disorders; in other words, from the symptoms in individual cases. If gout have retroceded local measures to solicit its return are indicated. For this end warm and stimulating applications are to be made to the part or parts in which the regular manifestations have been seated.

Rheumatoid Arthritis—Deforming Arthritis.

This disease has received a variety of names, of which may be mentioned *rheumatic gout*, *nodosity of the joints* (Haygarth), *chronic rheumatic arthritis* (Adams), *nodular rheumatism*, *dry arthritis*, *proliferating arthritis*, *chronic osteo-arthritis*, *arthritis pauperum*, and *malum senile articulorum*. When the affection attacks the hip-joint it has been called *malum coxæ senile*. When the vertebral articulations are affected it is called *spondylitis deformans*. The term rheumatic gout is much used by practitioners to designate this disease, but it is objectionable. It implies that the disease is a combination of rheumatism and gout. Rheumatism and gout very rarely occur conjointly. It is quite certain that the disease under consideration has no relation with gout, and it is probable that it differs in its nature from rheumatism. The names which have been placed at the head of this article seem to be the most appropriate of those which have been suggested. Garrod introduced the name rheumatoid arthritis. Arthritis deformans is the name chiefly used by German writers.

Some writers make no distinction between rheumatoid arthritis and chronic rheumatism. It must be admitted that it is impossible to draw a sharp distinction between these affections. Indeed, we have very little positive knowledge concerning the etiology and pathology of many chronic affections of the joints. It is probable that under the names chronic rheumatism and rheumatoid arthritis are embraced a number of essentially distinct diseases, but we have no insight into the real nature of these diseases. Anatomically, those cases are usually designated as chronic rheumatism in which the extensive osseous formations and deformities which characterize rheumatoid arthritis are wanting, but it will not infrequently happen that a case which has been considered as one of chronic rheumatism takes on the characteristics of rheumatoid arthritis.

The ANATOMICAL CHARACTERS of rheumatoid arthritis are as follows: Early in the case there may be an accumulation within the affected joints of clear or slightly turbid fluid which in the progress of the disease is absorbed. Fluid effusion, however, does not necessarily occur at any period of the disease, and as a rule upon post-mortem examination the cavities of the joints are found so dry that the disease has received the name of dry arthritis (arthritis sicca). Important changes occur in all the structures which compose the joints. The articular cartilages are absorbed, especially their central parts, where opposing cartilaginous surfaces come into contact. The ends of the bones, being denuded of cartilage, come into contact, and acquire a hard, smooth, polished appearance in consequence of the friction to which they are subjected. This condition of bone is called eburnation. Around the margin of the articulation there is a new growth, in nodular masses, of cartilage and of bone. In this way the articular extremities of the bones become enlarged by the formation of a rough, irregular, bony and cartilaginous ring which surrounds the eburnated surfaces. After the disappearance of the cartilage the process of absorption may invade the articular ends of the bones. Thus, when the hip-joint is affected, not only the head, but a considerable portion of the neck, of the femur may be absorbed. In consequence of the thickened condition of the end of the bone from the marginal osseous growths, the appearance is often as if the eroded head of the femur had been pushed down nearer to the shaft. Irregular bony outgrowths or nodosities are formed around the sockets. The sockets are sometimes widened and sometimes deepened and contracted. Dislocations may have occurred and new sockets have been formed. The synovial membranes and the ligaments of the joints are also affected. The little polypoid or villous outgrowths which are normally present to a certain extent upon the inner surface of the synovial membrane often become greatly increased in number and size, so that the inner surface of the thickened membrane may acquire a shaggy appearance, especially where it extends for a short distance over the articular extremities. To these hypertrophied synovial villi the name dendritic vegetations of the synovial membrane is sometimes applied. Some of these hypertrophied villi may become changed into cartilaginous or even into osseous nodules, which are often connected by narrow pedicles with the inner surface of the synovial membrane. The pedicles sometimes rupture, so that the bony or cartilaginous nodules are detached and exist as foreign bodies in the articular cavity. The ligaments may also become thickened. Plates of cartilage and of bone may develop in these thickened ligaments. An ossifying periostitis exists to some extent around the ends of the bones. Osseous ankylosis is said never to occur in arthritis deformans, but the joints may be rendered immovable by the thickened condition of the ligaments and by the new bony growths. The interarticular cartilages are generally destroyed, and also the round ligament of the hip and the long tendon of the biceps brachii when the joints in which they are situated are affected. The disused muscles are atrophied. The atrophy is said to be sometimes greater than can be accounted for by the disuse of the limbs, so that some writers consider that a neuropathic element is involved.

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[1] For a discussion of mineral waters in general in gout, and the relative value of different European springs, vide Garrod, *The Nature and Treatment of Gout and Rheumatic Gout*, London, 1859.

The microscopical examination of the cartilage in this disease shows a proliferation of the cartilage-cells and a breaking up of the basement-substance into fibrils. The spaces enclosed by the capsules of the cartilage-cells become greatly enlarged, especially in a vertical direction. These enlarged spaces contain proliferating cartilage-cells. The intercellular substance is thus reduced in amount. According to Cornil and Ranvier, the capsules on the surface rupture into the articular cavities, into which the cartilage-cells are discharged. The intercellular substance between the ruptured capsules projects for a time in the form of little villi from the surface of the cartilage, to which they give a filamentous, velvety appearance. In the progress of the disease the filaments are destroyed and the cartilage-spaces are gradually opened until the whole central part of the cartilage is removed. At the periphery the articular cartilage is covered by synovial membrane. Here the cartilage-spaces filled with cells do not rupture, but the proliferative process results in the formation of new cartilage, instead of in absorption. This new cartilage may undergo ossification.[1] The so-called chalky deposit of gout is wanting, but rheumatoid arthritis may occur in persons who have this deposit as an effect of attacks of gout which have previously occurred.

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[1] According to Ziegler, cartilaginous and fibrous nodules develop in the bone beneath the cartilage. These nodules may be converted into cysts. A rarefying ostitis occurs,

The disease is usually, from the first and during its course, subacute. Sometimes the local and general symptoms denote a certain degree of acuteness, causing a resemblance to acute rheumatism. A sudden invasion successively of different joints, however, does not take place as in the latter affection, and there is no liability to pericarditis or to endocarditis.

The development and the progress of the disease are slow. The smaller joints may be first affected, and subsequently the larger joints, or the reverse of this may obtain. A feature of the disease is its progressive character as regards increase of the anatomical changes in the joints affected and extension to other joints. It may, however, remain stationary, in both these respects, for a long time. Pyrexia, except at times, is wanting, and there may be but little constitutional disturbance. The appetite, digestion, and nutrition are sometimes but little affected. The affected joints are painful, especially on motion.

A particular deformity of the fingers is characteristic—namely, a lateral deflection in the ulnar direction. The toes sometimes present also this peculiarity. The situation and motions of the thumb generally are unaffected.

In the progress of the disease the anatomical changes lead to permanent extension or flexion of parts, dislocations, nodulations, and notable distortions. Extending, as it sometimes does, over all the joints of the body (polyarthritis deformans), the trunk is bent and rigid, the neck is immovable, the jaws are more or less fixed, the limbs are distorted, the muscles are attenuated from disuse, and the patient presents a most distressing spectacle of utter helplessness. Between this picture of extreme progress and the early appearances of the disease there is every degree of gradation.

The disease may occur at all ages, but in the majority of cases patients are in middle life, and much oftener women than men. It occurs in the poorer classes of society and among those who suffer from hardships, exposure, and deprivations; but it is by no means confined to these. Persons are rarely affected when in full health and vigor. Its victims are those whose constitutions are broken down by various circumstances affecting body and mind. Large hospitals in which chronic cases are received are seldom without examples of this disease in the different stages of its progress.

More than forty years ago Prof. J. K. Mitchell maintained that diseases of the joints resulted from affections of the spinal cord. More recently attention has been directed by Remak, Benedikt, Weir Mitchell, Charcot, and others to morbid conditions of the cord and injuries of nerves as giving rise to arthritic affections. On the basis of facts exemplifying this pathological connection, together with the symmetrical occurrence of the joint affections and apparent etiological relations in some instances, the theory that rheumatoid arthritis is of nervous origin is not irrational. This theory is supported by the well-known occurrence of trophic disturbances of the muscles as effects of diseases or injuries of the cords and nerves.

Different varieties of arthritis deformans can be distinguished. In one group of cases (the polyarticular) the affection begins primarily in the small joints of the hand and foot, where it either remains or extends subsequently to the larger joints, as the knee, elbow, shoulder, and hip. In other cases the large joints are first affected, and here there is often a tendency in the disease to confine itself to a single joint, especially to the hip-joint or the shoulder-

resulting in enlargement of the medullary spaces and absorption of the bony trabeculæ. Ziegler observed changes which he interprets as proliferation of the bone-cells (*Virchow's Archiv*, 1877, Bd. 70).

joint (monarticular form). In some cases the vertebral column, and often at the same time the pelvic articulations and the hip, are solely involved (spondylitis deformans). Charcot considers that the affection characterized by nodosities of the small joints, particularly the distal phalangeal joints of the hand, should be separated from other forms of deforming arthritis. He proposes for the last variety the name Heberden's rheumatism or nodosities, from the author who first accurately described them. Whether these are to be regarded as varieties of the same morbid process or as different diseases is uncertain; but at present they are most conveniently grouped under the same heading.

The DIAGNOSIS involves discrimination from rheumatism and gout. From chronic rheumatism it is not readily discriminated prior to the occurrence of its deforming effects, especially if the affection have not been preceded by acute rheumatism. These effects constitute the diagnostic criterion. It is distinguished from gout by the larger joints being primarily affected in a certain proportion of cases; by the smaller joints of the hands being affected without a previous affection of the toes; by the absence of the paroxysms or exacerbations which are characteristic of gouty attacks; by the absence of the chalk-like deposit and of an excess of uric acid in the blood; and by its occurrence in a certain proportion of cases in young subjects and especially in women.

Although not involving immediate danger to life, rheumatoid arthritis is a hopeless disease as regards recovery. All that can be hoped for is a certain measure of improvement, non-progression, and indefinite tolerance.

The first object of TREATMENT is the improvement of the general health. Tonic remedies, long continued and varied from time to time, in conjunction with a nutritious diet and other hygienic measures calculated to invigorate the system, are to be employed for this object. Remedies which have a special influence in cases of rheumatism and gout—namely, alkalies, colchicum, and salicin or the salicylic acid—are unavailing in this affection. All depressing remedies are contraindicated. Iodine in the form of either the iodide of iron or the iodide of potassium, guaiacum, and arsenic appear to be useful in certain cases. Cod-liver oil may be given in conjunction with tonics if there be a tendency to waste. Chalybeates are especially indicated if the patient be anæmic. Warm baths are recommended by many writers. The alkaline mineral waters and those which act powerfully upon the bowels or kidneys are hurtful. On the other hand, chalybeate and tonic waters may prove beneficial.

Local measures of treatment are important. Leeching or cupping and severe counter-irritation are not indicated. Small blisters and the application of iodine may be useful if there be effusion within the joints. Rest is indicated while effusion exists or when the symptoms denote inflammation; and afterward passive motion, friction, warm douches, shampooing, and the constant current constitute the most important part of the treatment.

CHAPTER XII.

SCORBUTUS.—PURPURA.—HÆMOPHILIA.

Scorbutus, or Scurvy: Anatomical Characters; Clinical History; Pathological Character; Causation; Diagnosis; Prognosis; Prevention; Treatment.—Purpura Simplex, Purpura Rheumatica, and Purpura Hæmorrhagica.—Hæmophilia.

THIS concluding chapter will be devoted to three affections which, as regards symptomatic events, are closely allied to each other—namely, *scorbutus*, or *scurvy*, *purpura*, and *hæmophilia.* The first of these affections is of interest and importance especially in view of the fact that our knowledge of its pathology and causation, although by no means complete, is sufficient to serve as a basis of effective treatment, and has rendered the disease preventable.

Scorbutus, or Scurvy.

This disease appears to have been known to the ancients, but it began to prevail frequently and extensively when, with improvements in navigation, long voyages were undertaken, and hence it was called *sea-scurvy.* During the last five or six centuries it has been pre-eminently the disease destructive to life in armies, navies, and exploring or emigrating expeditions by land and by water. Notwithstanding that it has been rendered preventable by our knowledge of its pathology and causation, it has prevailed largely and proved greatly destructive to life within the present century. Hammond states that at Council Bluffs in 1820 nearly the entire garrison was attacked, and many died; and the efficiency of the United States forces in the Florida and the Mexican Wars was very materially lessened by its occurrence.[1] It prevailed enormously in the English and French armies in the Crimean campaign. It contributed not inconsiderably to the mortality of our armies in the late Civil War. The inconsistency between these facts and the existing state of knowledge is in part to be explained by the inability always to secure the means of prevention in military operations; but it is in a greater measure to be accounted for by a censurable ignorance or neglect of these means.

There has been of late years great success in preventing scurvy in the naval services of the chief nations of the world. According to Wales,[2] in 1881, out of 82,629 cases of disease in the chief naval services of the world, there were only 34 cases of scurvy—a ratio of 41 per 1000. The statistics of the mercantile marine are less favorable.

ANATOMICAL CHARACTERS.—It is probable that the primary and essential morbid changes relate to the blood. Our present knowledge, however, does not enable us to state in what consist the changes peculiar to the disease. Observers differ as regards the appearances as well as the results of chemical and microscopical examinations. The color has been found sometimes darker and sometimes lighter than that of normal blood. The blood in the heart-cavities and vessels has been in some instances fluid and in other instances coagulated. The blood-corpuscles have been considered by some observers diminished in number, and by others unaffected in this regard. No uniform changes in the form or constituents have been determined. It is not established that a deficiency of potassa is a constant feature. Contradictory results have been obtained respecting the white corpuscles, the quantity of fibrin, and the proportion of albumen. In short, quoting Immerman, "If we take a general survey of the total analyses made of the blood of scurvy patients up to this time, we can hardly avoid a feeling of dissatisfaction. From the various and contradictory statements by different authors, we obtain but *one*, and that a decidedly *negative* result—namely, that the condition of the blood of scurvy patients, so far as we understand it at the present time, shows no peculiarities which might not occur as well in other pathological processes."

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[1] "Report on Scurvy," *United States Sanitary Commission*, by William A. Hammond, M. D., 1862.

[2] Article "Scurvy," in *A System of Practical Medicine, by American Authors*, vol. ii. p. 168.

Extravasations of blood are found within and beneath the skin, where they are apparent before death, and between the fibres of muscles. The gums are swollen, ulcerated, and sometimes sloughing. A bloody serum is often found in the joints, associated sometimes with intra-articular ulcerations. A sero-sanguinolent liquid, often in considerable or large quantity, together with inflammatory products in a certain proportion of cases, is contained in the pericardial and pleural cavities. The spleen is often enlarged and softened. Parenchymatous and fatty degenerations of the heart, muscles, liver, and kidneys have been found. The kidneys may be infiltrated with blood and inflamed. Ecchymoses are observed in many internal situations—namely, on the serous membranes, the bronchial mucous membrane, within the whole tract of the alimentary canal, and in the urinary passages. The extravasations are evidently secondary to, and dependent upon, the morbid blood-changes. The mode, however, in which the latter give rise to the former is not clearly understood. Either the blood is so altered that it escapes from the vessels, or the nutrition of the walls of the vessels is impaired, so that they do not offer adequate resistance to the pressure of the blood, or, again, paralysis affecting the vaso-motor nerves may be a pathological element. It is not impossible that each of these conditions enters into the mechanism.

CLINICAL HISTORY.—The symptomatic events which are characteristic of the disease are preceded by general weakness and lassitude. Patients are listless, apathetic, and indisposed to either mental or physical exertion. The appetite fails and pallor of the surface is marked. These symptoms progressively increase. The more characteristic events are swelling and sponginess of the gums, which bleed either spontaneously or on slight pressure, hemorrhage from the mucous surfaces in other situations, and extravasation of blood within and beneath the skin. The fungous appearance of the gums is chiefly marked around the teeth. It is less marked in situations where the teeth are wanting. The teeth may become loose, and they sometimes either fall out or can be easily removed. The prolabia are notably pale, presenting a striking contrast to the redness of the gums. Hemorrhages are frequent from the different outlets—namely, the nostrils, mouth, vagina, and intestinal canal. It is rare from the bronchial tubes. The ecchymoses apparent on the cutaneous surface are either in the form of petechiæ or of patches of variable size; and generally these forms are conjoined. They are either limited to or most abundant on the trunk and lower extremities. The ecchymoses undergo the same successive alterations in color as when due to contusions. A slight bruise suffices to produce them in any part of the surface of the body. They occur sometimes beneath the conjunctiva and within the globe of the eyes; into the joints, between the muscles, in the serous cavities, occasionally between the meninges of the brain, and beneath the periosteum. Fetor of the breath is usually a marked symptom.

The skin is dry and rough, compared by Larrey to that of a plucked fowl. The ankles are often œdematous, and some œdema of the face is not infrequent. The patient complains of pain in the loins and in the extremities, especially the lower limbs. The muscles of the lower limbs are swollen and hard. Palpitation and dyspnœa are excited by slight exertion. Hæmic murmurs are heard over the heart. Pain referred to the chest is not uncommon. The bowels are in some cases constipated, in other cases loose, or looseness may succeed constipation in the progress of the disease in the same case. The urine is diminished in quantity. The excretion of urea and of chlorides is diminished. The excretion of potash, either absolutely or relatively to that of soda, is increased. The phosphates are also increased in quantity. The specific gravity is usually higher than normal, and the color is more intense. The urine often is albuminous. There is absence of pyrexia except as symptomatic of an inflammatory complication. The pulse is sometimes slower than in health, but it becomes rapid on any exertion. It is always soft and compressible. The temperature of the body is somewhat lowered. The spirits are depressed. The patient frequently longs for fresh vegetables and fruit.

These symptoms are more or less marked. In extreme cases or in an advanced stage of the disease the prostration is great. Slight exertion occasions syncope. The patient may fall into a state of collapse. This is likely to follow abundant hemorrhage. Old ulcers and cicatrices sometimes reopen, and bones which had been fractured and united may become separated. Ulcerations occur within the mouth and over the ecchymoses on the skin. Weakness of vision, day-blindness, and night-blindness are not uncommon. Inability to sleep is common, but generally the mental faculties, although weakened, remain intact. In cases which are comparatively mild general debility, mental depression, pain in the back and lower limbs, together with the other symptoms, are more or less marked, but not excessive; and associated with these symptoms are spongy swelling of the gums and ecchymoses beneath the skin.

Scorbutus may exist as the sole affection, but it is frequently combined with other diseases. It may be combined with dysentery, and the latter affection is then called *scorbutic dysentery*. It may coexist with typhus and with typhoid fever. It may be accidentally associated with a variety of diseases which are liable to mask the scorbutic affection. The recognition of its coexistence with other diseases is of great importance with reference to appropriate treatment.

Secondary affections or true complications which are likely to occur are lobar pneumonia, pulmonary hemorrhagic infarctions, pulmonary gangrene, pericarditis and pleuritis with bloody effusion, and acute diffuse nephritis.

PATHOLOGICAL CHARACTER.—The essential characteristic blood-change in this affection is not as yet determined. There is doubtless a deficiency as regards certain of the constituents of the blood, but the particular deficiency which belongs specially to the affection is a matter of question. Garrod considers that the special deficiency relates to the salts of potassa. That this deficiency enters into the pathological condition is probable, but that it constitutes the pathological condition exclusively or chiefly in all cases may be doubted, in view of the fact that supplying the salts of potassa, although highly useful, is not always the most effective method of treatment. With our present knowledge we must be content with saying that owing to incomplete nutritive supplies the constitution of the blood is impaired, giving rise to certain ascertained morbid changes common to this and other affections, and causing other changes, not yet ascertained, which are peculiar to this disease. It is noteworthy that the phenomena of scurvy are not produced by prolonged abstinence from food. This fact goes to show that the unknown blood-change in this disease is of a special character.

A view which has been recently advanced is that scurvy is an infectious disease caused by a specific micro-organism. This view, however, has not received any substantial support from the study of the disease.

CAUSATION.—Scurvy is a dietetic disease. It is the broadest expression of the causation to say that the disease is due to the want of certain alimentary supplies. A less comprehensive but probably correct statement is to say that the disease is caused by an insufficiency of nutritive principles contained in vegetable food. For a long time the disease was attributed to the free use of salted meat. It is, however, certain that the disease is not produced by this diet, except in so far as it displaces other articles of food containing principles necessary for the normal constitution of the blood; and scurvy has repeatedly been developed when the diet has been too much restricted to fresh meat. Garrod's theory of the pathological character of the disease involves the conclusion that it is attributable to a diet wanting the requisite quantity of the salts of potassa, these salts being abundant in vegetable food. Admitting a deficient supply of the salts of potassa to be an important element in the causation, other elements are involved. The dietetic principles, the deficiency of which causes this disease, are abundant in various articles of food distinguished as antiscorbutic, experience having shown that they prevent the development of the disease.

Although causes relating to diet are essential to the production of the disease, other causes act powerfully as auxiliaries. Among the auxiliary causes are exposure to cold and wet, overcrowding, deficient ventilation, and depressing moral influences. The disease is oftener developed during the winter than during the summer. I have known an instance in which the disease appeared to be produced by great and prolonged mental depression, but probably in this instance, from an utter indifference to food, dietetic causes were involved. The disease is oftener developed in the aged than in young persons, and the cause of the disease will act more speedily in persons in whom the constitution has been impaired by previous disease, by injurious medication, and by excesses of any kind. There is reason to believe that the morbid condition of the blood which exists in scurvy is not infrequently produced, to an extent falling short of that requisite to give rise to the striking manifestations of the disease, by an undue restriction of the diet to a few articles of food, either from poverty or false notions of dietetics. It is important for the physician to recognize this fact in medical practice.

DIAGNOSIS.—In view of the striking symptomatic features of this disease, and of the fact that generally a greater or less number of persons become affected under circumstances which sufficiently account for its development, the diagnosis is easy, provided the characters of the disease be marked. In isolated cases the diagnosis involves discrimination from *purpura hæmorrhagica.* This differential diagnosis will be considered in treating of the latter affection.

The morbid condition of the blood peculiar to scurvy may exist to a greater or less extent when the most striking symptomatic features of the disease are not present—namely, the subcutaneous ecchymoses and hemorrhages from mucous outlets. Under these circumstances the disease may be overlooked. Thus, Hammond, Woodward, and others state that in cases called in army sick-reports cases of *general debility* the disease is not infrequently incipient or not fully-developed scorbutus. The pain in the back and muscles of the lower limbs may lead the practitioner for a time to consider the disease as muscular rheumatism or myalgia. When associated with other diseases, such as dysentery and the essential fevers, it is liable to be overlooked. The appearance of the gums is highly important with reference to the diagnosis in the cases in which petechiæ or vibices are wanting; and when, from the appearance of the gums and other symptoms, the scorbutic condition is suspected, an investigation of the diet may lead to the knowledge of facts which will tend to settle the diagnosis. This investigation is important, not only in army or naval practice and in public institutions, but in private cases in which the symptoms point to scurvy. It is to be added that in children before dentition and in aged persons who have lost their teeth the characteristic appearances of the gums may be wanting. They are also wanting in rare instances irrespective of these circumstances.

Prognosis.—In cases of scurvy in which the ecchymoses are many and large, or in which hemorrhage from the mucous outlets is profuse, the danger to life is great. Life may be destroyed by rapid hemorrhagic effusion into the pleural or pericardial cavities. Meningeal hemorrhage is sometimes the immediate cause of death. Sudden death sometimes occurs from syncope induced by muscular exertion. If the causes which have produced the disease continue in operation, it proves fatal in a large proportion of cases. Before the pathology and causation were as well understood as now the mortality from this disease among soldiers and seamen was often very large. Associated with other diseases, it contributes indirectly to their fatal termination, and it stands in the way of recovery from wounds and surgical operations which otherwise would not prove serious.

Promptly recognized and judiciously treated, the disease may be expected to end in recovery in a large proportion of cases. It has no definite duration. It continues for a longer or shorter period, according to the extent to which it has advanced, the amount and situations of hemorrhagic extravasation, the previous condition of the patient, etc. The recovery is usually gradual, not infrequently tedious, and the system is likely to remain debilitated for a long time.

Prevention.—The disease may be prevented by a diet embracing the requisite variety of alimentary principles, and this is secured by a proper variety of animal and vegetable food. With the knowledge of this fact, and with due attention to its practical application, scurvy should never occur except under circumstances which render the means of its prevention unavailable. Its occurrence is always an occasion for reproach when articles of food containing adequate supplies for the healthful constitution of the blood can be obtained. It is chiefly in military campaigns and long sea-voyages that there are difficulties in the way of prevention. These difficulties, however, may generally be met by providing antiscorbutic articles of diet; and with reference to these difficulties the knowledge of the various articles which may furnish the needed alimentary principles is of vast importance. The prevention of this disease has been made practicable under all circumstances by the preservation of meats, vegetables, and fruits in hermetically-sealed cans.

Lemon-juice or lime-juice has long been known as a most valuable antiscorbutic. Experience has abundantly shown that one or two ounces *per diem* of the fresh juice will prove an effectual preventive against scurvy during very long voyages without fresh provisions. For convenience of transportation, the juice can be reduced in bulk by evaporation. Orange-juice is also an effectual antiscorbutic. Pure citric acid, although it appears to be useful, is not a substitute for the fresh juice of the fruit. One of the most efficient of antiscorbutic vegetables is the potato. It is most efficient when eaten raw. Pickles, onions, raw cabbage or sauerkraut, the water-cress, and other of the *Cruciferæ*, are to be reckoned among the valuable antiscorbutic vegetables. Green corn, green apples, and most of the esculent fruits are preventives. They are more so unripe than ripe. Spruce and birch beer and yeast are antiscorbutics. Many vegetables which do not enter much into ordinary diet may often be used as antiscorbutics in military campaigns when the articles which have been named are not to be obtained. Among the vegetables now referred to which experience has shown to be effectual in the prevention of scurvy are the sorrel (*Rumex acetosella*), lamb's quarters (*Chenopodium album*), the bulb of the wild artichoke, a species of cactus known as the *Agave Americana* or *maguey*, indigenous in Texas, California, and Mexico, the prickly pear (*Cactus opuntii*), and the dandelion (*Leontodon taraxacum*).

Wales suggests that "in the event of the occurrence of scurvy and the exhaustion of the fresh-vegetable stores, various quickly-growing vegetables, such as mustard, radishes, turnips, and cresses, could be cultivated on shipboard if seeds are provided."[1]

That the antiscorbutic virtue of certain vegetables and fruits depends, to a greater or less extent, on the presence of the salts of potassa is altogether probable. These salts, isolated, are useful in the prevention as well as in the treatment, but experience has shown that they cannot take the place of antiscorbutic articles of diet.

Finally, in addition to dietetic means, attention to ventilation and cleanliness, avoidance of unnecessary exposure and physical exertion, and measures of a nature to produce cheerfulness, constitute an important part of the prophylaxis.

TREATMENT.—The dietetic means of prevention constitute an essential part of the treatment. The juice of lemons, oranges, and other fruits should be taken freely, but of course not to such an extent as to produce disorder of the stomach. The antiscorbutic vegetables and fruits should enter into the diet as abundantly as the digestive powers permit, in conjunction with meat, milk, and farinaceous food. Remedies to improve the appetite and strengthen digestion are indicated—namely, quinia, salicin, the bitter infusions or tinctures, the mineral acids, and chalybeate preparations. The salts of potassa are useful. The bitartrate or the citrate may be selected. Wine, spirits, or cider in small quantities, if an agreeable cordial effect be produced, will be useful. In short, the chief objects of treatment are the assimilation of alimentary principles needed for the healthy constitution of the blood and the invigoration of the system.

Particular symptoms or disorders claim appropriate measures in addition to those having reference to the objects just stated. Constipation is to be relieved by mild laxatives or simple enemas, and, on the other hand, diarrhœa is to be controlled by anodyne and astringent remedies. The dryness of the skin indicates sponging with tepid water or the tepid bath. Pain in the back and limbs may be alleviated by friction with anodyne and mildly-stimulating liniments. Stiffness of the joints, which sometimes occurs, is to be prevented or removed by passive movements. The condition of the mouth calls for astringent collutories. Ulcerations in the mouth or on the skin are to be treated locally on general principles. Hemorrhage from the different outlets is to be restrained by hæmostatic remedies.

It is hardly necessary to add that the scorbutic condition, when associated with dysentery, typhus or typhoid fever, or with any disease, claims the same dietetic and other measures of treatment as does scorbutus existing alone.

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[1] *Loc. cit.*

Purpura Simplex, Purpura Rheumatica, and Purpura Hæmorrhagica (Morbus Maculosus Werlhofii).

The term *purpura* is applied to circumscribed extravasations of blood into the skin. Purpuric spots are bright-red, livid, or dark purplish-red in color, do not disappear on pressure, and are unattended by itching or other signs of local irritation. Small extravasations of blood, from the size of a pin-point to that of a finger-nail, are called petechiæ. Larger extravasations are called ecchymoses. If the hemorrhages take the form of lines or broad stripes, they are called vibices. The purpuric spots generally are not elevated, but they sometimes appear as small papules around the hair-follicles (purpura papulosa, lichen lividus), or as wheal-like nodules (purpura urticans), or rarely there are formed bullæ containing blood (purpura bullosa). The extravasated blood undergoes the usual changes of color, becoming brown, green, and yellow.

Purpura may be due to a great variety of causes. It is customary to distinguish between *secondary* or *symptomatic* purpura and *primary* or *idiopathic* purpura; but it is probable that in many cases so-called idiopathic purpura is really symptomatic of some underlying morbid condition.

The following may be mentioned as the leading causes of secondary purpura: injury, including the bites of insects; passive congestion; various drugs, such as iodide of potassium, phosphorus, copaiba balsam, ergot, chloral hydrate, and mercury; snake-bites; embolism or thrombosis of cutaneous vessels; cachexiæ, as tuberculosis, cancer, Bright's disease, and cirrhosis of the liver; profound anæmia, as pernicious anæmia and leucocythæmia; diseases of the nervous system; infectious diseases, as smallpox, typhus fever, and typhoid fever; acute ulcerative endocarditis, measles, scarlet fever, acute articular rheumatism, malaria, cholera, yellow fever, cerebro-spinal meningitis, and icterus gravis; and, finally, scorbutus. Purpura may be accidentally present in the course of any disease.

Purpura is distinguished as primary or idiopathic when it is not dependent upon any other disease. As has already been mentioned, there is some doubt whether even in these cases the purpura be not symptomatic, but with our present knowledge it is convenient to treat of purpura as sometimes an idiopathic disease.

Idiopathic or primary purpura is usually subdivided into three varieties—namely, purpura simplex, purpura rheumatica, and purpura hæmorrhagica, or morbus maculosus Werlhofii. No sharp lines of distinction can be drawn between these varieties. They merge into each other by every grade of transition, so that it is often difficult to classify cases according to these subdivisions.

Purpura simplex is the mildest form of idiopathic purpura. Hemorrhage occurs only in the skin, and not from mucous membranes. Purpura simplex occurs both in the robust and in the debilitated. The purpura may be preceded or accompanied by languor, loss of appetite, aching of the limbs, and slight fever, or there may be no constitutional disturbance whatever. Purpuric spots occupy chiefly the lower extremities, or they may extend to the trunk and upper extremities, and in rare instances to the face. Only a single crop of purpuric patches may develop, in which case recovery takes place in between ten and fourteen days, or the disease may be prolonged for several weeks by the appearance of successive crops. Even in mild cases of purpura, hemorrhages from mucous surfaces, particularly hæmaturia, may occur, thus showing the close relationship which exists between the different varieties of the disease. Purpura simplex is not uncommon in elderly persons. It is then called purpura senilis.

Purpura rheumatica—or, as it is also called, *peliosis rheumatica*—is characterized by the combination with purpura of rheumatic pains in the joints, and sometimes with actual inflammation of the joints. There are often moderate fever, debility, and gastric disturbances. The joint affection may precede or accompany the purpura, or it may disappear when the purpuric eruption occurs. The duration is generally between ten days and three weeks, but relapses are common, especially when the patient walks too soon. Rheumatic purpura is most common in males between twenty and thirty years of age. The affection of the joints is etiologically distinct from acute articular rheumatism. Joint affections are common in all forms of primary purpura, and in general in diseases characterized by a hemorrhagic diathesis. The propriety, therefore, of distinguishing rheumatic purpura as a separate variety of the disease is questionable. The occurrence of hemorrhages from mucous membranes in some cases which have been classified as rheumatic purpura shows the relationship of this to the following variety of idiopathic purpura.

Purpura hæmorrhagica is not an appropriate name for the variety of purpura now to be described, as every kind of purpura is hemorrhagic. The name *morbus maculosus Werlhofii*, or Werlhof's disease, is the one adopted by German and many English writers. This variety of purpura is characterized especially by the occurrence of hemorrhages, often profuse, from mucous surfaces. The hemorrhages in the skin usually are larger and greater in number than in purpura simplex. Constitutional symptoms are more marked and the prognosis is graver. The disease is somewhat more frequent in females than in males. It develops oftenest between fifteen and thirty years of age. It may occur in previously healthy individuals, but it is more common in those whose constitution is enfeebled by disease or bad hygienic surroundings. Pregnancy and the puerperal state have been thought to aid in the causation.

The first manifestation of morbus maculosus Werlhofii may be the appearance of purpuric spots in the skin, or, less frequently, hemorrhage from some mucous surface. Often, however, there are vague premonitory symptoms, such as malaise, anorexia, vomiting, pains in the muscles, and even slight fever. Pain and swelling of the joints may occur as an early symptom. Hemorrhages occur from the nose, mouth, throat, stomach, intestine, and urinary organs. Bronchial and pulmonary hemorrhages are rare. In severe cases hemorrhages take place into serous cavities, and even into the substance of internal organs. Petechiæ and ecchymoses can be detected in the visible mucous membranes. As in all forms of purpura, hemorrhages are liable to occur in parts of the skin subjected to pressure or injury. In distinction from scurvy, the gums are not swollen and inflamed. Constitutional disturbance may be slight or even wanting, but often it is marked. There may be fever, which is usually moderate, but is sometimes high. The patient sometimes sinks into a typhoid state. There may be diarrhœa, colicky pains, and other severe gastric and intestinal symptoms. Endocarditis and acute hemorrhagic nephritis are occasional complications. There sometimes is acute splenic tumor. Perforative peritonitis has been known to occur. Symptoms of anæmia, such as vertigo, weakness, functional cardiac disorders, and œdema, develop in proportion to the loss of blood. Hemorrhage may occur into the brain or its meninges. Death may result from loss of blood or from grave constitutional disorder, marked especially by typhoid symptoms or from some complication.

The duration of the disease is usually between two and four weeks, but it may be protracted for many months. The prognosis in general is favorable, but it is necessary to be cautious in expressing a positive opinion on this point, as even in apparently mild cases grave symptoms may develop. As has already been remarked, there are cases representing every grade of transition from the mildest purpura simplex to the most severe purpura hæmorrhagica.

The DIAGNOSIS from scorbutus is based especially upon the absence of swelling of the gums and of ulcerative inflammations elsewhere, by the lack of the cachexia which antedates and accompanies scurvy, by the absence of the dietetic causation of scurvy, and by the comparative inefficacy of the dietetic regimen, which, as a rule, promptly relieves the symptoms of scurvy. According full force to these points of difference, it must be admitted that they do not establish conclusively the non-identity of purpura hæmorrhagica and scorbutus, nor do they always suffice to enable the practitioner to make with positiveness the differential diagnosis.

Nothing is known as to the essential cause of purpura hæmorrhagica. Changes have been surmised to exist in the walls of the blood-vessels, in the composition of the blood, and in the nervous system, but none have been demonstrated which can fairly be considered causative. Infection has also been suggested as a cause.

The objects of TREATMENT in cases of purpura hæmorrhagica are—the restoration of the normal constitution of the blood, increase of the vital powers, and arrest or restraint of hemorrhage. With reference to these objects the diet should be nutritious and varied, and digestion is to be aided by tonic remedies, together with wine or spirits in small quantity if the immediate effect be good. In view of the difficulty in always discriminating purpura from scorbutus, and of there being ground for supposing that the same morbid condition of the blood exists in both affections, the treatment should embrace the dietetic and other measures which experience has shown to be useful in cases of scorbutus. Of tonic remedies, the dilute sulphuric acid is especially recommended. This remedy has the advantage of being agreeable to the taste if added to a proper quantity of water and syrup. It may be given in doses of from five to fifteen drops every two hours. Quinia is often combined with the sulphuric acid. Hæmostatics are to be given with reference to the hemorrhage. Ergot or ergotin has been highly recommended. The gallic acid, the muriated tincture of iron, and the acetate of lead are remedies which may be employed. The oil of turpentine has been recommended as a valuable remedy in this affection. It is important that the patient should preserve the recumbent posture in bed, avoiding all unnecessary exertion.

Hæmophilia.

Hæmophilia, or hæmatophilia, is a congenital and usually an hereditary affection, characterized by a tendency to immoderate bleeding, traumatic or spontaneous, and often associated with swelling of the joints. Persons with this diathesis are called "bleeders." No other disease illustrates in so striking a manner transmission by inheritance. The diathesis has been traced through successive generations of the same family for more than two hundred years. Males are far more liable to the disease than females, the ratio being as 11 of the former to 1 one of the latter.[1] The diathesis is transmitted, however, far more frequently through the female members of bleeder families than through the male members. The daughters of a bleeder family, even when not themselves bleeders, transmit the diathesis to their sons. A son of a bleeder family, if himself a bleeder, should he live to beget children more frequently transmits the disease to his grandsons, through his daughters, than directly to his own children. The male members of a bleeder family, if not themselves bleeders, rarely transmit the disease to their descendants. It rarely happens that all of the members of a family are bleeders. It is uncertain whether the diathesis ever dies out in a family, although it may pass over one or more generations. It has been noted that the females of bleeder families are very fertile. In 12 such families there were 120 children (Wachsmuth).

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[1] Of 780 cases collected by Dunn, 717 were males and 63 females (*American Journ. of Med. Sciences*, Jan., 1883).

Hæmophilia may develop, as it were spontaneously, in individuals coming from a healthy family stock. Such persons may become the starting-points of new bleeder families. Even in these spontaneous cases the diathesis seems to be congenital. Probably in a considerable number of the apparently spontaneous cases a full knowledge of the ancestral history would reveal transmission by heredity.

A larger number of instances of hæmophilia have been reported among the Anglo-Germanic nations than among other races.[1] The affection has been repeatedly observed in Jews. It seems to be more common in cold, moist climates than in warm climates. Although a large proportion of bleeders have been blondes, with fair, delicate skin and prominent superficial veins, it is not likely that any such constitutional peculiarity is involved in the etiology of hæmophilia.

The essential causes of hæmophilia are wholly unknown. It is reasonable to suppose that abnormal constitution of the vascular walls and deficient coagulability of the blood are involved in the causation, but we have no positive data upon which to base these suppositions. No morbid changes have been found, with any uniformity, after death from hæmophilia. An abnormal thinness of the arterial walls has been noted in several cases. It is possible that there are defects pertaining to the fibrin-factors or to the blood-plates, but these have not been ascertained.

There is usually nothing indicative of either this diathesis or aught abnormal prior to the occurrence of a hemorrhage, which is with difficulty, if at all, controlled. The diathesis is sometimes manifested immediately after birth by profuse and sometimes uncontrollable bleeding from the umbilicus. More frequently, the affection is made apparent at a later date, but during the first few years of life. It is exceptional for the diathesis not to be discovered until after the second dentition. Persons having this diathesis continue to lose blood from an insignificant wound, such as that produced by a puncture, a slight cut, a leech-bite, or the extraction of a tooth, all efforts to arrest the hemorrhage proving ineffectual until syncope occurs, which may prove fatal, and if not fatal the loss of blood induces a notable degree of anæmia. The hemorrhagic diathesis also leads to extravasation of blood, often extensive, within and beneath the integument, resulting from slight contusions. Hemorrhage is also likely to occur from the nose and the gums, and less frequently the stomach, the intestine, and the urinary passages. Hemorrhages into internal organs which are removed from any external mechanical influences are very rare, if indeed they occur at all. In females, in whom the diathesis is generally less marked than in males, the menstrual flow usually is profuse and postpartum hemorrhage is abundant.

As in other diseases with a hemorrhagic diathesis, there is a tendency to swelling of the joints, most frequently the knee-joint, particularly after some injury. The swelling seems to be due to hemorrhage into the joint. Neuralgia, especially of the trigeminus, is not infrequent.

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[1] A full account of the American literature on this subject will be found in the article by Dunn above cited, and in the article on "Hæmophilia," by Osler, in *A System of Practical Medicine, by American Authors*, Philadelphia, 1885, vol. iii.

All degrees of the hæmophilic diathesis may exist, from the rudimentary form, in which there is simply a tendency to excessive but not dangerous hemorrhage after a wound, to the fully-developed form which has been described. Children affected in a marked degree with this diathesis rarely live to adult age, but in some instances the diathesis diminishes or ceases after childhood; and if it continue in a moderate or slight degree it may not interfere with longevity.

The prophylactic treatment is the most important in the management of hæmophilia. Wounds are of course to be avoided as far as possible. The diathesis constitutes an insuperable objection to all surgical operations which are not unavoidable. Teeth should never be extracted. The operation of circumcision should not be permitted. Vaccination should be performed with great care. Leeches should never be applied to "bleeders." Measures should be employed to invigorate the system, such as a nutritious diet, out-of-door life, frequent bathing, and tonic remedies. The marriage of female members of bleeder families and that of male members who are bleeders should be prohibited. When hemorrhage occurs, either from wounds or spontaneously, local measures to arrest the flow of blood are to be resorted to if practicable, and hæmostatic remedies are to be given internally.

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